Constipation and Encopresis in Childhood

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IMPORTANT POINTS

1. **Constipation is a symptom, not a diagnosis, that has multiple etiologies and multiple alternative treatments.**
2. **Encopresis or fecal soiling has many etiologies, not all of which are associated with fecal retention or responsive to laxatives.**
3. **Children who have Hirschsprung disease almost never have encopresis because the aganglionic segment maintains tonic contraction due to the absence of inhibitory neurons containing nitric acid and vasoactive intestinal polypeptide.**
4. **Functional retentive constipation is associated with loss of lower colonic muscle tone and will not resolve until complete repetitive evacuation of the lower colon allows muscle tone to be restored.**
5. **Successful treatment of constipation and encopresis requires a combination of patient and parent education, medical therapy, nutritional intervention, behavioral modification, and long-term monitoring of compliance.**

Introduction

Constipation is a very common frustration for children, parents, and physicians. It is reported to account for nearly 5% of all outpatient visits to pediatric clinics and more than 25% of all referrals to pediatric gastroenterologists. Painful defecation and encopresis (involuntary passage of stool from the anus) usually are the first manifestations noted. Constipation generally is defined by the hard nature of the stool, the pain associated with its passage, or the failure to pass three stools per week. It would be preferable to define constipation as the failure to evacuate the lower colon completely with a bowel movement. This allows the parents and physician to realize that a child who has three small stools daily may not yet have evacuated the colon, while a child who has two large soft stools a week may not be constipated.

The frequency of stools in most children decreases from a mean of four per day in the first week of life to 1.7 per day by the age of 2 years. Over this interval, stool volume increases more than tenfold while maintaining a consistent water content of approximately 75%.

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Peristaltic activity in the colon has been demonstrated as early as the eighth fetal week, with normal colonic haustra seen by 12 weeks. Intestinal transit time from mouth to rectum increases from 8 hours in the first month of life to 16 hours by 2 years of age to 26 hours by the age 10.

Normal continence is maintained by the resting tonicity of the internal anal sphincter and can be enhanced by contraction of the puborectalis muscle, which creates a 90-degree angle of rectum to the anal canal. When more than 15 cc of stool enters the normal rectum, stretch receptors and nerves in the intramural plexus are activated. Inhibitory interneurons decrease the resting tone in the involuntary smooth muscle of the internal anal sphincter, relaxing the sphincter and allowing stool to reach the external anal sphincter that is composed of voluntary skeletal muscle. The urge to defecate is signaled. If the child relaxes the external anal sphincter, squats to straighten the anorectal canal, and increases intra-abdominal pressure with the Valsalva maneuver, the rectum is evacuated of stool. If, however, the child tightens the external anal sphincter and the gluteal muscles, the fecal mass is pushed back into the rectal vault and the urge to defecate subsides. Repetitive denial of evacuation leads to stretching of the rectum and eventually of the lower colon, producing a reduction in muscle tone and retention of stool. The longer the stool remains in the rectum, the more water is removed, and the harder the stool becomes to the point of impaction.

Encopresis or fecal soiling usually is the result of looser stool leaking or overflowing from a rectum that has been distended by retained stool. Encopresis is three to six times more common among males than females and is acknowledged in 3% of 4-year-olds and 1.6% of 10-year-olds. The child is not aware of the soiling until it is nearly complete, an observation confirmed by anorectal motility studies that document decreased sensitivity to distension in a chronically distended rectum. Although encopresis presents predominantly between 3 and 7 years of age, in one study, 35% of the children had hard stools within the first 6 months of life, 40% experienced delays in toilet training, and 60% reported painful stools before the age of 3 years.

More than 90% of chronic encopresis occurs in the context of functional fecal retention, but two forms of encopresis are not associated with fecal retention. Organic incontinence can occur in children who have damaged corticospinal pathways such as lumbosacral myelomeningocele. Other children have anorectal dysfunction after operative pullthrough surgery for high imperforate anus or colectomy. Incontinence also occurs in children who have diarrhea because pelvic floor muscles fatigue in less than 1 minute of continuous contraction above resting tone as they try to hold in a loose stool. The second form of encopresis is functional—the voluntary or impulsive passage of variable volumes of stool into the diaper or clothes. This may result from simple anxiety and the desire to continue diaper use or from more severe passive-aggressive behavior in the context of emotional impulsivity. It is critical to recognize that these children do not have underlying constipation or fecal retention

