Childhood Functional Gastrointestinal Disorders: Neonate/Toddler

PAUL E. HYMAN,* PETER J. MILLA,† MARC A. BENNINGA,§ GEOFF P. DAVIDSON,‖ DAVID F. FLEISHER,¶ and JAN TAMINIAU§

*Pediatric Gastroenterology, University of Kansas Medical Center, Kansas City, Kansas; †Paediatric Gastroenterology, Great Ormond Hospital for Children, London, England; §Department of Pediatrics, Emma Kinderziekenhuis ACM, Amsterdam, The Netherlands; ‖Gastroenterology Department, Women's and Children's Hospital, North Adelaide, South Australia, Australia; and ¶Department of Child Health, University of Missouri School of Medicine, Columbia, Missouri

Recognizing the importance of childhood functional gastrointestinal disorders in understanding adult functional gastrointestinal disorders, and encouraging clinical and research interest, the Rome Coordinating Committee added a pediatric working team to Rome II in 1999. For Rome III, there was an increase from 1 to 2 pediatric working teams. This report summarizes the current consensus concerning functional disorders in infants and toddlers. Another report covers disorders diagnosed more often in school-aged children and adolescents. The symptoms from functional gastrointestinal disorders in children younger than 5 years depend on maturational factors in anatomy, gastrointestinal physiology, and intellectual and affective functioning. There has been little or no change for infant regurgitation, infant rumination syndrome, or infant dyschezia. Cyclic vomiting syndrome may be diagnosed after 2 rather than 3 episodes. The description of infant colic has been expanded, although there was consensus that infant colic does not reflect gastrointestinal malfunction. The greatest change was in functional constipation. Functional constipation and functional fecal retention in the 1999 report were merged into a single entity: functional constipation. Data-driven changes in diagnostic criteria for functional constipation appear to be less rigid and more inclusive than previous criteria.

Infant and toddler functional gastrointestinal disorders include a variable combination of often age-dependent, chronic or recurrent symptoms not explained by structural or biochemical abnormalities (Table 1). Functional symptoms during childhood are sometimes accompaniments to normal development (eg, infant regurgitation), or they may arise from maladaptive behavioral responses to internal or external stimuli (eg, in functional constipation, retention of feces is a learned response to painful defecation).

The clinical expression of a functional gastrointestinal disorder depends on an individual's autonomic, affective, and intellectual developmental stage and on concomitant organic and psychological disturbances. For an example of development affecting expression of functional symptoms, consider infant regurgitation, which is a problem for a few months of the first year. Similarly, toddlers' diarrhea affects infants and toddlers.

The decision to seek medical care for symptoms arises from a parent's or caretaker's concern for the child. The caretaker's threshold for concern varies with his or her experiences and expectations, coping style, and perception of illness. For this reason, the office visit is not only about the child's symptom but also about the family's conscious and unconscious fears. The clinician must not only make a diagnosis but also recognize the impact of the symptom on the family's emotions and ability to function. Therefore, any intervention plan must attend to both the child and the family.

Effective management depends on securing a therapeutic alliance with the parents. Through the first years, children cannot accurately report symptoms such as nausea or pain. The infant and preschool child cannot discriminate between emotional and physical distress. Therefore, clinicians depend on the reports and interpretations of the parents, who know their child best, and the observations of the clinician, who is trained to differentiate between health and illness.

Table 1. Functional Gastrointestinal Disorders

G. Functional disorders: neonates and toddlers
G1. Infant regurgitation
G2. Infant rumination syndrome
G3. Cyclic vomiting syndrome
G4. Infant colic
G5. Functional diarrhea
G6. Infant dyschezia
G7. Functional constipation

0016-5085/06/$32.00
doi:10.1053/j.gastro.2005.11.065

Disability from a functional symptom is related to maladaptive coping. Childhood functional gastrointestinal disorders are not dangerous when the symptoms and parental concerns are addressed and contained. Conversely, failed diagnosis and inappropriate treatments of functional symptoms may cause needless physical and emotional suffering. In severe cases, well-meaning clinicians inadvertently cocreate unnecessarily complex and costly solutions to functional symptoms, prolonging emotional stress and promoting disability.

