The Embryology and Management of Congenital Pouch Colon Associated With Anorectal Agenesis

By Rajiv Chadha, Deepak Bagga, C.J. Malhotra, Anup Mohta, Ajay Dhar, and Ajay Kumar
New Delhi, India

● **Forty-one infants with a pouch colon malformation accompanied by a high anorectal anomaly were treated between January 1986 and December 1990. The 41 cases constituted 9% of all anorectal malformations and 15.2% of high defects managed during this period. There were 32 boys and nine girls; three of the girls had an associated cloaca. Many of the babies presented in poor condition, with gross abdominal distension caused by the distended colonic pouch. The typical radiological feature was an enormously distended colonic shadow occupying more than 50% of the width of the abdomen. At the time of surgery, the patients were classified into 4 subgroups based on the length of the normal colon. All but three infants had a high wide fistula, with the genitourinary tract consisting of a colovesical fistula in males and a colovaginal or colocloacal fistula in females. Frequent associated malformations included duplication of the appendix and vesicoureteric reflux. The operations performed initially were a window colostomy of the pouch with or without division-ligation of the fistula, end-colostomy after fistula ligation, or subtotal pouch excision with tubularization of the remaining colon and end-colostomy. Thirteen of the 41 patients have undergone a definitive pull-through operation using the posterior sagittal approach, including two children in whom one-stage reconstruction of a cloaca was performed. Standardized management of this complex anomaly is proposed for the initial operation and for definitive reconstruction.**

INDEX WORDS: Short colon, congenital; pouch colon, congenital; anorectal malformations, high; cloaca.

THE ASSOCIATION of a high-type anorectal malformation with a pouchlike dilatation of a shortened colon was first described by Trusler et al in 1959.[1] Since then, a number of case reports and a few series have appeared in the literature. Noteworthy is the fact that most of the large series have been from India;[2-11] only a few cases or small series have been reported elsewhere.[12-22] With this unusual malformation, a varying length of the colon is replaced by a dilated pouch that, almost invariably, has a wide high fistulous communication with the genitourinary tract. This anomaly is frequently associated with a cloaca.

MATERIALS AND METHODS

From January 1986 to December 1990, 41 patients with a congenital pouch colon anomaly associated with anorectal agenesis were managed by the Department of Pediatric Surgery, Kalawati Saran Children's Hospital, New Delhi, India. During this period, 458 infants with anorectal malformations were admitted for treatment. These included 269 patients with a high or intermediate variety of anorectal malformations and 189 patients with low anorectal malformations. Thus, infants with congenital pouch colon comprised 9% of all and 15.2% of high anorectal malformations.

Of the patients with congenital pouch colon, 32 were male and nine were females (male:female ratio, 3.55:1). All the boys and seven of the girls presented in the neonatal period. Two girls presented at the age of 3 and 6 months, respectively.

Clinical and radiological suspicion of the presence of this anomaly was confirmed during primary surgery in all 41 patients. The four group types described by Narasimharao et al[7] were used to categorize the cases (Fig 1).

Type I. Normal colon is absent and the ileum opens directly into the colonic pouch.

Type II. The ileum opens into a short segment of cecum, which then opens into the colonic pouch.

Type III. Significant normal colon (7 to 8 cm in the newborn) is present between the pouch and the ileum.

Type IV. Near-normal colon, with only the terminal portion (rectum and sigmoid) converted into a pouch.

Primary Operation

The initial operation was performed using a transverse suprapubic incision extending upward and laterally on the left side, for 3 to 4 cm. For cases in which the anomaly was not suspected preoperatively, the incision made for the sigmoid colostomy was extended after study of the operative findings.

Before 1988, a window colostomy of the pouch with or without disconnection of any genitourinary fistula was performed in patients with a type I or II pouch colon malformation. In a few infants in whom the fistula was easily accessible, division and ligation of the fistula along with an end-colostomy of the pouch was performed. Since 1988, these procedures were performed only in patients who were ill and had septicemia at the time of admission, with gross abdominal distension or with other severe congenital anomalies. Infants in good general condition and without associated life-threatening anomalies were treated by mobilization of the colonic pouch, division of the fistula to the genitourinary tract, subtotal excision of the pouch along its antemesenteric border, and tubular colorraphy with end-colostomy in the left lower quadrant of the abdomen (Fig 2). In one of the two girls with a type I pouch colon accompanying a persistent cloaca, a cutaneous vesicostomy was performed in addition to disconnection of the fistula, tubular colorraphy, and end-colostomy.

For infants with a type III or IV anomaly, the initial operation included disconnection of the fistula with an end-colostomy of the pouch; disconnection of the fistula, and excision of the pouch with end colostomy; or a loop colostomy proximal to the pouch. In the

From the Department of Pediatric Surgery, Lady Hardinge Medical College and Kalawati Saran Children's Hospital, New Delhi, India.
Date accepted: November 10, 1992.
Address reprint requests to Rajiv Chadha, MD, G-123, Vikaspuri, New Delhi-110018, India.