TABLE 1. Differential Diagnosis of Chronic Constipation

Functional:
Infantile rectal confusion
Functional fecal retention/withholding
Irritable bowel syndrome
Neurologic:
Aganglionosis (Hirschsprung)
Neuronal dysplasia (Hyperganglionosis)
Hypoganglionosis (Chagas)
Familial dysautonomia
Spinal cord dysplasia/Hypotonia syndromes
Botulism
Obstructive:
Anterior ectopic anus
Congenital or acquired anal ring stenosis
Small left colon
Meconium ileus or equivalent (cystic fibrosis)
Rectal or sigmoid stricture (postoperative, post-necrotizing enterocolitis)
Adenocarcinoma
Pelvic tumor, mass, or pregnancy
Endocrine/Metabolic Pseudo-obstructive:
Congenital pseudo-obstruction
Visceral myopathy, neuropathy
Amyloidosis
Collagen-vascular: Scleroderma, lupus, dermatomyositis
Diabetes/diabetes insipidus
Hypothyroidism
Hyperparathyroidism/multiple endocrine adenoma
Pheochromocytoma
Porphyria
Graft versus host disease
Medicinal:
Laxative abuse
Diuretics
Tricyclic antidepressants
Narcotics/codeine
Aluminum antacids
Vincristine
Calcium channel blockers
Iron
Lead poisoning

and that laxative therapy could be detrimental. Psychological counseling may be equally valuable in all forms of encopresis because the stress of soiling is independent of etiology.

Differential Diagnosis of Constipation

The functional and nonfunctional etiologies of constipation are listed in part in Table 1. It is impossible to discuss all of these in detail, but a few clinical points will be reviewed. A very complete review of Hirschsprung disease was published in *Pediatrics in Review* in January 1995.

FUNCTIONAL

Straining with the passage of soft stool is normal in neonates and infants. It is related to their inability to coordinate pelvic floor relaxation with the Valsalva maneuver and to straighten the anorectal canal when lying down. More erratic stooling patterns are reported in infants who have gastroesophageal reflux. Although breastfed infants often have multiple loose stools each day, it is equally normal to have only one a week. The introduction of a formula supplement or rice cereal may reduce stool frequency and increase consistency quickly. Insertion of a glycerin suppository or rectal stimulation with a lubricated thermometer will induce reflex anal sphincter relaxation and the desired evacuation, but regular rectal manipulation is discouraged. If the stools become firm balls or rectal fissures evolve, softening of the stool is indicated. This often can be accomplished by substituting barley cereal for rice; by using a nondigestible sugar such as fructose, sorbital (prune juice), or lactulose; or by introducing higher fiber vegetables into the diet. Mineral oil is not used in infancy, and corn syrup is a less effective osmotic agent because older infants can digest it. Honey should be avoided because of the risk of botulism, a theoretic risk with corn syrup as well. The concerns of most parents and physicians can be addressed by eliciting a careful history and family history and performing a physical examination that includes perineal and rectal examination followed by education and reassurance about the normal variation of infantile stool patterns. Generally, the passage of a large soft stool at intervals of less than 72 hours in infants is not associated with rectal fecal retention.

Functional fecal retention is the most common explanation for childhood constipation. As noted previously, voluntary tightening of the external anal sphincter and contraction of the gluteal muscles prevent the passage of stool. This may be initiated solely as a “control” issue with unsuccessful toilet training, the logical response to painful stools in the context of anal inflammation from fissures, perianal streptococcal infection, perianal abscess, or a perceived threatening event such as a television show, birth of a sibling, or desire to avoid defecation in a strange toilet when away from home. Some toddlers and older children (especially those who have attention deficit hyperactivity disorder) are too distracted to evacuate.

The resulting distension produces a variety of symptoms from gaseous distension to delays in evacuation of up to 1 week or more. Enormous

stools to the point of being "toilet-plugging-specials" are passed with significant pain and a prepassage ritual of gluteal tightening and posturing so dramatic that parents may videotape the performance to be sure the doctor "knows how bad this is." Other symptoms of increasing fecal accumulation in the colon include early satiety, desire to eat small volumes all day, increasing irritability, and unpredictable spasms of abdominal pain usually located in the lower abdomen. Encopresis becomes increasingly frequent. Painless rectal bleeding from internal anal fissures also may be noted on the surface of stools or on the toilet tissue after defecation. After the passage of an enormous stool, symptoms generally resolve for a few days, then recur.

A rectal examination confirms normal anal tone with a massive rectal ampulla filled with formed stool. Abdominal examination may reveal palpable stool in a dilated lower colon. Toddlers who have functional fecal retention cannot be toilet trained until the fear of pain has been allayed and normal rectal sensation has been restored. Interestingly, the constipated toddler who refuses toilet training has no greater frequency of other behavioral concerns than other toddlers. Successful management requires complete evacuation of the colon, sustained evacuation by a "motivational" technique that uses laxatives in younger children and lower dose laxatives with behavioral modification in older children as well as stool softening by dietary fiber, mineral oil, or lactulose. Relapse rates are high and require close follow-up.