G1. Infant Regurgitation

Regurgitation of stomach contents into the esophagus and mouth is common and normal in infants. Uncomplicated regurgitation in otherwise healthy infants is a developmental issue, not a disease. Regurgitation is the involuntary return of previously swallowed food or secretions into or out of the mouth. Regurgitation is distinguished from vomiting, which is defined by a central nervous system reflex involving both autonomic and skeletal muscles in which gastric contents are forcefully expelled through the mouth because of coordinated movements of the small bowel, stomach, esophagus, and diaphragm. Gastroesophageal reflux refers to movement of gastric contents retrograde and out of the stomach. When gastroesophageal reflux causes or contributes to tissue damage or inflammation (eg, esophagitis, obstructive apnea, reactive airway disease, pulmonary aspiration, feeding and swallowing difficulties, or failure to thrive), it is called gastroesophageal reflux disease.

G1. Diagnostic Criteria for Infant Regurgitation

Must include *all* of the following in otherwise healthy infants 3 weeks to 12 months of age:

1. **Regurgitation 2 or more times per day for 3 or more weeks**
2. **No retching, hematemesis, aspiration, apnea, failure to thrive, feeding or swallowing difficulties, or abnormal posturing**

Rationale for change in diagnostic criteria. There have been no changes from the Rome II criteria. The forcefulness of the regurgitation or its outflow through the mouth or nares does not carry any diagnostic relevance. The duration of 3 weeks was chosen because infants come to medical attention more quickly and symptoms cause more parental anxiety than in older children and adults.

Clinical evaluation. Regurgitation occurs more than once a day in 67% of healthy 4-month-old infants. Many parents believe regurgitation is abnormal; 24% bring this symptom to their clinician's attention during their infant's sixth month.[1] Daily regurgitation decreases with age to 5% of infants 10–12 months old. Prematurity, developmental delay, and congenital abnormalities of the oropharynx, chest, lungs, central nervous system, heart, or gastrointestinal tract are risk factors for gastroesophageal reflux disease. Milk allergy may be present when frequent regurgitation is associated with eczema or wheezing. Failure to thrive, hematemesis, occult blood in the stool, anemia or food refusal, and swallowing difficulties should prompt an evaluation for gastroesophageal reflux disease. Moreover, regurgitation associated with early satiety, food refusal, or excessive crying, with or without failure to thrive, may be a consequence of pain or emotional arousal rather than an innocent event. Regurgitation persisting past the first year of life should be evaluated to exclude an anatomic abnormality such as malrotation or gastric outlet obstruction.

A natural history of infant regurgitation is one of spontaneous improvement,[1] and therefore treatment goals are to provide effective reassurance and symptom relief while avoiding complications. Effective reassurance includes the following: (1) an empathetic, accurate response to the stated and unstated fears of the caretakers, such as "What is wrong with my baby? Is it dangerous? Will it go away? What can we do about it?" and (2) a promise of continuing availability and reassessment. Symptom relief requires adjustment in infant care. Left side and prone position[2] and thickened feedings reduce regurgitation.[3,4] No drugs routinely reduce symptoms.[5,6] Improving the maternal-child interaction is often aided by (1) relieving the parent's fears about the infant's symptoms and (2) identifying sources of physical and emotional distress and eliminating them. Stress may be lessened by respite periods, particularly for the mother.

G2. Infant Rumination Syndrome

Infant rumination syndrome is a rare disorder characterized by voluntary, habitual regurgitation of stomach contents into the mouth for self-stimulation. Rumination is regurgitation of recently swallowed food, rechewing, and either reswallowing or spitting out the food. Although rumination is a functional symptom, infant rumination syndrome is a life-threatening psychiatric disorder caused by social deprivation. Rumination

in healthy older children and adults is discussed in other reports in this supplement.

G2. Diagnostic Criteria for Infant Rumination Syndrome

Must include *all* of the following for at least 3 months:

1. Repetitive contractions of the abdominal muscles, diaphragm, and tongue
2. Regurgitation of gastric content into the mouth, which is either expectorated or rechewed and reswallowed
3. Three or more of the following
   a. Onset between 3 and 8 months
   b. Does not respond to management for gastroesophageal reflux disease or to anticholinergic drugs, hand restraints, formula changes, and gavage or gastrostomy feedings
   c. Unaccompanied by signs of nausea or distress
   d. Does not occur during sleep and when the infant is interacting with individuals in the environment

Rationale for change in diagnostic criteria. There have been no changes from the Rome II criteria.