0022-3468/94/2903-0020$03.00/0

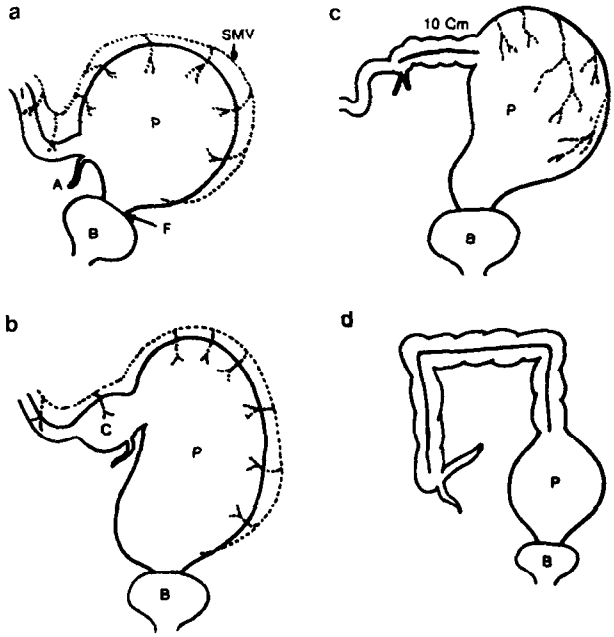

Fig 1. The four types of congenital pouch colon associated with anorectal agenesis. (a) type I, (b) type II, (c) type III, (d) type IV. P, colon pouch; A, appendix; I, ileum; B, bladder; F, fistulous communication into the bladder; C, cecum; SMV, blood supply from superior mesenteric vessels. (Reprinted with permission.[7])

two patients who presented with peritonitis caused by perforation of the pouch, exteriorization of the perforation (as a window colostomy) was performed along with flank drainage.

Postoperatively the patients were examined to detect any associated congenital malformations. All patients had radiographs of the chest and spine. Twenty-four patients had an echocardiogram and abdominal ultrasonogram; 18 had a micturating cystourethrogram (MCU).

At the age of 1 to 3 months, after documentation of improvement in the child's general status and nutrition, four patients with a

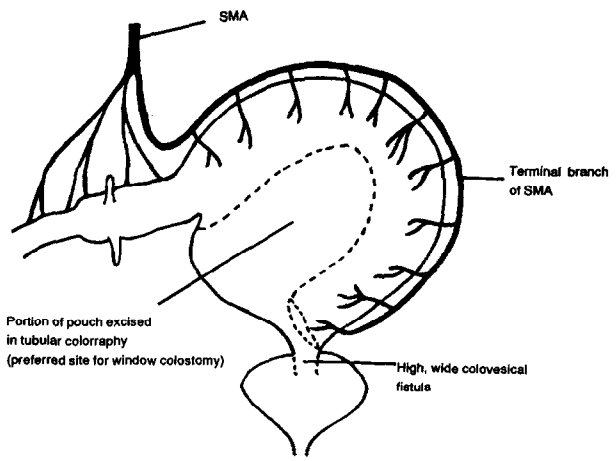

Fig 2. The anatomy and vascular supply of a typical type II pouch colon malformation. Dashed line indicates incision for subtotal excision of the pouch and tubular colorraphy. If a window colostomy is necessary, the best site is the portion of the pouch that would be excised when tubular colorraphy is performed at a later stage. SMA, superior mesenteric artery.

window colostomy of the pouch underwent division-ligation of the fistula, subtotal excision of the pouch, and tubular colorraphy.

Definitive Operation

The definitive operation was performed when the child was thriving, with adequate weight gain, generally at 4 to 18 months of age. Thirteen of the 41 patients have undergone reconstruction (10 boys and three girls). In all cases the posterior sagittal route was used, as described by deVries and Peña,[23] with an additional abdominal component that is mandatory with this anomaly. A wide bore rubber tube was placed through the pelvis during the initial posterior sagittal dissection. During the abdominal phase of the surgery, the particular procedure depended on the operative findings. The end of the tubularized colonic pouch or appropriately tapered proximal bowel was sutured to the rubber tube and brought to the proposed site for the anus. Suturing of the muscle layers and anoplasty were then performed as described by deVries and Peña.[23]

RESULTS

All 41 patients with congenital pouch colon had a high variety of anorectal malformation and presented with absence of the anal opening. In 16 boys and six girls there was gross abdominal distension, with a tense shiny abdominal wall and dilated superficial veins. Fecaluria with passage of meconium and flatus through the urethra was present in 18 boys (56%). In three of the 18, the fecaluria was gross and severe, and abdominal distension was absent. Similarly, in three girls, adequate decompression of the colonic pouch occurred through the vaginal or cloacal fistula, and abdominal distension was not marked.