Irritable bowel syndrome is manifested in some children by intermittent passage of firm stool in painful bowel movements. In contrast to functional fecal retention, the constipation of irritable bowel syndrome is not associated with distension of the colon but with spasm in a colon of normal calibre. Encopresis is rare, and management is based on increased dietary fiber rather than laxatives.

NEUROLOGIC

Hirschsprung disease or aganglionosis occurs in 1 in 5,000 births. The male-to-female ratio is 4 to 1, and the incidence increases with longer segments of disease. The diagnostic lack of ganglion cells in the myenteric and submucosal plexus of the bowel wall extends proximally from the internal anal sphincter. Among 80% of the involved children, the aganglionic segment does not extend above the sigmoid, while the entire colon and some small bowel may be involved in 3%. The aganglionic bowel has thickened nerve fibers staining for acetylcholinesterase, offering a second pathologic feature on histologic examination. The aganglionic segment of bowel fails to relax because of the absence of inhibitory neurons containing nitric oxide and vasoactive intestinal polypeptide. Thus, difficulty with evacuation is present from birth; meconium is not passed in the first 48 hours of life in 40% of involved infants.

The presence of early obstructive features (60% by 3 months of age) and the absence of encopresis distinguish Hirschsprung disease from functional fecal retention.

Recurrent abdominal distension, emesis, failure to thrive, and acute enterocolitis allow diagnosis of 60% of patients by 3 months of age. The presence of early obstructive features, onset in infancy, and nearly complete absence of encopresis distinguish Hirschsprung disease from functional fecal retention. On rectal examination, the aganglionic bowel is tight around the finger and the rectal ampulla is not dilated. A barium enema usually allows visualization of the transition zone between the tonically contracted aganglionic segment and the dilated proximal bowel. The enema should be performed without preparation, which distorts the distal anatomy. Manometric studies of the rectum demonstrate the failure of the internal anal sphincter to relax when distended. Confirmation of the diagnosis requires rectal biopsy that employs a suction technique for screening and full-thickness technique for definitive operative decisions.

Hirschsprung disease is a heterogenous genetic disorder with risk rates for siblings ranging from 3% with short segment disease to 25% with a female who has long segment disease. An autosomal dominant form occurs with mutation in the RET gene. Syndromes associated with Hirschsprung disease include trisomy 21, deletion of chromosome 13q, Smith-Lemli-Opitz, Waardenberg, Laurence-Moon-Biedl-Bardet, congenital deafness, and congenital central hypoventilation.

Neuronal dysplasia, in contrast to aganglionic disease, is associated with increased numbers of ganglion cells (hyperganglionosis) in the lower colon. It may present throughout childhood with variable constipation or features of pseudo-obstruction. It is more frequent among children who have neurofibromatosis and has been associated with multiple endocrine neoplasia type IIb due to glioneuromas of the intestinal tract. Surgical intervention is individualized and based on the severity of symptoms and manometric demonstration of severely impaired rectal relaxation.

Reduced numbers of ganglion cells (hypoganglionosis) usually is an acquired disease of ganglion cell destruction seen in Chagas disease or paraneoplastic syndrome. Mild reductions in ganglion cell numbers have been reported in some children who have congenital short bowel, malrotation, and pyloric hypertrophy.

OBSTRUCTIVE

Some obstructive conditions present in infancy, such as anterior ectopic anus, congenital anal ring stenosis, and small left colon, while others are acquired in later childhood. Anterior ectopic anus is defined by a measurable displacement of the anal opening based on the ratio of the anus-to-fourchette to the coccyx-to-fourchette being less than 0.34 in females and less than 0.46 in males. On rectal examination, the shift creates a broad posterior rectal shelf that increases with distension of the ampulla. Surgical correction occasionally is required, but most patients do well if distension of the

ampulla is minimized. Anal ring stenosis presents with painful constipation in infancy. A narrow ring or band is noted on rectal examination with a dilated ampulla above it. The stenosis usually responds to gradual dilatation, but lysis of the stenotic band is a surgical option taking care to preserve sphincter function.

ENDOCRINE/METABOLIC

The term "pseudo-obstruction syndrome" has been applied to all nonanatomic disorders of abnormal peristalsis. Motility abnormalities may manifest with delayed gastric emptying, small bowel stasis, and/or constipation. The primary form is familial, presenting in infancy with failure to thrive, distension, and progressive dysmotility with delayed gastric emptying and constipation.

