Motility After Small Bowel and Colonic Surgery

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 Surgery of the small intestine and colon is commonly performed in children for a variety of indications ranging from congenital anatomic abnormalities to need for enteral feeding access to underlying motility disorders. Under most circumstances, non-emergent operations allow time for a multidisciplinary team approach between surgeons and gastroenterologists to devise a thorough preoperative diagnostic strategy. Unfortunately, abdominal catastrophes such as malrotation with volvulus often preclude the luxury of time before surgery necessitating a strong relationship between surgeon and gastroenterologist to address the potential consequences of such an event. In both cases, the motility of the small bowel and colon remains a critical feature that often predicts the success of an operation and, most importantly, the prognosis of the patient. This chapter aims to address several of the more prevalent motility disorders observed in children after small bowel and colonic surgery.

Small Bowel Motility After Resection

 Resection of short or long segments of the small bowel may be necessary for different indications including *surgical emergencies* such as bowel ischemia or necrosis from volvulus and perforation; *congenital anomalies* such as intestinal atresia, malrotation, and gastroschisis; or *acquired etiologies* encompassing stricturing Crohn's disease, ulcerative colitis, severe necrotizing enterocolitis, intestinal pseudoobstruction, or abdominal trauma. New advances in intestinal rehabilitation

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such as home TPN, lipid solutions, frequent small bowel bacterial decontamination, and new central line technology that decrease the number of line infections dramatically have changed the prognosis of infants after a small bowel resection. Preservation of bowel length, particularly the small intestine, is critical to insure adequate absorption of nutrients, fluids, and electrolytes but is contingent on circumstances such as extent of the necrosis or ischemia. The consequences of a more extensive resection of small bowel include symptoms such as frequent diarrhea, malnutrition, and bloating due to bacterial overgrowth and may result in the need for parental nutrition with its associated complications.

Small intestinal resections are classified into three categories based on length of residual small bowel: short resection with 100–150-cm length remaining, large resection with 40–100 cm remaining, and massive resection with 40 cm or less remaining. In general, massive resections particularly in the context of an absent ileocecal valve are associated with inability to wean completely from parenteral nutrition [1]. The absence of ileocecal valve has been associated with increased diarrhea and small bowel bacterial overgrowth $(SBBO)$.

 While mucosal adaptation has been extensively studied, there is a paucity of data regarding changes in motility after small intestinal resection. A better functional outcome is associated with proximal compared to distal resection, which may be related to both the adaptive capacity and intrinsic properties of the jejunum and ileum. Adaptation involves all layers of the bowel wall, including intestinal smooth muscle. The intestinal smooth muscle is coordinated by both hormonal and neuronal components which regulate the transit of intestinal contents through the gastrointestinal tract [2]. Activation of this complex circuitry allows changes in the peristaltic reflex to modulate the intestinal motility pattern from propagative to segmenting. This is accomplished through a complex integration of signals that trigger a jejunal and ileal break mechanism in response to nutrients, most notably fats. Mediators involved in this response include peptide YY, chemosensitive afferent neurons, noradrenergic

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nerves, myenteric serotonergic neurons, and opioid neurons [3]. Following proximal resection of small bowel, for example, it has been demonstrated that the postprandial motilin response is decreased, whereas transient increases in neurotensin and peptide YY have been noted after distal resection $[4]$.

 After intestinal loss, a combination of shorter bowel length and disruption of normal physiological mechanisms may lead to poor absorption and malnutrition. Increased contractile response and proliferative changes in intestinal smooth muscle cells may contribute to the compensatory adaptive mechanism to slow intestinal transit and improve nutrient absorption. While the cellular mechanism for this process is not well defined, mechanisms such as epidermal growth factor receptor signaling have been shown to play a role in adaptation of the smooth muscle cellular compartment $[2]$.

 Little is known about changes in the migrating motor complex (MMC) after resection. Animal studies often reveal conflicting results with a broad spectrum of motility changes depending on the extent and location of resection. For example, after extensive distal small bowel resection, postoperative changes such as decreased MMC velocity and longer intervals between MMCs during fasting with slight recovery of propagation frequency in the chronic phase have been observed $[5, 6]$. Findings such as shorter phase I duration and discoordinate clustered MMC activity have also been seen using the same model $[7]$. There are very limited motility studies in humans after small bowel resection $[8-10]$. With extensive distal resection, motility changes include shorter duration and more frequent MMCs as well as a reduction in phase 2 activity; however, limited ileal resection does not result in detectable manometric changes of jejunal motility [9]. The postprandial motor response is not well defined, but appears to be shorter in patients after resection [10].

Short Bowel Syndrome Perioperative Evaluation

 The goal of surgery for patients with short bowel syndrome include maximizing intestinal absorption, improving motility and transit of the dilated aperistaltic segments, as well as delaying intestinal transit time in some cases. Laparotomy or laparoscopy is also required in some cases to close stomas or address causes of obstruction such as abdominal adhesions [11].