The typical radiological feature on an invertogram or a plain radiograph of the abdomen was an enormous air-fluid level, occupying more than 50% of the width of the abdomen (Figs 3 and 4). This was present in 27 patients (66%); 24 of them were found to have a type I/II pouch colon malformation, and three had a type III anomaly. Gas within the bladder was observed in 10 patients (24%). The typical radiological features were absent in six patients who had a type I/II pouch colon and a very wide genitourinary fistula, in two with a type I anomaly who had perforation of the pouch with pneumoperitoneum, and in six with a type III/IV pouch colon malformation.

The typical findings during surgery are shown in Figs 1 and 2. Although in the majority of cases the colonic pouch was enormously distended with meconium and flatus, it was small and thick-walled in six patients in whom the fistulous communication was very wide. In all patients the colonic pouch lacked haustrations, appendices epiploicae, or tenia coli, and there was an abrupt transition from the distended pouch to proximal bowel of normal caliber. In type I/II cases, the ileum or proximal colon was seen to enter the pouch from right to left; and in patients with

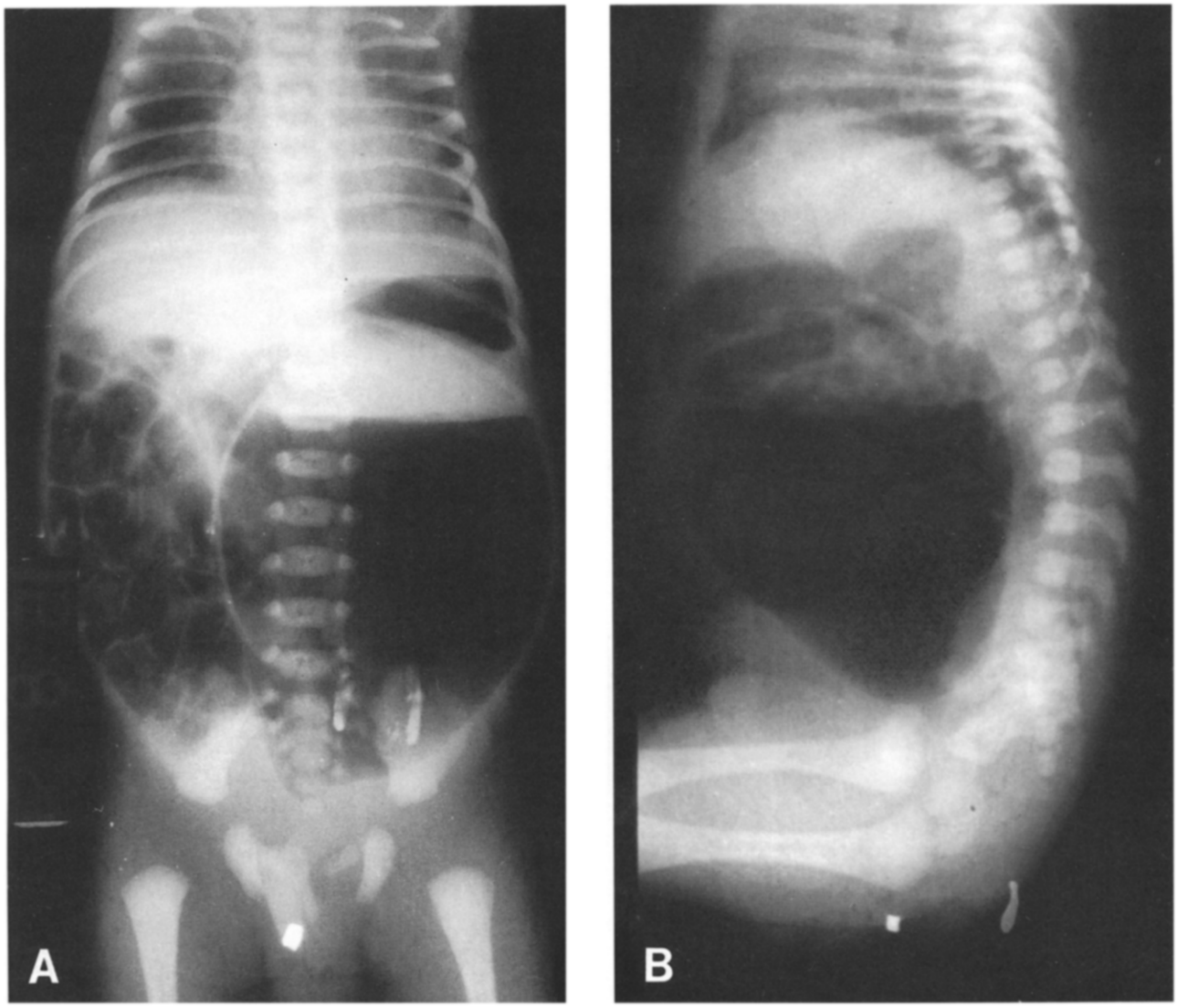

Fig 3. (A) Anteroposterior and (B) lateral invertogram of an infant with a type I pouch colon malformation. Note the distended colonic pouch lying on the left side of the abdomen and occupying more than 50% of the width of the abdomen.