The secondary or acquired forms of pseudo-obstruction encompass a wide variety of neurologic, muscular, pharmacologic, and metabolic disorders. Based on the remainder of the clinical history, physical features, or laboratory abnormalities, each eventually can be excluded. In children who have primary myopathy, collagen-vascular disease, or amyloidosis, the degree of dysmotility and constipation becomes progressive. We have had several children who had diabetes demonstrate progressive failure to thrive with obstipation, only to reverse the intestinal manifestations when we confirmed and treated the coexistent gluten-sensitive enteropathy.

Complications of Constipation

Abdominal or rectal pain and encopresis have been emphasized as the primary complications of childhood constipation, but there are other potential problems (Table 2). Enuresis is acknowledged in more than 40% of children who have encopresis and must be addressed. In some, the enuresis resolves when the pelvic mass of retained rectal stool is evacuated, thereby allowing the bladder to expand. A greater urologic concern is the role that dilated lower colon plays in increased frequency of urinary tract infection and potential obstruction of the left ureter.

The dilated lower colon may lose enough tone to allow internal prolapse or intussusception, which may manifest as rectal prolapse after evacuation of even soft stool. Chronic low-grade internal prolapse creates an ischemic ulcer of the rectal wall (solitary rectal ulcer syndrome), which presents clinically with bloody mucus independent of the character of the associated stool. The diffuse irritation of the colon caused by firm stool even may lead to protein-losing enteropathy. Stasis syndrome is seen primarily with pseudo-obstruction.

The social stigma associated with increased flatulence and the odor of encopresis can be very debilitating to any child. Most children who have chronic encopresis will go through an interval of denying the obvious when asked and even may hide the soiled clothes from the parent.

TABLE 2. Complications of Chronic Constipation

• Pain: Anal or abdominal
• Rectal fissure
• Encopresis
• Enuresis
• Urinary tract infection/ureteral obstruction
• Rectal prolapse/solitary ulcer
• Stasis syndrome —Bacterial overgrowth —Carbohydrate fermentation, maldigestion —Bile acid deconjugation —Steatorrhea —Protein-losing enteritis
• Social exclusion/depression/anxiety

Diagnostic Investigations

There is an interesting dichotomy in the approach to constipation in childhood—the differential diagnosis is lengthy, but the likelihood of undertaking an extensive exclusionary evaluation is small.

HISTORY

No diagnostic endeavor is of greater importance than the history. It begins with the birth history of gestational complications, birthweight, timing of passage of meconium, and tolerance of early feedings. Constipation frequently begins with transitions from breast to formula and from strained foods to table foods; the introduction of cow milk is the most constipating component of the young child's diet. Transitions to child care and initiation of all-day school with consequent loss of privacy also may be contributing factors. The greatest transition, of course, is from diaper to toilet training and is the prime time for withholding. Family history is reviewed for evidence of genetic factors, such as aganglionosis, cystic fibrosis, hypothyroidism, neurofibromatosis, or myopathies.

The character of the stools is reviewed from birth, especially for the first 24 hours, for consistency, calibre, volume, and frequency. Recurrent, small pellet stools are a feature of incomplete evacuation. Intermittent massive stools are a feature of functional fecal retention. Abdominal distension, narrow calibre stool, failure to thrive, and lack of encopresis all favor a distal obstruction, whether it is Hirschsprung disease, anal ring stenosis, anterior ectopic anus, small left colon, sacrococcygeal teratoma, or even infantile Crohn disease.

The age and circumstances at onset of encopresis should be documented. Soiling may be more erratic than progressive. Encopresis in the absence of constipation suggests an organic or behavioral origin. The response of the parent to the soiling also should be documented.

A history of possible sexual or rectal abuse should be elicited and

suspected if the age of onset of constipation is older than usual or the condition is more resistant to treatment. Internal rectal fissures are usually a complication of constipation; external rectal fissures may be a cause of constipation initiated by abuse, inflammatory proctitis, or Crohn disease.

The medical history addresses prior surgery, neonatal complications such as necrotizing enterocolitis, and courses of medications that may contribute to constipation (Table 1). Laxative abuse should lead to a suspicion of ano-rexia or bulimia; chronic depression or attention deficit hyperactivity disorder may lead to use of tricyclic antidepressants.

Although the emphasis on bowel pattern addresses constipation, intervals of significant diarrhea should raise concern about prestenotic enterocolitis in infants or irritable bowel in the adolescent. Many children who have encopresis evacuate enough loose stool for the parent to label it diarrhea.