 A thorough and focused evaluation must be performed to determine the best surgical option in patients with short bowel. Perioperative evaluation may include assessment of intestinal length and caliber, motility, and intestinal transit. An upper gastrointestinal series with small bowel follow, for instance, can determine bowel anatomy and identify the

presence of obstruction leading to possible adhesiolysis or remodeling of an anastomosis [12]. Determination of intestinal transit can also be assessed to some extent with an upper gastrointestinal series; however, the study has several limitations. First, it does not quantitatively evaluate motility. In addition, the chemical composition of the contrast itself may alter motility giving a false impression of the intestinal transit. The authors believe that antroduodenal and colonic manometry are crucial in the study of these patients. Unfortunately, motility studies are not systematically used in patients with short bowel syndrome, especially before operative management. The preoperative value of colonic and antroduodenal manometry in differentiating peristaltic versus aperistalsic bowel segments was recently addressed in a case series [13]. In this series, a normal colonic manometry was the basis for preserving continuity of the colon in a patient with short bowel syndrome. In contrast, abdominal distension and feeding intolerance with absent distal colonic motility markedly improved after placement of a left-sided colostomy in a patient with prior gastroschisis and short bowel syndrome (Dr. J. Balint, personal communication) (Fig. [30.1](#page-2-0)).

Short Bowel Syndrome Surgical Approaches

Procedures to Alter Intestinal Transit

 Delaying the intestinal transit time has been recognized as an important mechanism in order to increase absorption and maximize contact of the nutrients in patients with short gut syndrome. Several procedures have been designed for this purpose. For example, creation of intestinal valves by placing a Teflon collar around the circumference of the bowel, or by everting the small bowel mucosa, creating a small intussusceptum can induce proximal dilatation increasing adaptation [14, 15]. Reversed antiperistalsic segments of intestine have also been proposed as an alternative for delaying intestinal transit. The reversed segment is usually short and is placed as distal as possible to prevent obstruction. This procedure has been used in adults with short bowel syndrome with 50% of patients being able to wean off total parenteral nutrition $[16]$. The study was based on previous findings in canine models in which the reversed segment was observed to cause retrograde peristalsis disrupting the motility of the proximal intestine [17]. Colonic interposition has also been used to delay intestinal transit time [18]. However, this study was limited by a small number of patients and lack of perioperative assessment of motility changes.

 Dilation of a segment of small bowel is frequently associated with poor motility and presence of bacterial overgrowth. Therefore, increasing motility of the dilated segment has been an important aim in many types of autologous reconstructive

 Fig. 30.1 Patient with history of gastroschisis resulting in short bowel syndrome with persistent abdominal $distension(a)$ and feeding intolerance after STEP. Antroduodenal manometry demonstrated adequate small bowel motility after STEP (**b**). Absence of motility was shown in the distal colon (c). Subsequent placement of a left-sided colostomy resulted in symptom resolution and tolerance of enteral nutrition. (Courtesy of Drs. Gomez and Burns, Nemours Children's Hospital, Orlando, FL)

b $R₀$

bowel surgery. Tapering enteroplasty reduces the caliber of the bowel lumen, preserving the length, and thereby improving peristalsis $[19, 20]$ $[19, 20]$ $[19, 20]$. The impact of this tapering on the different phases of the MMC or postprandial motility indices is not clear.

Intestinal Lengthening

 Surgical procedures including longitudinal intestinal lengthening and tailoring (Bianchi's LILT) or serial transverse enteroplasty (STEP) were designed to increase the length of the intestine and maximize absorption in patients with short bowel syndrome $[21, 22]$. These procedures are usually performed after a period of intestinal adaptation and not immediately after resection. LILT isoperistaltic bowel lengthening entails longitudinal division of the bowel with isoperistaltic end-to-end anastomosis effectively doubling the length of that portion of the bowel. The STEP procedure involves the sequential linear stapling of the dilated small bowel from alternating directions perpendicular to the long axis of the intestine [22].

 Both LILT and STEP have been shown to successfully result in increased caloric absorption and preserved intestinal motility [23, 24]. After LILT, there is an increased tolerance of enteral feeds, improved growth, and decreased frequency of catheter infections. Significant improvement in stool counts, intestinal transit time, D -xylose absorption, and fat absorption resulting in discontinuation of parenteral nutrition has also been observed $[25, 26]$ $[25, 26]$ $[25, 26]$. After LILT, 55–79 % of the patients are able to wean from parenteral nutrition with survival rates up to 77% [$27, 28$]. Limitations of the LILT procedure include its technical difficulty, involvement of at least one intestinal anastomosis, and risk

to the mesenteric blood supply. It is also best performed if the bowel is symmetrically dilated. Complications such as ileal valve prolapse and recurrent small bowel dilatation have been reported after the operation [24].