a type I anomaly, the ileum often opened into the pouch at a low position, close to the genitourinary fistula.

Table 1 shows the incidence of the various subtypes. Twenty-nine of the 32 boys had an associated colovesical fistula. In two of them (one with type II, one with type III), the fistula was replaced by a short fibrous cord extending to the posterior wall of the bladder. In one boy with a type IV malformation, the colonic pouch ended blindly in the pelvis. A high colovaginal fistula was present in six of the nine girls; the other three had a persistent cloaca with a colocloacal fistula.

A detailed examination of the vascular supply was performed in 15 patients. In 12 (all type I/II) the inferior mesenteric artery was absent, with only a lengthened hypertrophied terminal branch of the superior mesenteric artery supplying the pouch (Fig 2). In three patients (type III/IV) the pouch was supplied by the inferior mesenteric artery.

Histopathological examination of the tissue resected from the colonic pouch was performed for eight patients. All eight specimens had normal colonic mucosa and ganglion cells. The associated malformations for the patients in this series are noted in Table 2.

Table 3 shows the various procedures performed initially. There were 11 deaths after the initial procedure (27% mortality). The causes of death included (1) suture-line leak and peritonitis in three patients who had undergone tubular colorraphy, (2) postoperative pneumonia and septicemia in three patients, (3) associated esophageal atresia with tracheoesophageal fistula and severe pneumonia in one child, and (4) associated congenital heart disease in one patient.

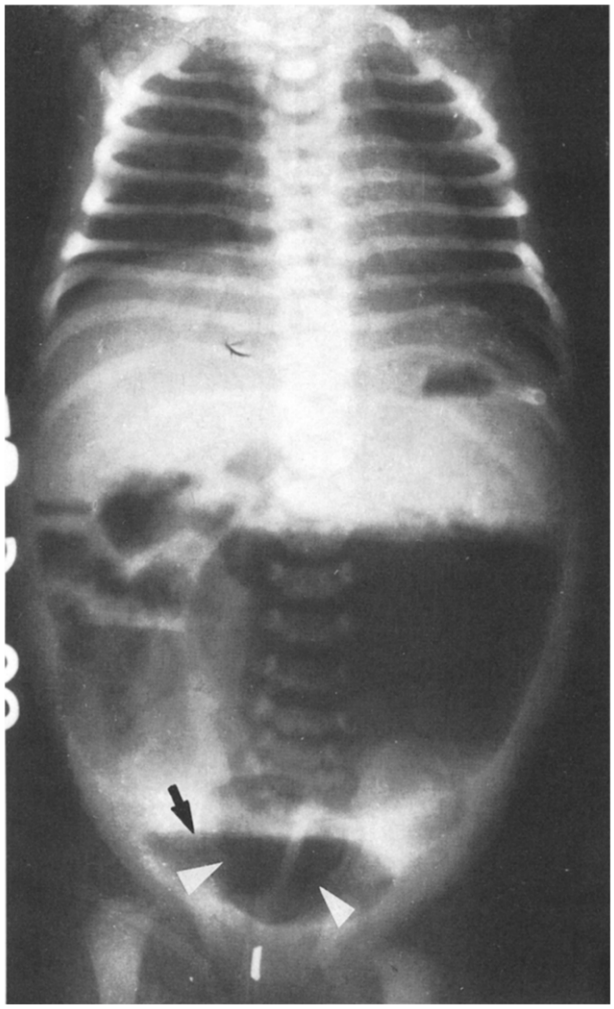

Fig 4. Anteroposterior invertogram of a girl with a type I pouch colon associated with a persistent cloaca. Note the huge air-fluid area in the colonic pouch and the smaller air-fluid areas in the bladder (arrow) and the two halves of the septate vagina (arrowheads).

One patient had postoperative adhesive small intestinal obstruction 3 weeks after surgery and died before reexploration. Two children with a window colostomy without disconnection of the fistula were admitted 2 and 3 months (respectively) after initial surgery because of malnutrition and urinary tract sepsis; they died shortly after.

Six patients with window colostomies had complications related to the colostomy stoma. Revision of the colostomy was performed because of stomal stenosis in four and prolapse in two.