When possible, ask the family to prepare a 5- to 7-day symptom and diet history prior to the visit, noting the frequency and character of the stool and any episodes of pain or encopresis. Documentation of the diet allows an estimate of caloric and nutrient adequacy as well as fiber content, monotony of intake, and some idea of what foods the child likes that could be increased.

PHYSICAL EXAMINATION

The examination begins with documentation of growth and weight gain, emphasizing alterations in the velocity or rate of growth prior to and during the active illness. The remainder of the general examination should focus on features of systemic diseases and include a thorough neurologic evaluation.

The abdomen is examined for degree of distension and documented by the umbilical girth, a measurement that can be repeated at home to record response. Bowel sounds are documented, and the perineum is inspected for evidence of encopresis, streptococcal or monilial infection, fissures, and anal wink. The anal opening is observed, and the distance to the coccyx and either vaginal or scrotal fourchette determined if anterior ectopic anus is suspected. Hemorrhoids are very rare in childhood, even with long-standing constipation. An anal fold may be seen with long-term passage of wide stools. Perirectal manifestations of Crohn disease include fistula, abscess, or ulceration in addition to rectal strictures.

The rectal examination is performed with the child positioned as comfortably as possible, even on the parent's lap. The anal canal, often tight on entry, should relax in the absence of a stenotic ring, stricture, or aganglionic segment. The normal ampulla is slightly dilated and may contain stool. A dilated ampulla filled with retained firm stool is a feature of functional retention. Internal fissures may be palpable. Pressure in all directions excludes the presence of a pelvic mass or tumor. An asymmetric posterior rectal shelf is characteristic of an anterior ectopic anus.

The abdominal examination may demonstrate palpable dilated loops of sigmoid and distal colon. Palpable stool in the colon in the presence of an empty ampulla suggests recent enema use or proximal obstruction from tumor or post-necrotizing enterocolitis strictures. The back should be examined for sacral skin clues to lower spine deformity.

LABORATORY INVESTIGATIONS

Blood testing is indicated to exclude some of the systemic disorders suggested by findings of the history or physical examination. These may include thyroid functions, serum calcium concentrations, electrolyte levels, magnesium concentrations, or urea nitrogen. Urinalysis and urine culture should be considered in all children who have a history of rectosigmoid impaction, especially if it is associated with encopresis.

The plain abdominal radiograph may be of value in the child in whom an abdominal examination is difficult or to monitor the completeness of evacuation in a child who exhibits marginal compliance. Lumbosacral spine radiographs or magnetic resonance imaging can be employed to visualize the sacral spine. As discussed previously, the unprepped barium enema is an invaluable tool for assessing the transition zone of aganglionic bowel. The barium enema may appear normal in the presence of total colonic aganglionosis, but the stricture following necrotizing enterocolitis will be defined easily. In normal children, the diameter of the rectum exceeds that of the sigmoid; in Hirschsprung disease, the opposite is true. The contrast enema defecogram has a definite role in assessing pelvic muscle function following surgery or in the context of central nervous system disease.

Anorectal manometry is available to evaluate internal anal sphincter relaxation and determine the level of pressure awareness in older children. It also will identify the 25% of chronically constipated children who exhibit a paradoxic increase in external anal sphincter pressure during defecation, a feature that predicts a long-term course to recovery and a likely role for biofeedback therapy. A few centers now offer total colonic motility, a valuable tool in the evaluation of neuropathic or muscular dysmotility in chronic intestinal pseudo-obstruction.

The value of the suction rectal biopsy has increased with the ability to stain the tissue for both ganglion cells and acetylcholinesterase, the former of which is lacking and the latter of which is increased in Hirschsprung disease. The rectal biopsy also can be of diagnostic value in the child who has amyloidosis, graft versus host disease, lipid storage disease, or Crohn disease. Full-thickness biopsies are obtained by the surgeon to document normal segments of colon at the time of diverting colostomy for aganglionosis.

Management of Chronic Constipation and Encopresis

There are three phases of management: complete evacuation or disimpaction, sustained evacuation to restore normal colorectal tone, and weaning from intervention. The success of each depends on the cooperation and understanding of the parent and, when possible, the child. The key issues in this educational process are listed in Table 3.