 STEP has become widely accepted among pediatric surgeons as it is technically easier to perform than LILT and preserves the natural mesenteric vasculature to the intestine [29]. STEP has been shown to improve weight retention, nutritional status, and intestinal absorptive capacity in an animal model. Its results are comparable to LILT with around 80 % of the patients being able to wean off parenteral nutrition $[27, 30]$. Motility studies performed in a STEP animal model suggest that the MMC phase III is preserved after resection and anastomosis maintaining the amplitude and frequency of small bowel contractions $[22]$. The small bowel motility index was similar to controls. Nonspecific abnormalities observed in both groups included simultaneous or tonic contractions as well as contractions present in only proximal or distal segments. The duration of phase III after octreotide was also increased in STEP animals $[22]$. These findings are difficult to reproduce in the clinical setting especially in patients with severe intestinal ischemia or gastroschisis and baseline abnormal motility even before STEP. After STEP, intestinal motility continues to be affected correlating with feeding intolerance and TPN dependency (Fig. 30.2). Thus, preoperative severe dysmotility is a risk factor for poor outcomes from STEP [31].

Intestinal Transplantation

 Intestinal transplantation has become an increasingly accepted treatment for children with intestinal failure with 3- and 5-year survival rates of 84 % and 77 %, respectively,

 Fig. 30.2 Small bowel and colonic motility in a 4-year-old boy with a medical history of NEC, short bowel syndrome, and post-STEP procedure. (a) Presence of simultaneous contractions in the antrum and small

bowel in the first eight channels. (b) HAPCs in the sigmoid after Bisacodyl stimulation (arrow). (Courtesy of Dr. Carlo Di Lorenzo and Dr. Hayat Mousa, Nationwide Children's Hospital, Columbus, OH)

with most patients becoming independent of TPN [32]. The most frequent cause of intestinal failure is short-gut syndrome (SGS) defined by malabsorption, malnutrition, and growth retardation secondary to extensive loss of intestinal length or functional gut mass [33, [34](#page-7-0)]. Gastroschisis, volvulus, necrotizing enterocolitis, intestinal atresia, chronic intestinal pseudoobstruction, and congenital enteropathy are frequent conditions associated with SGS [32].

 Small bowel or multivisceral organ transplantation is often necessary for children after massive intestinal resection including those with less than 25 cm of small bowel without ileocecal valve, congenital intractable mucosal disorders, persistent hyperbilirubinemia, and diminishing venous access, often associated with recurrent episodes of sepsis [35, [36](#page-7-0)]. The role of performing small bowel motility studies as a gauge to determine whether intestinal transplantation should be undertaken is unclear, but has been proposed as a potential prognostic tool [37]. Most studies have focused on the impact on intestinal motility after transplantation [38].

 After intestinal transplantation, maintenance of intestinal motility with coordinated smooth muscle function and adequate absorptive capability is paramount. Animal models have confirmed that intrinsic nerves are generally preserved after transplantation [39, [40](#page-7-0)]. The consequence of extrinsic denervation from the small bowel may lead to poor functioning of the grafted intestine. In a canine model, for instance, body weight and serum albumin levels remain stable after autotransplantation. However, transplanted animals demonstrated significant defects in fat and D-xylose absorption compared to controls, possibly attributed to overgrowth in fecal flora $[39]$. In a similar model, dogs undergoing autotransplantation experienced rapid intestinal transit compared to short-gut animals which may suggest that adaptive responses of the transplanted intestine may be impaired by neuromuscular injury associated with denervation or ischemia [41].

 Intestinal motility after small bowel transplantation has been studied in children using antroduodenal manometry. Interdigestive phase III motor activity with normal manometric characteristics was seen as early as 3 months posttransplantation in the majority of patients. However, disruption of an orderly MMC was noted across the anastomosis as well as abnormal postprandial motility, which may in part be responsible for abnormal intestinal transit and poor absorption $[38]$. These studies emphasize how little is known about the effect of small bowel transplantation on motility and underscore the need for future prospective research. Because a significant part of graft motility depends on the Cajal cells, particularly in the context of extrinsic denervation, inflammation of the tunica muscularis either by ischemia reperfusion or by frequent episodes of rejection or infections often leads to poor functioning of the graft and presence of bacterial overgrowth $[42]$. In animal

models, small bowel graft, rejection is associated with decreased MMC phase III amplitude and propagation of contractions [43, [44](#page-7-0)].