Clinical evaluation. Observing rumination is necessary for diagnosis. Such observations require time, patience, and stealth because rumination may cease as soon as the infant notices the observer. Diagnostic efforts should be directed toward the parents and the infant because infant rumination syndrome results from malfunction in the infant-caregiver relationships.[7]

The emotional and sensory deprivation that prompts rumination may occur in sick infants living in environments that prevent normal handling (eg, neonatal intensive care units). It may also occur in otherwise healthy infants whose mothers are emotionally unconnected. Loss of previously swallowed food may cause progressive inanition and death.[8] Eliminating rumination is accomplished by providing a temporary mother-substitute to hold, comfort, and feed the infant. The nurturing person recognizes when the infant withdraws into the self-absorbed state that fosters rumination and promptly responds by engaging the baby. Treatment includes helping the mother improve her ability to recognize and respond to her infant's physical and emotional needs. Infant rumination may be exacerbated by the noxious stress of the workup that accompanies "diagnosis by exclusion." After rumination subsides, it usually does not recur, even in families whose mental health remains poor.

G3. Cyclic Vomiting Syndrome

Cyclic vomiting syndrome (CVS) consists of recurrent, stereotypic episodes of intense nausea and vomiting lasting hours to days that are separated by symptom-free intervals lasting weeks to months.[9] The frequency of episodes ranges from 1 to 70 per year and averages 12 per year. Attacks occur at regular intervals or sporadically. Typically, episodes begin at the same time of day, most commonly during night or morning. Episode duration tends to be the same in each patient. CVS reaches its highest intensity during the first hours. Vomiting tends to diminish thereafter, although nausea continues until the episode ends. Episodes usually end as rapidly as they begin and are marked by prompt recovery of well-being, provided the patient has not incurred major deficits of fluids and electrolytes. Signs and symptoms that accompany cyclic vomiting include pallor; weakness; increased salivation; abdominal pain; intolerance to noise, light, and/or odors; headache; loose stools; fever; tachycardia; hypertension; skin blotching; and leukocytosis.[10] Eighty percent identify circumstances or events that trigger attacks, such as heightened emotional states, infections, asthma, or physical exhaustion.[10]

G3. Diagnostic Criteria for Cyclic Vomiting Syndrome

Must include *both* of the following:

1. Two or more periods of intense nausea and unremitting vomiting or retching lasting hours to days
2. Return to usual state of health lasting weeks to months

Rationale for change in diagnostic criteria. The working team reached consensus that a diagnosis of CVS requires just 2, not 3 episodes. We expect that this change will facilitate early recognition and reduce patient suffering.

Clinical evaluation. Patients do not vomit between episodes, but two thirds of those who can provide a history have irritable bowel syndrome, and 11% have migraine headaches. Motion sickness occurs in 40%. More than half have family histories of irritable bowel syndrome, and nearly half have a family history of migraine headache. CVS can occur at any age. The differential diagnosis includes diseases having similar presentations during at least part of their courses. Brainstem gliomas may infiltrate the vomiting centers and cranial nerves around the medulla, causing intermittent vomiting without obstruction of cerebrospinal fluid flow or signs of increased intracranial pressure. Occult

upper respiratory tract infection, particularly if associated with vestibulitis, and obstructive uropathy may cause symptoms indistinguishable from CVS. Gastrointestinal causes mimicking CVS include peptic disease with pyloric outlet obstruction, enteropathy especially if there is a marked duodenitis, recurrent pancreatitis, intermittent small bowel obstruction, chronic intestinal pseudo-obstruction, and the vomiting crises of familial dysautonomia. Endocrine and metabolic conditions include pheochromocytoma, adrenal insufficiency, diabetes mellitus, ornithine transcarbamylase deficiency and other urea cycle defects, medium-chain acyl coenzyme A dehydrogenase deficiency, propionic acidemia, isovaleric acidemia (the chronic intermittent form), and porphyria. Pediatric condition falsification may be erroneously diagnosed as CVS.

Treatment. In patients with frequent, severe, and prolonged episodes, daily treatment with amitriptyline, pizotifen (in the United Kingdom and Australia), cyproheptadine, phenobarbital, or propranolol may reduce frequency or eliminate episodes.[11,12] Foods, emotional factors, or physical stressors that trigger episodes may be identified and avoided. Aborting episodes is possible in some children with a recognizable prodrome. Before the onset of nausea, oral medications such as ondansetron or a long-acting benzodiazepine may be useful. During the prodrome, it may be helpful to begin treatment with an oral acid-inhibiting drug agent to protect esophageal mucosa and dental enamel and lorazepam for its anxiolytic, sedative, and antiemetic effects. Deep sleep for several hours may prevent the episode.