TABLE 3. Key Issues in Parent and Patient Education

1. Patience, patience, patience . . . months of treatment and awareness are needed.
2. The physical components of constipation began long before the encopresis first was noted.
3. Encopresis or soiling is involuntary and will not respond to behavior modification or punishment alone.
4. Even so, soiling will occur more at home or play than in school.
5. No treatment will be effective unless complete evacuation is achieved.
6. Because colon muscle tone can be restored in childhood, physical dependence on laxatives is not long-term.
7. Most treatment failures are due to inadequate medication or stopping too soon.
8. Successful management of encopresis requires a combination of medical therapy, behavior modification, and counseling that is customized to the needs of the child.
9. Behavior modification stresses habitual toilet use and rewards the effort more than the success. The effort comes from the child; the success comes from the treatment.
10. Dietary fiber is effective at improving the efficacy of evacuation only after the muscle tone of the colon and rectum is restored.
11. Toilet training should not be attempted nor will it succeed until rectal awareness is restored and defecation is pain-free.
12. Referral for psychological counseling does not obviate the need for medical or dietary therapy.

Families are counseled about normal patterns of evacuation, the physical and psychological components of the present predicament, the role of diet and counseling, the options of therapy, the realistic expectations for response to therapy, the level of compliance required of the child, and the need for patience. The older child is encouraged to be involved by keeping a sticker chart or calendar to document efforts, successes, and failures. Many of these older children never have been truly toilet trained. They are encouraged to establish a "habit" of toilet use independent of the rest of treatment. One simple but critical step in management is to use a footstool for the child to maximize abdominal pressure during the Valsalva maneuver. Behavioral modification rewards are geared to reward the effort of evacuation, counting on the medications and diet to assure success. As discussed previously, counseling of the child and family can be invaluable to the educational process.

There are two phases to the nutritional component of management. At presentation, the family is counseled to reduce constipating foods such as dairy products and starches. Additional fiber has no value when colorectal tone is diminished in the child who has active functional fecal retention. In the second phase, when tone is being restored, additional fiber is of great value to improve the "efficiency" of evacuation and usually is provided in the form of a supplement. Once remission is achieved, dietary fiber is maximized, starches and dairy are increased to moderate levels, and medications are weaned gradually. Table 4 lists medical and nutritional supplements available for treatment, and Table 5 reviews a few of the general and age-specific issues in treatment.

PHASE 1: COMPLETE EVACUATION OR DISIMPACTION

No management plan will succeed if complete evacuation is not achieved initially. Sometimes, treatment begins immediately after the passage of a massive stool that indicates an empty colon. Occasionally manual disimpaction is required, but the general rule is to minimize rectal manipulation.

If the rectal examination reveals a firm, wide impaction of stool, enemas will be required for up to 2 to 5 days to minimize cramping and pain. We usually begin with a mineral oil enema, which is designed to soften the rectal mass and lubricate the mucosa. It is followed in 30 minutes with a hypertonic phosphate enema. The enemas usually are administered at home in the evening until evacuation is confirmed. Use beyond 3 to 5 days is discouraged because of the potential for electro-lyte disturbance. Tap water, herbal, and soap suds enemas should be avoided because of the documented complications of water intoxication, bowel perforation, and bowel necrosis. If the child fails to evacuate with the use of mineral oil and phosphate enemas, a hyperosmolar enema can be given. One option, water-soluble contrast media, is administered by the radiologist under fluoroscopic control. Another option is a home brew of 50:50 milk and molasses.

An efficient but costly alternative is under study at some referral centers. "Pulsed irrigation-enhanced evacuation" entails the placement of a rectal tube and the delivery of pulses of warm irrigating solution with simultaneous drainage of rectal contents. Disimpaction occasionally is achieved with oral high-dose mineral oil for 3 days. Magnesium citrate is not used in a child who has impaction.

If disimpaction is not achieved with enemas or irrigation, the child must be admitted to the hospital for oral lavage (instillation and leaving in) with polyethylene glycol-electrolyte solution. Because volumes of 30 to 40 mL/kg per hour are used, most children require a nasogastric tube. Normal children being prepped for emergency colonoscopy usually evacuate in 6 to 8 hours; those who retain feces chronically may not evacuate for more than 24 hours. Oral clear liquids are allowed. There is no need for routine monitoring of electrolytes during treatment. Some cramping