Roux-en-Y Jejunostomy and Bariatric Surgery

 Roux-en-Y gastrojejunostomy has been employed in both children and adults for a variety of indications including postgastrectomy for peptic ulcer disease, as a component of bariatric surgery, and for jejunal feeding access [41]. The technique limits reflux of bile into the gastric remnant and esophagus. Common postoperative symptoms attributed to secondary dysmotility include abdominal fullness, distension, pain, nausea, and vomiting [45]. These symptoms are likely the result of interrupted slow-wave electrical conduction which occurs after transecting the jejunum resulting in shortened phase III MMC duration and abnormal motor response to meals [46]. The consequence of disruption of the enteric nervous system may include serious conditions such as ascending cholangitis due to stasis of bowel contents in the proximal limb of the roux segment, known as blind-loop syndrome [47].

 It has been shown in both adults and animals that using an "uncut" Roux-en-Y technique may avoid the problems observed with jejunal transection by prolonging the phase III MMC, thereby enhancing digestive clearance [47]. While gastrectomy is uncommon in children, there has been an increase in pediatric gastric surgery to treat obesity particularly in adolescents [48]. Both laparoscopic adjustable gastric banding and laparoscopic Roux-en-Y gastric bypass have been performed in children, but there is a paucity of data examining the effects of these operations on gut motility. Overall, there seems to be an improvement in health-related quality of life based on early studies, which may suggest limited disturbances in motility in these patients [49].

Congenital Diaphragmatic Hernia

Congenital diaphragmatic hernia (CDH) is a developmental defect present in less than 1 of 1000 live births resulting in herniation of abdominal viscera into the chest $[50, 51]$. It is associated with other anatomic malformations in 30 % of the patients resulting in increased mortality [52, [53](#page-7-0)]. Long-term gastrointestinal problems, most notably refractory gastroesophageal reflux disease (GERD), have been described in patients with prior CDH repair [54]. In a recent multivariate analysis, the incidence of GERD was shown to be 39 % immediately after repair and 16 % 12–18 years after repair. Patients with an intrathoracic stomach and patch closure of the diaphragm seemed to demonstrate the most significant reflux symptoms in the early postoperative period $[55]$.

 Reports of intestinal motility disorders in patients with CDH are limited. However, foregut dysmotility has been postulated after CDH repair as evidenced by persistent upper GI symptoms noted in association with abnormal gut fixation seen in nearly 10% of patients [56]. For example, antral hypomotility with low-amplitude and prolonged phase III contractions has been observed after CDH repair manifesting as symptoms of severe gastroesophageal reflux and delayed gastric emptying scintigraphy testing [57].

Gastroschisis

 Gastroschisis is a full-thickness defect in the abdominal wall usually adjacent to the insertion of the umbilical cord with an incidence between 0.4 and 3 per $10,000$ births $[58]$. A variable amount of intestine and abdominal organs may herniate through this defect without the protective covering of the peritoneal sac [59]. Ten percent of infants with gastroschisis develop ischemic injury to the bowel due to vascular insufficiency which may result in intestinal stenosis or atresia [58, [60](#page-8-0)]. Gastroschisis represents one of the major causes of intestinal failure often necessitating consideration of intestinal transplantation. Approximately 40 % of patients with gastroschisis require parenteral nutrition by the age of 4 months and 10% by the age of 2 years [61].

 Patients with gastroschisis tend to have persistent gut dysmotility with symptoms suggestive of intestinal pseudoobstruction $[62]$. Even after repair with adequate bowel length, these patients have evidence of profound feeding problems, increased hospitalizations, and mortality $[63, 64]$. Many of these patients with feeding problems may have neuropathic predominant changes based on antroduodenal manometry (Author RG, unpublished case series). Interestingly, in postnatal autopsy studies, there is no evidence of ganglion cell or generalized myenteric nervous system abnormalities to explain the motility disorders that often accompany cases of gastroschisis [65].

Motility Disorders After Repair of Malrotation and Intestinal Atresia

Malrotation is defined by the absence of midgut rotation before reentering the abdominal cavity during the 12th week of gestation [66]. By this time in embryonic development, the neurons forming the ENS have already migrated from the neural crest to the intestine. Surgical correction (Ladd's procedure) involves division of a fibrous stalk of peritoneal tissue attaching the cecum to the abdominal wall, known as Ladd's bands; widening the small bowel mesentery; appendectomy; and appropriate placement of the colon. Small bowel motility

abnormalities including complete absence of motor activity, low-amplitude or slow-frequency contractions, and slow propagation of phase III of the MMCs have been described after performing a Ladd's procedure for these patients [67]. These manometric abnormalities have been associated in some patients with histological changes such as distended neuronal axon hypoganglionosis or vacuolated nerve tracts in the small bowel $[68]$.