Once an episode starts, patients should be sedated until the episode ends. Symptoms may be interrupted by titrating intravenous lorazepam or another long-acting benzodiazepine until the patient enters restful sleep. Intravenous fluids, electrolytes, and H_2-histamine receptor antagonists are administered until the episode is over. Complications during episodes include water and electrolyte deficits, hematemesis due to prolapse gastropathy, peptic esophagitis and/or Mallory–Weiss tears, deficits in intracellular potassium and magnesium levels, hypertension, and inappropriate secretion of antidiuretic hormone. When lorazepam is not effective, the goal continues to be inducing deep sleep so that suffering is eliminated and the patient is amnestic for the episode. Continuous infusions of propofol or pentobarbital or intermittent intravenous diphenhydramine and chlorpromazine are alternatives to lorazepam.

G4. Infant Colic

The term "colic" implies abdominal pain caused by obstruction to flow from the kidney, gallbladder, or intestine. In contrast, "infant colic" is a behavioral syndrome of early infancy involving long crying bouts and hard-to-soothe behavior. Infant colic was defined as "paroxysms of irritability, fussing or crying lasting >3 hours per day and occurring >3 days each week."[13] There is no proof that crying in infant colic is caused by pain in the abdomen or any other body part. Nevertheless, parents often assume that the cause of excessive crying is abdominal pain of gastrointestinal origin.

Because infants with colic are often referred to pediatric gastroenterologists, the Working Team achieved consensus to include infant colic in this report. Familiarity with infant colic is necessary to avoid diagnostic and therapeutic misadventures.[14] Although crying may occur from pain due to inflammation in infants who are sensitive to cow milk protein,[15] by definition, infant colic is not caused by organic disease. Bouts of infant colic start and stop suddenly without obvious cause and are likely to occur late in the day. Crying tends to resolve spontaneously by 3–4 months of age or, in the case of babies born prematurely, 3–4 months after term.[16] On average, crying peaks at 6 weeks and then diminishes by 12 weeks.[17] Infant colic probably represents the upper end of the normal "crying curve" of healthy infants. Colic "is something infants do, rather than a condition they have."[16]

G4. Diagnostic Criteria for Infant Colic

Must include *all* of the following in infants from birth to 4 months of age:

1. **Paroxysms of irritability, fussing, or crying that start and stop without obvious cause**
2. **Episodes lasting 3 or more hours per day and occurring at least 3 days per week for at least 1 week**
3. **No failure to thrive.**

Rationale for Change in Diagnostic Criteria

Rome II excluded infant colic from consideration as a functional gastrointestinal disorder. Nevertheless, the abdominal pain attribution persists and pediatric gastroenterologists receive referrals of babies with refractory colic or infants who cry excessively due to unsuspected colic. The Working Team achieved consensus to include infant colic in the list of childhood functional gastrointestinal disorders because familiarity with the "colic syndrome" is necessary to avoid diagnostic and therapeutic misadventures.

Clinical Evaluation

The colicky crying pattern results from organic disease in <10%. Prolonged, inconsolable crying, crying after feedings, facial grimace, abdominal distention, increased gas, flushing, and legs flexed over the abdomen are not indicative of pain or disease, but they explain parents' concerns. A diagnosis of infant colic can be made in any infant younger than 4–5 months whose crying has temporal features of infant colic, who has no signs of central nervous system or intrinsic developmental difficulties, is normal on physical examination, and has normal growth patterns.

Treatment

Nonanalgesic, nonnutritive soothing maneuvers, such as rhythmic rocking and patting 2–3 times per second in a quiet environment, may quiet the baby who may nevertheless resume crying as soon as he or she is put down. A common maneuver that does not eliminate pain but stops the crying (like a car ride) has diagnostic and therapeutic value. Management consists of helping the parents get through it.[18–20] Any measure that parents perceive as helpful is worth continuing, provided it is harmless. If there is a question of milk intolerance or esophagitis, a time-limited therapeutic trial of a hydrolyzed protein formula or medication to suppress gastric acid secretion is warranted. Relief should be apparent within 48 hours.

G5. Functional Diarrhea

Functional diarrhea is defined by daily painless, recurrent passage of 3 or more large, unformed stools for 4 or more weeks with onset in infancy or preschool years. There is no evidence for failure to thrive if the diet has adequate calories. The child seems unperturbed by the loose stools, and the symptom resolves spontaneously by school age.