TABLE 4. Treatment of Chronic Constipation

Disimpaction (Usually 2 to 5 days):		
Enema:	Mineral oil	2 oz per 10 kg up to 4.5 oz
	Hypertonic phosphate	1 oz per 10 kg up to 4.5 oz
	Milk and molasses	50:50 mix up to 6 oz maximum
Oral/nasogastric:	Mineral oil	1 oz per year of age BID up to 8 oz BID × 3
	Polyethylene glycol	10 to 40 mL/kg per hour for 12 to 36 h
Sustain Evacuation (Usually 3 to 12 months or more):		
Habitual toilet use with positive rewards, daily star chart, and behavioral therapy		
Diet:	Initial:	Low dairy, low fiber
	Transition:	Moderate dairy, high fiber with supplements
Fiber:	Barley malt	Barley cereal, malt soup extract (Maltsupex)
	Cellulose	Citrocel
	Psyllium	Metamucil, Fiberall
	Polycarbophil	Fibercon, Konsyl
Lubricants:	Mineral oil	Kondremul, Agarol
	Surfactant	Docusate
Hyperosmolar sugars:	Fructose/Sorbitol	Prune juice
	Lactulose	1 to 2 mL/kg per day
Stimulants:	Salts	Milk of magnesia, magnesium citrate
	Senna	Senekot
	Diphenylmethane	Bisacodyl
	Prokinetics	Cisapride, Metaclopramide
Combination agents also are marketed		
Phenolphthalein should not be used in children		

is inevitable, and mild emesis may be noted.

Glycerine suppositories have no value once rectal distension develops, so they rarely are of value in those older than 6 months of age. Bisacodyl suppositories have value in the constipated child who does not have impaction, but otherwise they simply are inserted into the middle of the fecal mass and never dissolve or reach the mucosa.

PHASE 2: SUSTAIN COMPLETE EVACUATION

Once rectal evacuation has been confirmed, the challenge is to keep the rectum empty. This is accomplished by habitual toilet use, stool softeners, and "motivator" laxatives to achieve daily complete evacuation. This phase can last from 2 to 6 months or longer. The aim is to allow the distended colon to return to normal calibre and tone. When this is successful, encopresis due to functional fecal retention can resolve within days. Children who have organic or behavioral encopresis never should be managed in this fashion. The best approach is a combination of medical therapy, behavioral modification, and counseling. Older children who have incontinence, paradoxic failure of anal relaxation, or postoperative low imperforate anus are candidates for biofeedback therapy with an experienced therapist.

During infancy, softening of the stool alone is usually sufficient. As noted in Table 5, this can be addressed with barley cereal, barley malt extract, 4 to 6 oz prune juice, or after 6 months of age, with lactulose. All can be associated with transient increased gassiness for a few days until evacuation begins. Rice cereal and bananas should be avoided. Once a variety of strained foods are begun, most infants can stop the nondigestible sugars, but recurrence may occur with the transition to whole milk and table foods at 1 year of age.

Toddlers are the most resistant to early treatment. They are hard-core fecal withholders, poorly compliant with medication, and resistant to behavior modification. The parents also tend to undermedicate unless they are convinced of the need for and safety of such agents. Toilet training should not be attempted until both the physical and psychological components of the constipation are under control. Stool softeners and moderate-to-high-dose stimulant laxatives are employed for several months or longer.

In toddlers, stools usually are softened with either lactulose or mineral oil. Lactulose is begun at 5 to 10 mL BID, increasing as required up to 45 mL BID. Lactulose is available only by prescription, which often is a cost advantage. Increased gas and looser-than-desired stools occur initially. Emulsified mineral oil is an alternative for toddlers and older children. It is begun at 2 mL/kg per dose BID and increased as needed up to 6 to 8 oz per day. Transient oil leakage may occur until the right dose is achieved. Concerns about mineral oil interfering with absorption of fat-soluble vitamins have not been substantiated in short-term studies.

Motivating laxatives such as milk of magnesia and senna usually are employed. Preschoolers are begun at 2 tsp BID, with adjustments made to reach a goal of one to three substantial stools a day over 1 to 2 weeks. Older children will prefer the tablets, taking one to three twice daily and adjusting the dose to meet the same goal, with the ideal of no need to evacuate in school.

The increasing use of properistaltic agents to maximize gastric emptying has led to investigation of cisapride as a laxative. It is used widely in Europe, and recent experience with its use in the United States has been encouraging. When given beyond infancy in a dose of 0.2 mg/kg per dose TID with a

TABLE 5. Age-Related Issues in the Management of Constipation

Universal Principles
Educate patient and parents
Aim for complete evacuation
Sustain evacuation to restore tone and rectal awareness
Monitor compliance for at least 6 months
Use counseling and behavior modification to supplement medical therapy
Infant
Patient mentality: "I can't decide what to tighten and what to relax."
Organic etiologies must be excluded by history and examination
Dietary factors: Transitions in diet from breast to formula to milk
Softeners: Barley milk or cereal, prune juice, lactulose (after 6th month)
Stimulants: Rarely needed; avoid chronic rectal manipulation
Toddler
Patient mentality: "I'm not pooping, and I doubt you can make me."
Functional retention and withholding far exceed organic etiologies
Dietary factors: Reduce cheese and constipating solids; forget fiber for now
Softeners: Mineral oil or lactulose daily
Stimulants: Senna or milk of magnesia, moderate-to-high doses for months; wean very slowly until toilet training is easy
School age and older
Patient mentality: "It hurts, it leaks, and everybody knows . . . Help!"
Assess and treat complications; use lots of counseling
Dietary factors: Increase fiber when evacuation is assured
Softeners: Mineral oil, fiber, lactulose, colace
Stimulants: Use toilet habitually, modest dose (often QOD) laxative; if resistant to treatment, consider cisapride, biofeedback

maximum of 10 mg per dose, success has been reported in children who had intractable constipation and encopresis. It usually is added to the existing laxative regimen. Metaclopramide also has been used.