 Intestinal atresia is a frequent cause of bowel obstruction in neonates. Operative management includes resection of the atresia with primary bowel anastomosis, resection with tapering enteroplasty, temporary ostomy with intestinal resection, enterostomy with web excision, and longitudinal intestinal lengthening procedures. After surgical correction, symptoms of adhesive bowel obstruction occur in close to 25 % of the patients with prolonged adynamic ileus in 9 % and enterostomy prolapse in 2% [69]. Prolonged small bowel obstruction due to atresia or malrotation can lead to severe refeeding problems in the neonatal period. Cezard et al. described a form of postobstructive enteropathy (POE) of the apparently normal small intestine segment proximal to the obstruction. POE patients showed significant abnormal peristalsis as characterized by barium and carmine transit times. Small bowel manometric recordings are characterized by an absence or abnormal phase III of the migrating motor complex and decreased motility index of the small intestine above the obstruction [70, [71](#page-8-0)].

Colectomy and Partial Colonic Resection

 Colonic resection in children is reserved for chronic conditions such as refractory ulcerative colitis, Crohn's colitis, familial adenomatous polyposis, severe constipation, Hirschsprung's disease, and debilitating motility disorders such as intestinal pseudoobstruction. Small bowel and residual colonic function is contingent on the region and extent of colonic resection as well as the underlying pathology necessitating surgery. As an example, subtotal colectomy is a surgical option to treat severe cases of constipation associated with colonic dilatation. While extensive resection of colon may accomplish reduction in intestinal transit time, it may not eliminate symptoms of pain and bloating suggesting the possibility of a more generalized motor disorder of the gut [72]. Colectomy in these patients may also be associated with uncontrolled diarrhea and fecal incontinence as well as relapsing constipation [73].

The difficulties associated with subtotal colectomy may be due to the adaptive changes in the MMC resulting in increased anaerobic bacterial colonization of the small intestine [74, [75](#page-8-0)]. Partial colonic resection may alleviate some of symptoms observed after subtotal colectomy particularly if a

 Fig. 30.3 Example of two manometry catheters placed in a retrograde fashion from a colostomy and from the anus. The *top panel* shows the radiology image of the two manometry catheters. The *bottom panel* shows the manometry study. There is evidence of propulsive contrac-

tions proximal to a diverting colostomy (top eight channels in the manometry tracing) and absent motility in the distal four channels in the distal colonic segment

performed in conjunction with preoperative motor assessment including Sitz markers, scintigraphy, and antroduodenal and colonic manometry [75–77].

 In patients with refractory constipation and colonic dilatation, colonic and antroduodenal manometry may be key diagnostic tests to determine the optimal surgical approach [77–79]. In the absence of demonstrable colonic motility, a decompressive ileostomy or proximal colostomy for several months may allow improvement in the degree of colonic dilatation with return of some degree of motor function in the distal, diverted colon [77, [79](#page-8-0)]. Performing a subsequent colonic manometry study after a diverting ileostomy or colostomy may allow a more objective surgical decision between ostomy takedown and reanastomosis alone versus reanastomosis combined with partial resection of colon particularly in the context of adequate small bowel motility (Fig. 30.3). A permanent ileostomy may be indicated in the context of persistently absent colonic high-amplitude propagating contractions (HAPCs) particularly in association with abnormal small bowel motility [77].

Summary

 The need for small bowel and colonic surgery for a variety of indications is a common occurrence in children. The impact of operative manipulation and interventions on subsequent gut motility may have serious implications in terms of the functional capacity of the remaining intestine to effectively absorb nutrients without gastrointestinal symptoms. Thus, motility testing in children whether performed in the preoperative or

postoperative phase of management may play a significant role in the surgical decision-making process. Future studies are needed to better discern the underlying mechanisms responsible for motility problems observed after small intestine and colonic surgery.