G5. Diagnostic Criteria for Functional Diarrhea

Must include *all* of the following:

1. **Daily painless, recurrent passage of 3 or more large, unformed stools**
2. **Symptoms that last more than 4 weeks**
3. **Onset of symptoms that begins between 6 and 36 months of age**
4. **Passage of stools that occurs during waking hours**
5. **There is no failure-to-thrive if caloric intake is adequate**

Rationale for Change in Diagnostic Criteria

There were no changes from the Rome II criteria.

Clinical Evaluation

The clinician queries about recent enteric infections, laxatives, antibiotics, or diet changes. Stools often contain mucus and/or visible undigested food. A diet history will assess overfeeding, excessive fruit juice or sorbitol consumption, excessive carbohydrate ingestion with low fat intake, and food allergy. In the absence of failure to thrive, malabsorption is unexpected.

Treatment

It is important to avoid restrictive diets that may cause calorie deprivation.[21] Children recover spontaneously, and usually no treatment other than effective reassurance for the parents is necessary. A daily diet and defecation diary helps to document that specific foods are not responsible for loose stools.

Disorders of Defecation

Defecation frequency in healthy infants and children decreases with age.[22] Breast-fed infants may defecate as frequently as 12 times per day or as infrequently as once in 3 or 4 weeks. Firm stools may occur from the first weeks of life in formula-fed infants. These infants may experience painful defecation and so have a predisposition toward developing functional constipation (see following text).

There is a decline in stool frequency from an average of more than 4 stools daily in the first week of life to 1–2 each day at 4 years of age. Approximately 97% of 1- to 4-year-old children pass stool 3 times daily to once every other day.[23] Acquisition of bowel and bladder control cannot be accelerated by early or high-intensity toilet training.[24,25] The child's initiative is a reliable indicator that the child is developmentally capable of being clean and dry. Many children achieve partial voluntary bowel control by 18 months, but the age of complete control varies. By 4 years, 98% of normal children are toilet trained. Concerns related to defecation problems are responsible for 25% of outpatient visits to pediatric gastroenterologists.[26]

G6. Infant Dyschezia

Parents describe infants with dyschezia as straining for many minutes, screaming, crying, and turning red or purple in the face with effort. The symptoms

persist for 10–20 minutes, until there is passage of soft or liquid stool. Stools pass several times daily. The symptoms begin in the first months of life and resolve spontaneously after a few weeks.

G6. Diagnostic Criteria for Infant Dyschezia

Must include *both* of the following in an infant younger than 6 months of age:

1. At least 10 minutes of straining and crying before successful passage of soft stools
2. No other health problems

Rationale for change in diagnostic criteria. There were no changes in the diagnostic criteria from Rome II.

Clinical evaluation. The examiner performs a history (including diet), conducts a physical examination (including rectal examination) to exclude anorectal abnormalities, and charts the infant's growth. Parents are reassured by a methodical physical examination completed in their presence. Failure to coordinate increased intra-abdominal pressure with pelvic floor relaxation results in infant dyschezia. The infant's cries increase intra-abdominal pressure. Defecation requires increasing intra-abdominal pressure and simultaneously relaxing the pelvic floor. Coordination of these 2 independent events to effect successful defecation may occur by chance, but eventually defecation is learned. Parents are happy to accept the explanation that the child needs to learn to relax the pelvic floor at the same time as bearing down. To encourage the infant's defecation learning, the parents are advised to avoid rectal stimulation, which produces artificial sensory experiences that may be noxious or that may condition the child to wait for stimulation before defecating. Laxatives are unnecessary.

G7. Functional Constipation

Constipation represents the chief complaint in 3% of pediatric outpatient visits. Approximately 40% of children with functional constipation develop symptoms during the first year of life.[27,28] Sixteen percent of parents of 22-month-old children reported constipation.[29]

G7. Diagnostic Criteria for Functional Constipation

Must include 1 month of *at least 2* of the following in infants up to 4 years of age:

1. Two or fewer defecations per week
2. At least 1 episode per week of incontinence after the acquisition of toileting skills
3. History of excessive stool retention
4. History of painful or hard bowel movements
5. Presence of a large fecal mass in the rectum
6. History of large-diameter stools that may obstruct the toilet

Accompanying symptoms may include irritability, decreased appetite and/or early satiety. The accompanying symptoms disappear immediately following passage of a large stool.