Neither phenolphthalein nor castoria is advised in children because long-term therapy usually is required, and there are specific safety concerns about the development of cathartic colon.

PHASE 3: WEANING FROM MEDICATION

Once regular evacuation has been sustained, the use of laxatives is reduced gradually. Softening agents are continued daily. Milk of magnesia or senna is administered every other day in the same dose for 1 month, then every 3 days for 1 month if regular complete evacuation continues.

At the same time, ingestion of insoluble or soluble fiber should be increased. Most fiber supplements are soluble, fermented in part by fecal bacteria, which contributes to increased fecal mass. Doses are increased slowly to minimize gas production and distension. Fiber supplements are available as powders to mix in juice, wafers, or tablets. To maximize compliance, the powdered fiber can be mixed in juice and frozen into popsicles. In contrast to stool softeners, bulk fiber allows increased rectal distension and awareness of the need to evacuate, which may facilitate toilet training in some toddlers.

If setbacks occur or compliance wanes, a bisacodyl suppository should be given on the third day on which no stool is passed to prevent recurrence of colon distension. Close follow-up, including routine calls and return of the symptom diary, allows adjustments in the medication and diet as the child allows.

Older children are managed with the same principles, although a greater degree of compliance and a greater role for behavioral modification can be anticipated. Fiber is introduced earlier in the older child to maximize awareness. Establishing the habit of regular toilet use is critical.

Even in the best of circumstances, months of treatment will be required, and rates of recurrence approach 50%. Early recognition of recurrence minimizes the need for medication.

SUGGESTED READING

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PIR QUIZ

11. A 6-year-old boy has had soiling since starting school 4 months ago. There is no history of previous stooling difficulties. Findings on inspection of the anus include deep fissures at the 6 and 12 o'clock positions. Rectal examination reveals a large, capacious rectum containing hard stool. Which of the following studies will be *most* useful in evaluating this patient in whom you suspect functional encopresis?
    A. Anorectal manometry.
    B. Barium enema.
    C. Colonoscopy.
    D. Plain abdominal radiography.
    E. Rectal biopsy.

12. A 13-year-old boy who has longstanding constipation and encopresis has a paradoxic inability to relax the external sphincter when studied by anorectal manometry. The *best* therapy for management of this problem is:
    A. Biofeedback therapy.
    B. High-fiber diet.
    C. Hypertonic phosphate enema.
    D. Prokinetic agents.
    E. Psychiatric consultation.

13. An 8-year-old boy who has constipation and encopresis is complying poorly with his laxative regimen and has impacted stool. Of the following, which treatment will provide the *most* effective and complete stool evacuation in this child?
    A. Administration of a polyethylene glycol-electrolyte preparation via nasogastric tube.
    B. Administration of bisacodyl suppositories.
    C. Administration of cisapride.
    D. Administration of high doses of mineral oil for 2 to 4 days.
    E. Surgical disimpaction under anesthesia.

14. A 4-year-old girl who had pull-through surgery in infancy for a high imperforate anus has had chronic soiling for 1 year. The *most* likely cause for her incontinence is:
    A. Anorectal dysfunction.
    B. Behavioral issues.
    C. Functional fecal retention.
    D. Irritable bowel syndrome.
    E. Pelvic floor muscle fatigue.

15. A 10-year-old boy who has chronic constipation has been treated with cathartic medications for 1 week. You decide to place him on maintenance medication to sustain complete evacuation with a lubricating stool softenener. Among the following, the agent you are *most* likely to recommend is:
    A. Bisacodyl.
    B. Magnesium hydroxide.
    C. Malt soup extract.
    D. Mineral oil.
    E. Senna.

16. A 3-month-old boy develops diarrhea associated with fever and toxicity. He had delayed passage of meconium at birth and has had constipation since that time. You suspect Hirschsprung disease. The *most* serious complication of this disease is:
    A. Acute enterocolitis.
    B. Dehydration.
    C. Failure to thrive.
    D. Functional megacolon.
    E. Intestinal volvulus.

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