Table 1. Incidence of Subtypes of Pouch Colon Malformation

Type	No. of Males	No. of Females
I	16	5 (2 with persistent cloaca)
II	9	2
III	5	2 (1 with persistent cloaca)
IV	2	0
Total	32	9

Table 2. Associated Malformations

Malformation	No.
I. Gastrointestinal tract	
Double appendix	5
Tiny appendix	2
Meckel's diverticulum	4
Malrotation	2
II. Genitourinary tract	
Vesicoureteric reflux	14
Unilateral absent kidney	4
Hydronephrosis	4
Unilateral undescended testis	2
Cloacal anomaly	3
Septate vagina	2
Hypospadias	1
III. Cardiorespiratory system	
Ventricular septal defect	1
Cyanotic congenital heart disease	1
Esophageal atresia with tracheoesophageal fistula	1
IV. Skeletal system	
Partial sacral agenesis	4
Multiple vertebrae, hemivertebrae	2
Talipes equinovarus	1

The various operative procedures used for the 13 patients who have undergone definitive repair are shown in Table 4. There was one death in the early postoperative period, which was caused by aspiration pneumonia. Other postoperative complications included partial dehiscence of the perineal wound because of wound infection (two patients) and minor wound infections (three patients).

DISCUSSION

Congenital pouch colon in association with imperforate anus is an unusual malformation that appears

Table 3. Primary Operative Procedure

Type of Malformation	Procedure	No.	Postoperative Mortality
Type I & II (32)	Window colostomy alone	11	3
	Window colostomy with flank drainage	2	1
	Window colostomy after fistula disconnection	1	0
	Disconnection of fistula with end-colostomy	1	0
	Fistula disconnection, subtotal excision of pouch and tubular colorraphy	17	5
Type III (7)	Window colostomy alone	2	0
	Disconnection of fistula with end-colostomy	2	0
	Excision of pouch with end-colostomy	3	1
Type IV (2)	Loop sigmoid colostomy proximal to pouch	2	1

Table 4. Definitive Operative Procedures (13 Patients)

Status at Time of Definitive Operation	Definitive Operation (Combined Posterior Sagittal and Abdominal Approach)
Tubular colorraphy and end-colostomy (9) (Type I/II anomaly)	Disconnection of end-colostomy; pull through of previously tapered colon (2) Retubularization to tube of 2-2.5 cm diameter; pull-through of retubularized colon (4) (with protective ileostomy in 1 case) Excision of redilated colonic pouch; pull-through of proximal colon after tapering (2) Excision of small redilated pouch; pull-through of proximal ileum (1)
Window colostomy without disconnection of fistula (2) (Type I anomaly)	Disconnection of fistula, subtotal excision of pouch, tubular colorraphy, and pull-through of tubularized colon (2) (with protective ileostomy in 1 case)
End-colostomy of small pouch (1) (Type III anomaly with persistent cloaca)	Excision of pouch, one stage cloacal reconstruction including pull-through of proximal colon after tapering
Sigmoid loop colostomy proximal to pouch (1) (Type IV anomaly)	Excision of colostomy, disconnection of fistula, pull-through of colon proximal to colostomy

to be most common in India. Of note is that even in India there is a marked regional variation in incidence, with virtually all case reports and series originating from centers in Northern India.[2-9,11] In Northern India this malformation is fairly common; it comprises 4.38%[3] to 8.3%[7] of all and 10%[3] to 26%[11] of high anorectal malformations.

The marked regional variation in incidence is difficult to explain. It is possible that a racial, dietary, or environmental factor may be responsible. However, it appears that the incidence is sporadic, and there is no evidence of familial inheritance.

Although most earlier reports referred to this condition as "congenital short colon associated with imperforate anus," Narasimharao et al[7] redefined it as "pouch colon syndrome." We believe the best nomenclature is "congenital pouch colon associated with anorectal agenesis," which appears to be the most unambiguous description of the entity.

The anatomy of the typical pouch colon malformation is shown in Fig 2. Although a high wide fistula with the genitourinary tract is the norm, occasionally the pouch may end blindly or terminate as a fibrous cord.[8,9,11,13] In females, the fistula can open into the vestibule[4] or perineum.[12]

Associated malformations, mainly involving the gastrointestinal and genitourinary tracts, have been reported frequently. These include absence of the appendix in patients with a type I anomaly, duplication of the appendix,[1,4,6,7,10,21] and malrotation of the gut.[3,4,7,21] Vesicoureteric reflux appears to be very common.[7] In infant girls, a persistent cloaca is frequently present, often associated with a bicornuate or duplicated uterus and a septate vagina.[1,3,4,7] Partial or complete sacral agenesis has also been reported.[7]