References

- 1. Goulet O, Ruemmele F, Lacaille F, et al. Irreversible intestinal failure. J Pediatr Gastroenterol Nutr. 2004;38:250–69.
- 2. Martin CA, Bernabe KQ, Taylor JA, et al. Resection-induced intestinal adaptation and the role of enteric smooth muscle. J Pediatr Surg. 2008;43:1011–7.
- 3. Van Citters GW, Lin HC. Ileal brake: neuropeptidergic control of intestinal transit. Curr Gastroenterol Rep. 2006;8:367–73.
- 4. Thompson JS, Quigley EM, Adrian TE. Factors affecting outcome following proximal and distal intestinal resection in the dog: an examination of the relative roles of mucosal adaptation, motility, luminal factors, and enteric peptides. Dig Dis Sci. 1999;44:63–74.
- 5. Uchiyama M, Iwafuchi M, Matsuda Y, et al. Intestinal motility after massive small bowel resection in conscious canines: comparison of acute and chronic phases. J Pediatr Gastroenterol Nutr. 1996;23:217–23.
- 6. Uchiyama M, Iwafuchi M, Ohsawa Y, et al. Intestinal myoelectric activity and contractile motility in dogs with a reversed jejunal segment after extensive small bowel resection. J Pediatr Surg. 1992;27:686–90.
- 7. Quigley EM, Thompson JS. The motor response to intestinal resection: motor activity in the canine small intestine following distal resection. Gastroenterology. 1993;105:791–8.
- 8. Scolapio JS, Camilleri M, Fleming CR. Gastrointestinal motility considerations in patients with short-bowel syndrome. Dig Dis. 1997;15:253–62.
- 9. Remington M, Malagelada JR, Zinsmeister A, et al. Abnormalities in gastrointestinal motor activity in patients with short bowels: effect of a synthetic opiate. Gastroenterology. 1983;85:629–36.
- 10. Schmidt T, Pfeiffer A, Hackelsberger N, et al. Effect of intestinal resection on human small bowel motility. Gut. 1996;38:859–63.
- 11. Millar AJ. Non-transplant surgery for short bowel syndrome. Pediatr Surg Int. 2013;29:983–7.
- 12. Sommovilla J, Warner BW. Surgical options to enhance intestinal function in patients with short bowel syndrome. Curr Opin Pediatr. 2014;26:350–5.
- 13. Algotar A, Dienhart M, Jacob D, et al. Utility of motility studies in selected cases of intestinal failure. Presented at North American Society of pediatric gastroenterology hepatology and nutrition, Washington, DC, USA; 2015.
- 14. Stahlgren L, Roy R, Umana G. A mechanical impediment to intestinal flow; physiological effects on intestinal absorption. JAMA. 1964;187:41–4.
- 15. Georgeson K, Halpin D, Figueroa R, et al. Sequential intestinal lengthening procedures for refractory short bowel syndrome. J Pediatr Surg. 1994;29:316–20.
- 16. Beyer-Berjot L, Joly F, Maggiori L, et al. Segmental reversal of the small bowel can end permanent parenteral nutrition dependency: an experience of 38 adults with short bowel syndrome. Ann Surg. 2012;256:739–44.
- 17. Tanner WA, O'Leary JF, Byrne PJ, et al. The effect of reversed jejunal segments on the myoelectrical activity of the small bowel. Br J Surg. 1978;65:567–71.
- 18. Glick PL, de Lorimier AA, Adzick NS, et al. Colon interposition: an adjuvant operation for short-gut syndrome. J Pediatr Surg. 1984;19:719–25.
- 19. Almond SL, Haveliwala Z, Khalil B, et al. Autologous intestinal reconstructive surgery to reduce bowel dilatation improves intestinal adaptation in children with short bowel syndrome. J Pediatr Gastroenterol Nutr. 2013;56:631–4.
- 20. Pakarinen MP, Kurvinen A, Koivusalo AI, et al. Long-term controlled outcomes after autologous intestinal reconstruction surgery in treatment of severe short bowel syndrome. J Pediatr Surg. 2013;48:339–44.
- 21. Bianchi A. Intestinal loop lengthening—a technique for increasing small intestinal length. J Pediatr Surg. 1980;15:145–51.
- 22. Kim HB, Fauza D, Garza J, et al. Serial transverse enteroplasty (STEP): a novel bowel lengthening procedure. J Pediatr Surg. 2003;38:425–9.
- 23. Figueroa-Colon R, Harris PR, Birdsong E, et al. Impact of intestinal lengthening on the nutritional outcome for children with short bowel syndrome. J Pediatr Surg. 1996;31:912–6.
- 24. Javid PJ, Kim HB, Duggan CP, et al. Serial transverse enteroplasty is associated with successful short-term outcomes in infants with short bowel syndrome. J Pediatr Surg. 2005;40:1019–23.
- 25. Weber TR, Powell MA. Early improvement in intestinal function after isoperistaltic bowel lengthening. J Pediatr Surg. 1996;31:61–3.