Rationale for change in diagnostic criteria. The change from 12 weeks to 1 month of symptoms is based on data showing that the longer functional constipation goes unrecognized, the less successful treatment is. In constipated children younger than 4 years of age, prognosis was better when the child was treated before 2 years of age.[30] Recovery in children with fecal incontinence was associated with shorter symptom duration.[31,32] Diagnostic criteria for functional fecal retention are eliminated. Functional fecal retention was a term that was not accepted by most clinicians. Functional fecal retention put a high premium on retentive posturing, which was 1 of 2 obligatory diagnostic criteria. However, parents may not have observed or recognized retentive behavior.[33] We now assess parents' impression of excessive stool retention as 1 of 6 criteria, but it is not essential.

Clinical evaluation. Fecal incontinence (involuntary passage of colon contents) may occur in infants and toddlers who accumulate a rectal fecal mass. Two studies assessing the applicability of Rome II criteria recommended that fecal incontinence should be added to revised criteria. Incontinence is an objective marker for monitoring treatment. A painful bowel movement has been identified as an important cause for retentive behavior.[34] A fecal mass identified before evacuation (recognized during physical examination) or after a bowel movement is a feature of functional constipation. The painful evacuation of a fecal mass often conditions a child to avoid defecation.

Functional constipation is a diagnosis made by history and physical examination.[35] No testing is necessary or de-

sirable. Onset frequently occurs during 1 of 3 periods: (1) in infants with hard stools, often corresponding with the change from breast milk to commercial formula or introduction of solids; (2) in toddlers acquiring toilet skills, as they attempt to control bowel movements and find defecation painful; and (3) as school starts and children avoid defecation throughout the school day. Affected children are often described as standing on their toes, holding onto furniture, stiffening their legs, and hiding in a corner. Incontinence may be mistaken for diarrhea by some parents. The physical examination includes assessing the size of the rectal fecal mass, which is judged for height above the pelvic brim with bimanual palpation on either side of the rectus sheath. A rectal examination is performed after establishing rapport with the patient and the family. In functional constipation, the initial attempt at rectal examination occasionally causes the child to react with acute, intense fear and negative behaviors. An irrational fear of the rectal examination typifies the child with functional constipation and is rarely a problem in children with other complaints, including other defecation disorders. When the history is typical for functional constipation, the perineum should be inspected but a digital rectal examination may be delayed to facilitate the therapeutic alliance between the child and the clinician until after a treatment trial fails. If the clinician plans a consultation but no follow-up, a rectal examination is necessary to evaluate the child for the rare obstructing mass. Symptoms are explained by voluntary efforts to avoid defecation. Pain results from normal colonic contractions pushing luminal contents against a closed anal sphincter. Incontinence occurs when stool seeps around the fecal mass and leaks out when the child relaxes the pelvic floor or anal sphincter, either inadvertently (as in sleep), with fatigue, or with attempts to pass flatus. Initiation of intensive training before 27 months does not correlate with earlier completion of toilet training, suggesting little benefit to training before 27 months.[36] Parents of children at risk for functional constipation seem to be inconsistent in their approach to toilet training, varying from rigid to permissive.[37] In most cases, fear of painful defecation is the cause for fecal retention.[38]

Treatment. The first step in treatment is family education during the initial visit. The child and family appreciate a clinician who explains the problem, absence of disease, and safe and effective management. The clinician addresses the myths and fears: the child has functional constipation, the most common problem referred to the pediatric gastrointestinal specialist, and one of the most common problems in pediatrics. It goes away in time, and it is not dangerous. These statements assuage anxiety and create an expectation for positive change. Next, parents need to be helped to understand the child's point of view. For toddlers, toilet training will not proceed until the child's fear of painful defecation resolves. Parents must understand that coercive toilet training tactics are likely to backfire into an unwinnable struggle for control. Next, the clinician, child, and parent agree on a plan for evacuating the rectal fecal mass. Most experts favor a daily nonstimulant laxative such as polyethelene glycol, mineral oil, lactulose, or milk of magnesia, which slowly softens the mass until the child chooses to pass it, days or weeks later. This nonintrusive approach returns the control of the child's pelvic floor to the child. The key to effective maintenance is assuring painless defecation until the child is comfortable and acquisition of toilet learning is complete. Behavior modification using rewards for successes in toilet learning is helpful.

Functional constipation is as common in children and adolescents as it is in infants and toddlers. Aspects of this disorder in the older child are covered in the next report.