Embryology

According to traditional embryological doctrine, the cloaca is partitioned between the 4-mm (4-week) and 16-mm (6-week) stage, by downward growth of the urorectal or cloacal septum.[24] High anorectal malformations are caused by failure of the urorectal septum to complete cloacal septation, leading to a rectourinary fistula in the male or a rectocloacal fistula in the female.[25]

Recently, Bourdelat et al[26] studied the arterial supply of the anorectal region in human embryos and fetuses, and suggested vascular ischemia as a basis for the occurrence of anorectal malformations. They detected early anorectal vascularization by end-arteries without anastomoses, with the inferior mesenteric artery via the superior rectal artery, ensuring virtually the entire development of the hindgut. Dickinson[13] also proposed that a vascular compromise in the region of the hindgut during early intrauterine development could account for pouch colon malformation. We believe that the hypotheses of vascular compromise as suggested by Bourdelat et al[26] and Dickinson[13] can be extended to explain several of the anatomic features seen with pouch colon malformations. A vascular impairment in the region of the descending urorectal septum and adjacent cloaca at an early stage in cloacal partitioning (around 4 weeks' gestation) may interfere with division of the cloaca. This vascular compromise could involve the end-arteries supplying the adjacent developing hindgut and thus interfere with its development. The extent of this vascular impairment can vary, accounting for the different subtypes of this malformation. If only the terminal branches of the

inferior mesenteric artery are involved, a type IV pouch colon anomaly would eventually result, whereas involvement of more proximal branches would produce a type III malformation. If the main trunk of the inferior mesenteric artery is involved, a type II malformation would be the outcome, whereas additional involvement of the most distal branches of the superior mesenteric artery can explain a type I anomaly. Regardless of the extent of vascular occlusion, its presence during early cloacal partitioning explains the occurrence of a wide fistula high on the posterior wall of the bladder in males and the high incidence of persistent cloaca in females. In the later months of pregnancy, the abnormal, imperfectly developed, shortened portion of the hindgut undergoes distension by accumulation of meconium, leading to the pouchlike dilatation of this segment, with an abrupt transition to proximal bowel of normal caliber.

Other striking anatomic features seen in patients with a type I/II pouch colon anomaly are the left-sided location of the pouch, with the ileum or proximal colon entering it from right to left, and the vascular supply from a terminal branch of the superior mesenteric artery along its left or lateral border (Fig 2). A study of normal intestinal rotation may provide an explanation. Normal intestinal rotation involves a 270° counterclockwise rotation of both the proximal duodenojejunal and distal cecocolic loops around the axis of the superior mesenteric artery during return of the midgut from its ventral extracoelomic herniation (between 10 and 12 weeks of gestation).[27-30] Rotation of the cecocolic loop places the ileocecal junction in the right lower quadrant of the abdomen. It is likely that in cases with a type I/II pouch colon, the shortened aborted hindgut is unable to herniate into the extracoelomic space, and later, because of its extremely short length, cannot undergo normal rotation.

Management

The variety of operative procedures performed on patients with a pouch colon malformation testifies to the lack of a standardized approach to the diagnosis and management of this condition.

In earlier reports, a window or side colostomy with or without disconnection of the fistula was often performed initially. However, several reports as well as our experience indicate that it is very frequently associated with stomal stenosis,[1,7,19,20] prolapse of the colostomy, often with eversion of the entire colonic wall[2,6,7,20] as well as poor functioning and emptying.[1,2,7,19,20,22] Moreover, if the fistula with the genitourinary tract is not divided, persistent contamination of the urinary tract will occur, which is especially dangerous in view of the high incidence of associated vesicoureteric reflux.[7] End-colostomy after division-ligation of the associated fistula avoids contamination of the genitourinary tract and complications such as prolapse of the colonic wall.[2,21] However, inadequate motor function and peristalsis with poor emptying of the pouch still pose problems. Despite these limitations, a window colostomy or end-colostomy still has a role in the immediate management of poor-risk patients with a type I to III pouch colon anomaly.

Excision of the pouch, with a pull-through at the same stage or as a secondary procedure, has been advocated.[1-4,7,8,10,13] However, in patients with a type I/II pouch colon it is important to preserve as much of the colon as possible because of its absorptive function, and we believe that excision of the colonic pouch should be used only if this segment clearly is not viable.

In the first report on this subject, Trusler et al[1] suggested that, to preserve absorptive colonic surface as well as improve motility and peristalsis of the colonic pouch, a plastic procedure could be used to convert the sac into a longer, narrow, tubular structure. This suggestion was advocated by Singh and Pathak,[2] and subsequently several investigators reported performing subtotal excision of the pouch and tubular colorraphy.[4-7,11,15,19,21,22] Luzatto et al[22] recently reported use of the posterior sagittal approach (as advocated by deVries and Peña[23]) to place the tapered bowel within the sphincter complex. Our own experience also suggests that the posterior sagittal approach offers excellent exposure for accurate placement of the segment of bowel to be pulled through to its correct anatomic location.