- 26. Weber TR. Isoperistaltic bowel lengthening for short bowel syndrome in children. Am J Surg. 1999;178:600–4.
- 27. Sudan D, Thompson J, Botha J, et al. Comparison of intestinal lengthening procedures for patients with short bowel syndrome. Ann Surg. 2007;246:593–601.
- 28. Reinshagen K, Zahn K, Buch C, et al. The impact of longitudinal intestinal lengthening and tailoring on liver function in short bowel syndrome. Eur J Pediatr Surg. 2008;18:249–53.
- 29. Modi BP, Javid PJ, Jaksic T, et al. First report of the international serial transverse enteroplasty data registry: indications, efficacy, and complications. J Am Coll Surg. 2007;204:365–71.
- 30. Chang RW, Javid PJ, Oh JT, et al. Serial transverse enteroplasty enhances intestinal function in a model of short bowel syndrome. Ann Surg. 2006;243:223–8.
- 31. Javid PJ, Sanchez SE, Horslen SP, et al. Intestinal lengthening and nutritional outcomes in children with short bowel syndrome. Am J Surg. 2013;205:576–80.
- 32. Avitzur Y, Grant D. Intestine transplantation in children: update 2010. Pediatr Clin North Am. 2010;57:415–31. Table.
- 33. Galea MH, Holliday H, Carachi R, et al. Short-bowel syndrome: a collective review. J Pediatr Surg. 1992;27:592–6.
- 34. Georgeson KE, Breaux Jr CW. Outcome and intestinal adaptation in neonatal short-bowel syndrome. J Pediatr Surg. 1992;27:344–8.
- 35. Beath S, Pironi L, Gabe S, et al. Collaborative strategies to reduce mortality and morbidity in patients with chronic intestinal failure including those who are referred for small bowel transplantation. Transplantation. 2008;85:1378–84.
- 36. Kaufman SS, Atkinson JB, Bianchi A, et al. Indications for pediatric intestinal transplantation: a position paper of the American Society of Transplantation. Pediatr Transplant. 2001;5:80–7.
- 37. Mousa H, Bueno J, Griffiths J, et al. Intestinal motility after small bowel transplantation. Transplant Proc. 1998;30:2535–6.
- 38. Johnson CP, Sarna SK, Zhu YR, et al. Effects of intestinal transplantation on postprandial motility and regulation of intestinal transit. Surgery. 2001;129:6–14.
- 39. Kiyochi H, Ono A, Miyagi K, et al. Extrinsic reinnervation one year after intestinal transplantation in rats. Transplant Proc. 1996;28:2542.
- 40. Kiyochi H, Ono A, Yamamoto N, et al. Extrinsic nerve preservation technique for intestinal transplantation in rats. Transplant Proc. 1995;27:587–9.
- 41. Le Blanc-Louvry I, Ducrotte P, Peillon C, et al. Roux-en-Y limb motility after total or distal gastrectomy in symptomatic and asymptomatic patients. J Am Coll Surg. 2000;190:408–17.
- 42. von Websky MW, Kalff JC, Schafer N. Current knowledge on regulation and impairment of motility after intestinal transplantation. Curr Opin Organ Transplant. 2015;20:303–7.
- 43. Watanabe T, Hoshino K, Tanabe M, et al. Correlation of motility and neuronal integrity with a focus on the grade of intestinal allograft rejection. Am J Transplant. 2008;8:529–36.
- 44. Nishimoto Y, Taguchi T, Masumoto K, et al. Real-time monitoring for detecting rejection using strain gauge force transducers in porcine small bowel transplantation. Transplant Proc. 2004;36:343–4.
- 45. Zhang YM, Liu XL, Xue DB, et al. Myoelectric activity and motility of the Roux limb after cut or uncut Roux-en-Y gastrojejunostomy. World J Gastroenterol. 2006;12:7699–704.
- 46. Le Blanc-Louvry I, Ducrotte P, Lemeland JF, et al. Motility in the Roux-Y limb after distal gastrectomy: relation to the length of the limb and the afferent duodenojejunal segment—an experimental study. Neurogastroenterol Motil. 1999;11:365–74.
- 47. Klaus A, Weiss H, Kreczy A, et al. A new biliodigestive anastomosis technique to prevent reflux and stasis. Am J Surg. $2001;182:52-7$.
- 48. Jen HC, Rickard DG, Shew SB, et al. Trends and outcomes of adolescent bariatric surgery in California, 2005–2007. Pediatrics. 2010;126:e746–53.
- 49. Loux TJ, Haricharan RN, Clements RH, et al. Health-related quality of life before and after bariatric surgery in adolescents. J Pediatr Surg. 2008;43:1275–9.
- 50. Harrison MR, Bjordal RI, Langmark F, et al. Congenital diaphragmatic hernia: the hidden mortality. J Pediatr Surg. 1978;13: 227–30.
- 51. Skari H, Bjornland K, Haugen G, et al. Congenital diaphragmatic hernia: a meta-analysis of mortality factors. J Pediatr Surg. 2000;35:1187–97.
- 52. Cannon C, Dildy GA, Ward R, et al. A population-based study of congenital diaphragmatic hernia in Utah: 1988–1994. Obstet Gynecol. 1996;87:959–63.