Recommendations for Future Research

1. Validating the diagnostic criteria for the childhood functional gastrointestinal disorders will be an important goal for the next decade. Epidemiologic community-based studies and studies of populations believed to be at risk (eg, children of patients with functional gastrointestinal disorders, female child abuse victims) are needed to determine the applicability of the diagnostic criteria, which were arrived at by consensus rather than by data analysis.
2. Clinical trials measuring symptom change as the primary outcome measure will help us to learn which interventions improve outcomes in the childhood functional gastrointestinal disorders.
3. Definitions and diagnostic criteria for currently unrecognized childhood functional gastrointestinal disorders are needed.
4. A history of physical, sexual, and/or emotional abuse will be elicited from some children with functional bowel disorders. There is a need for education and training related to the evaluation and treatment of child abuse for all clinicians who care for children with functional gastrointestinal disorders.
5. The role of childhood functional gastrointestinal disorders in adult functional gastrointestinal disorders should be explored.
6. There is increasing evidence in adult practice of the role of inflammation in some functional gastrointestinal disorders. There are virtually no such studies in childhood. The role of inflammation in childhood functional gastrointestinal disorders requires systematic study.

References

1. Nelson SP, Chen EH, Syniar GM, Christoffel KK. Prevalence of symptoms of gastroesophageal reflux during infancy. Arch Pediatr Adolesc Med 1997;15:569–572.
2. Ewer A, James ME, Tobin JM. Prone and left lateral positions reduce gastrooesophageal reflux in preterm infants. Arch Dis Child Fetal Neonatal Ed 1999;81:2001–2005.
3. Vanderhoof JA, Moran JR, Harris CL, Merkel KL, Orenstein SR. Efficacy of a prethickened infant formula: a multicentre, double-blind, randomized, placebo controlled parallel group trial in 104 infants with symptomatic gastroesophageal reflux. Clin Pediatr 2003;42:483–495.
4. Wenzl TG, Schneider S, Scheele F, Silmy J, Heimann G, Skopnik H. Effects of thickened feeding on gastroesophageal reflux in infants: a placebo-controlled crossover study using intra luminal impedance. Pediatrics 2003;111:e355–e359.
5. Bourke B, Drumm B. Cochrane's epitaph for cisapride in childhood gastrooesophageal reflux. Arch Dis Child 2002;86:1–2.
6. Moore DJ, Tao BS, Lines DR, Hirta C, Heddle ML, Davidson GP. Double-blind placebo controlled trial of omeprazole in irritable infants with gastroesophageal reflux. J Pediatr 2003;143:219–223.
7. Fleisher DR. Infant rumination syndrome. Am J Dis Child 1979; 133:266–269.
8. Sauvage D, Leddet I, Hameury L, Barthelemy C. Infantile rumination: diagnosis and follow-up study of twenty cases. J Am Acad Child Psychiatry 1985;24:197–203.
9. Li BUK (guest ed). Cyclic vomiting syndrome: proceedings of the international scientific symposium on CVS. J Pediatr Gastroenterol Nutr 1995;21(Suppl 1):S1–S–S62.
10. Fleisher DR, Matar M. The cyclic vomiting syndrome: a report of 71 cases and a literature review. J Pediatr Gastroenterol Nutr 1993;17:361–369.
11. Anderson I, Sugerman K, Lockhart JR, Weinberg WA. Effective prophylactic therapy for cyclic vomiting syndrome in children using amitriplytine or cyproheptadine. Pediatrics 1997;100:977–981.
12. Gokhale R, Huttenlocher PR, Brady L, Kirschner BS. Use of barbituates in the treatment of cyclic vomiting during childhood. J Pediatr Gastroenterol Nutr 1997;25:64–67.
13. Wessel MA, Cobb IC, Jackson EB, Harris GS Jr, Detwiler AC. Paroxysmal fussing in infancy, sometimes called colic. Pediatrics 1954;14:421–434.
14. Fleisher DR. Coping with colic. Contemp Pediatr 1998;15:144–156.
15. Lothe L, Lindberg T. Cow's milk whey protein elicits symptoms of infant colic in colicky formula-fed infants: a double blind cross-over study. Pediatrics 1989;83:262–266.