Figure 5 is an outline of a proposed plan for short- and long-term management of patients with a pouch colon malformation. However, a few additional points must be emphasized. An important consideration is the extent of tailoring to be performed during tubular colorraphy. Early in our experience we tailored the pouch to a tube of approximately 2.5 cm in diameter. However, in many patients we found that, at the time of definitive surgery, the tubularized colon had again undergone considerable dilatation, necessitating retailoring and retubularization of the entire length. This may be because the pouch colon segment is structurally and functionally abnormal. Our recent experience indicates that this complication can be avoided by more radical tubularization of the pouch during the initial procedure, to a tube of approximately 1.5 cm in diameter.

We advocate performing a protective loop ileostomy at the time of pull-through of the tubularized colon (or normal proximal bowel) in patients with types I to III pouch colon malformations. We have not performed an ileostomy during the initial proce-

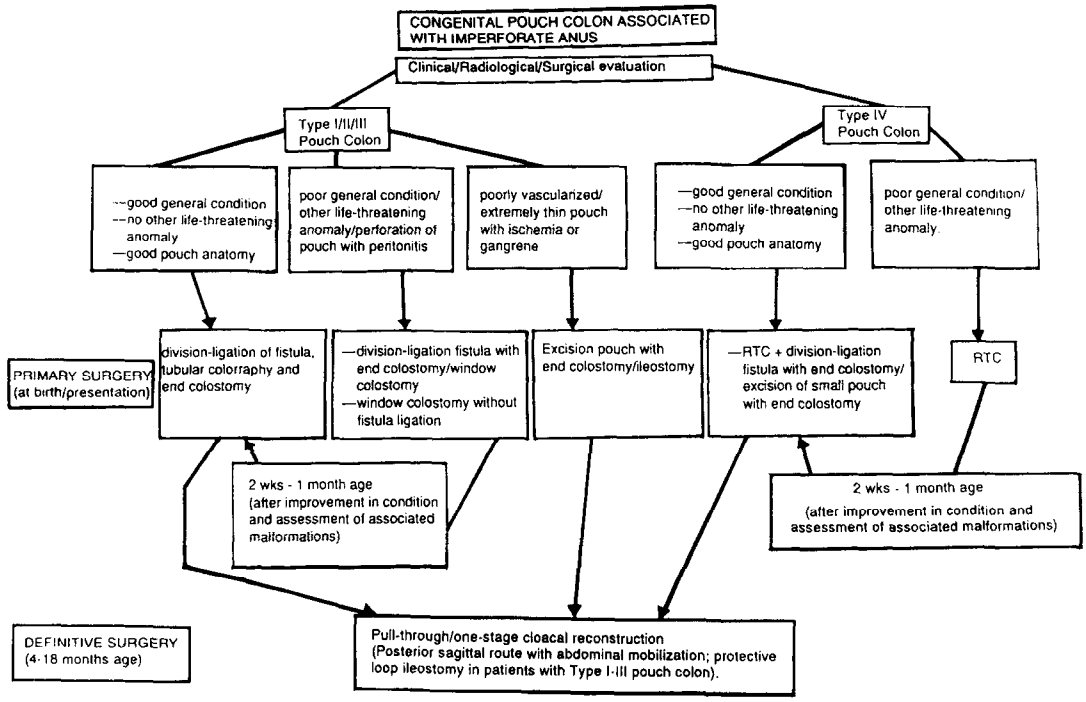

Fig 5. Plan for the surgical management of the various subtypes of pouch colon malformation associated with anorectal agenesis. RTC, right transverse colostomy.

dure or as an intermediate procedure before pull-through because of difficulties in managing ileostomy losses for long periods. An additional consideration for performing the ileostomy only at the time of definitive surgery is because a type I/II colonic pouch has a tenuous blood supply from a single terminal branch of the superior mesenteric artery and, even after complete mobilization of the tubularized colon, it is often difficult to bring it down to the perineum. Constructing the ileostomy after complete mobilization of the colonic tube ensures that it can be sited sufficiently proximal to not interfere with either the mobilization or vascularity of the bowel to be pulled through. Similarly, although a sigmoid colostomy is often technically possible, we advocate a protective transverse colostomy in patients with a type IV pouch colon because a gap of several centimetres must be bridged from the end-colostomy to the proposed site for the anus.

In conclusion, we believe that with a standardized approach to the diagnosis and management of this unusual malformation, future studies will show much decreased morbidity and mortality. Further study of this anomaly is warranted, particularly with regard to its unique epidemiology and geographical distribution. Detailed assessment of continence in patients who have undergone pull-through surgery is also required. Additional detailed histological and physiological studies of tissue obtained from the colonic pouch may help us understand the unique anatomic and physiological characteristics of the pouch colon segment.