- 53. Moore A, Umstad MP, Stewart M, et al. Prognosis of congenital diaphragmatic hernia. Aust N Z J Obstet Gynaecol. 1998; 38:16–21.
- 54. Vanamo K, Rintala RJ, Lindahl H, et al. Long-term gastrointestinal morbidity in patients with congenital diaphragmatic defects. J Pediatr Surg. 1996;31:551–4.
- 55. Peetsold MG, Kneepkens CM, Heij HA, et al. Congenital diaphragmatic hernia: long-term risk of gastroesophageal reflux disease. J Pediatr Gastroenterol Nutr. 2010;51:448–53.
- 56. Kieffer J, Sapin E, Berg A, et al. Gastroesophageal reflux after repair of congenital diaphragmatic hernia. J Pediatr Surg. 1995;30:1330–3.
- 57. Arena F, Romeo C, Baldari S, et al. Gastrointestinal sequelae in survivors of congenital diaphragmatic hernia. Pediatr Int. 2008; 50:76–80.
- 58. Kilby MD. The incidence of gastroschisis. BMJ. 2006;332:250–1.
- 59. Ledbetter DJ. Gastroschisis and omphalocele. Surg Clin North Am. 2006;86:249–60, vii.
- 60. Vermeij-Keers C, Hartwig NG, van der Werff JF. Embryonic development of the ventral body wall and its congenital malformations. Semin Pediatr Surg. 1996;5:82–9.
- 61. Hoyme HE, Higginbottom MC, Jones KL. The vascular pathogenesis of gastroschisis: intrauterine interruption of the omphalomesenteric artery. J Pediatr. 1981;98:228–31.
- 62. Phillips JD, Raval MV, Redden C, et al. Gastroschisis, atresia, dysmotility: surgical treatment strategies for a distinct clinical entity. J Pediatr Surg. 2008;43:2208–12.
- 63. Snyder CL, Miller KA, Sharp RJ, et al. Management of intestinal atresia in patients with gastroschisis. J Pediatr Surg. 2001;36: 1542–5.
- 64. Hoehner JC, Ein SH, Kim PC. Management of gastroschisis with concomitant jejuno-ileal atresia. J Pediatr Surg. 1998;33:885–8.
- 65. Kato T, Tzakis AG, Selvaggi G, et al. Intestinal and multivisceral transplantation in children. Ann Surg. 2006;243:756–64.
- 66. Durkin ET, Lund DP, Shaaban AF, et al. Age-related differences in diagnosis and morbidity of intestinal malrotation. J Am Coll Surg. 2008;206:658–63.
- 67. Penco JM, Murillo JC, Hernandez A, et al. Anomalies of intestinal rotation and fixation: consequences of late diagnosis beyond two years of age. Pediatr Surg Int. 2007;23:723–30.
- 68. Devane SP, Coombes R, Smith VV, et al. Persistent gastrointestinal symptoms after correction of malrotation. Arch Dis Child. 1992;67:218–21.
- 69. la Vecchia LK, Grosfeld JL, West KW, et al. Intestinal atresia and stenosis: a 25-year experience with 277 cases. Arch Surg. 1998; 133:490–6.
- 70. Cezard JP, Aigrain Y, Sonsino E, et al. Postobstructive enteropathy in infants with transient enterostomy: its consequences on the upper small intestinal functions. J Pediatr Surg. 1992;27:1427–32.
- 71. Cezard JP, Cargill G, Faure C, et al. Duodenal manometry in postobstructive enteropathy in infants with a transient enterostomy. J Pediatr Surg. 1993;28:1481–5.
- 72. Preston DM, Hawley PR, Lennard-Jones JE, et al. Results of colectomy for severe idiopathic constipation in women (Arbuthnot Lane's disease). Br J Surg. 1984;71:547–52.
- 73. Pikarsky AJ, Singh JJ, Weiss EG, et al. Long-term follow-up of patients undergoing colectomy for colonic inertia. Dis Colon Rectum. 2001;44:179–83.
- 74. Kayama H, Koh K. Clinical and experimental studies on gastrointestinal motility following total colectomy: direct measurement (strain gauge force transducer method, barium method) and indirect measurement (hydrogen breath test, acetaminophen method). J Smooth Muscle Res. 1991;27:97–114.
- 75. You YT, Wang JY, Changchien CR, et al. Segmental colectomy in the management of colonic inertia. Am Surg. 1998;64:775–7.
- 76. Lundin E, Karlbom U, Pahlman L, et al. Outcome of segmental colonic resection for slow-transit constipation. Br J Surg. 2002;89:1270–4.
- 77. Villarreal J, Sood M, Zangen T, et al. Colonic diversion for intractable constipation in children: colonic manometry helps guide clinical decisions. J Pediatr Gastroenterol Nutr. 2001;33:588–91.
- 78. Martin MJ, Steele SR, Mullenix PS, et al. A pilot study using total colonic manometry in the surgical evaluation of pediatric functional colonic obstruction. J Pediatr Surg. 2004;39:352–9.
- 79. Martin MJ, Steele SR, Noel JM, et al. Total colonic manometry as a guide for surgical management of functional colonic obstruction: preliminary results. J Pediatr Surg. 2001;36:1757–63.