16. Barr RG. "Colic" is something infants do, rather than a condition they "have": a developmental approach to crying phenomena, patterns, pacification, and (patho)genesis. In: Barr RG, SJ-RI, Keefe MR, eds. New evidence on unexplained early crying: its origins, nature, and management. Cincinnati, OH; Johnson & Johnson Pediatric Institute, 2001:87–104.
17. Brazelton TB. Crying in infancy. Pediatrics 1962;29:579–588.
18. Taubman B. Clinical trial of the treatment of colic by modification of parent-infant interaction. Pediatrics 1984;74:998–1001.
19. Meyer EC, Coll CT, Lester BM, Boukydis CF, McDonough SM, Oh W. Family-based intervention improves maternal psychological well-being and feeding interaction of preterm infants. Pediatrics 1994;93:241–246.
20. Wolke D, Gray P, Meyer R. Excessive infant crying: a controlled study of mothers helping mothers. Pediatrics 1994;94:322–332.
21. Lloyd-Still JD. Chronic diarrhea of childhood and the misuse of elimination diets. J Pediatr 1979;95:10–13.
22. Weaver LT. Bowel habit from birth to old age. J Pediatr Gastroenterol Nutr 1988;7:637–640.
23. Weaver LT, Steiner A. The bowel habit of young children. Arch Dis Child 1984;59:649–652.
24. Largo RH, Stutzle W. Longitudinal study of bowel and bladder control by day and night in the first 6 years of life. Epidemiology and interrelations between bowel and bladder control. Dev Med Child Neurol 1977;19:598–606.
25. Largo RH, Molinari L, Von Siebenthal K, Wolofensberger U. Does a profound change in toilet training affect development of bowel and bladder control? Dev Med Child Neurol 1996;38;1106–1116.
26. Taitz LS, Wales JK, Urwin OM, Molnar D. Factors associated with outcome in management of defecation disorders. Arch Dis Child 1986;61:472–477.
27. Davidson M, Kugler MM, Bauer CH. Diagnosis and management in children with severe and protracted constipation and obstipation. J Pediatr 1963;62:261–275.
28. Benninga MA, Büller HA, Taminiau JAJM. Chronic constipation in children; the effect of biofeedback training with a one year follow-up. Arch Dis Child 1993;68:126–129.
29. Issenman RM, Hewson S, Pirhonen D, Taylor W, Tirosh A. Are chronic digestive complaints the result of abnormal dietary patterns? Diet and digestive complaints in children at 22 and 40 months of age. Am J Dis Child 1987;141:679–682.
30. Loening-Baucke V. Constipation in early childhood: patient characteristics, treatment, and long term follow-up. Gut 1993;34: 1400–1404.
31. Loening-Baucke V, Krishna R, Pashankar DS. Polyethylene glycol 3350 without electrolytes for the treatment of functional constipation in infants and toddlers. J Pediatr Gastroenterol Nutr 2004; 39:536–539.
32. Van Ginkel R, Reitsma JB, Buller HA, Van Wijk MP, Taminiau JA, Benninga MA. Childhood constipation: longitudinal follow-up beyond puberty. Gastroenterology 2003;125:357–363.
33. Voskuijl WP, Heijmans J, Heijmans HS, Taminiau JA, Benninga MA. Use of Rome II criteria in childhood defecation disorders: applicability in clinical and research practice. J Pediatr 2004; 145:213–217.
34. Partin JC, Hamill SK, Fischel JE, Partin JS. Painful defecation and fecal soiling in children. Pediatrics 1992;89:1007–1009.
35. Baker SS, Liptak GS, Colletti RB, Croffie JM, Di Lorenzo C, Ector W, Nurko S. Constipation in infants and children; evaluation and treatment. A medical position statement of the North American Society for Pediatric Gastroenterology and Nutrition. J Pediatr Gastrenterol Nutr 1999;29:613–626.
36. Blum NJ, Taubman B, Nemeth N. Relationship between age at initiation of toilet training and duration of training: a prospective study. Pediatrics 2003;111:810–814.
37. Fishman L, Rappaport L, Cousineau D, Nurko S. Early constipation and toilet training in children with encopresis. J Pediatr Gastroenterol Nutr 2002;34:385–388.
38. Cox DJ, Ritterband LM, Quillian W, Kovatchev B, Morris J, Sutphen J, Borowitz S. Assessment of behavioral mechanisms maintaining encopresis: Virginia Encopresis-Constipation Apperception Test. J Pediatr Psychol 2003;28:375–382.

Received March 2, 2005. Accepted November 3, 2005.

Address requests for reprints to: Paul E. Hyman, MD, Pediatric Gastroenterology, University of Kansas Medical Center, Kansas City, Kansas 66160. e-mail: phyman@kumc.edu; fax: (913) 588-8169.