REFERENCES

1. Trusler GA, Mestel AL, Stephens CA: Colon malformation with imperforate anus. Surgery 45:328-334, 1959

2. Singh S, Pathak IC: Short colon malformation with imperforate anus. Surgery 71:781-786, 1972

3. Singh A, Singh R, Singh A: Short colon malformation with imperforate anus. Acta Paediatr Scand 66:589-594, 1977

4. Wakhlu AK, Tandon RK, Kalra R: Short colon associated with anorectal malformations. Indian J Surg 44:621-629, 1982

5. Yadav K, Narasimharao KL: Primary pull-through as definitive treatment of short colon associated with imperforate anus. Aust N Z J Surg 53:229-230, 1983

6. Kalani BP, Sogani KC: Short colon associated with anorectal agenesis: Treatment by colonorraphy. Ann Pediatr Surg 1:83-85, 1984

7. Narasimharao KL, Yadav K, Mitra SK, et al: Congenital short colon with imperforate anus (pouch colon syndrome). Ann Pediatr Surg 1:159-167, 1984

8. Gupta JP, Narayan AK: Non-functioning short colon with anorectal malformation. J Indian Med Assoc 83:69-70, 1985

9. Ashraf SM, Khan MA, Raza MH: Pouch colon syndrome. Indian Pediatr 26:81-82, 1989

10. Gopal G: Congenital recto-vaginal fistula with colonic reservoir. Indian J Surg 40:447-449, 1989

11. Wardhan H, Gangopadhyay AN, Singhal GD, et al: Imperforate anus with congenital short colon (pouch colon syndrome). Pediatr Surg Int 5:124-126, 1990

12. El Shafie M: Congenital short intestine and cystic dilatation of the colon associated with ectopic anus. J Pediatr Surg 6:76, 1971

13. Dickinson SJ: Agenesis of the descending colon with imperforate anus. Am J Surg 113:279-281, 1967

14. Blunt A, Rich GF: Congenital absence of the colon and rectum. Am J Dis Child 114:405-406, 1967

15. Chiba T, Kasai M, Asakura Y: Two cases of coloplasty for congenital short colon. Arch Jpn Chir 45:40-43, 1976

16. Li G, et al: Report of 2 cases of congenital cystic megacolon. New Med Wuhan (for pediatr surg) 4:88-89, 1978

17. Yadav K: Short colon associated with the prune belly syndrome. Int Surg 64:83-85, 1979

18. Li Z, et al: Congenital atresia of anus with short colon malformation. Chin J Pediatr Surg 2:30-32, 1981

19. Vaezzadeh K, Gerami S, Kalani P, et al: Congenital short colon with imperforate anus: A definitive surgical cure. J Pediatr Surg 17:198-200, 1982

20. Dénes J, Ziszi K, Bognár M, et al: Congenital short colon associated with imperforate anus (Zachary Morgan syndrome). Acta Pediatr Hung 25:377-383, 1990

21. Yuejie W, Rong D, Guie Z, et al: Association of imperforate anus with short colon: A report of eight cases. J Pediatr Surg 25:282-284, 1990

22. Luzatto C, Zanardo V, Guglielmi M: Imperforate anus with congenital short colon: Combined abdominal and posterior sagittal approach. Pediatr Surg Int 5:375-376, 1990

23. deVries PA, Peña A: Posterior sagittal anorectoplasty. J Pediatr Surg 17:638-643, 1982

24. Stephens FD: Congenital Malformations of the Urinary Tract. New York, NY, Praeger, 1983, pp 3-14

25. Bill AH Jr, Johnson RJ: Failure of migration of the rectal opening as the cause for most cases of imperforate anus. Surg Gynecol Obstet 106:643-651, 1958

26. Bourdelat D, Labbé F, Pillet J, et al: A study in organogenesis: The arterial supply of the anorectal region in the human embryo and fetus. Surg Radiol Anat 10:37-51, 1988

27. Gardner CE Jr, Hart D: Anomalies of intestinal rotation as a cause of intestinal obstruction: Report of two personal observations; review of 103 reported cases. Arch Surg 29:942-981, 1934

28. Snyder WH Jr, Chaffin L: Embryology and pathology of the intestinal tract: Presentation of 40 cases of malrotation. Ann Surg 140:368-380, 1954

29. Filston HC, Kirks DR: Malrotation—the ubiquitous anomaly. J Pediatr Surg 16:614-620, 1981

30. Moore K: The Developing Human—Clinically Oriented Embryology. Philadelphia, PA, Saunders, 1973, pp 181-186