GASTROINTESTINAL PROBLEMS IN THE INFANT

Second Edition

by JOYCE GRYBOSKI, M.D.

GASTROINTESTINAL PROBLEMS IN THE INFANT *Second Edition*

By JOYCE GRYBOSKI, M.D.

Professor of Pediatrics
Yale University
New Haven, Connecticut

and W. ALLAN WALKER, M.D.

Professor of Pediatrics
Harvard Medical School;
Chief, Pediatric Gastrointestinal and Nutrition Unit
Massachusetts General Hospital
Boston, Massachusetts

1983 W.B. Saunders Company
Philadelphia • London • Toronto
Mexico City • Rio de Janeiro • Sydney • Tokyo

W. B. Saunders Company: West Washington Square
Philadelphia, PA 19105

1 St. Anne's Road
Eastbourne, East Sussex BN21 3UN, England

1 Goldthorne Avenue
Toronto, Ontario M8Z 5T9, Canada

Apartado 26370—Cedro 512
Mexico 4, D.F., Mexico

Rua Coronel Cabrita, 8
Sao Cristovao Caixa Postal 21176
Rio de Janeiro, Brazil

9 Waltham Street
Artarmon, N.S.W. 2064, Australia

Ichibancho, Central Bldg., 22-1 Ichibancho
Chiyoda-Ku, Tokyo 102, Japan

Library of Congress Cataloging in Publication Data

Gryboski, Joyce.

Gastrointestinal problems in the infant.

1. Pediatric gastroenterology. I. Walker, W. Allan. II. Title. [DNLM: 1. Gastrointestinal diseases—In infancy and childhood. WS 310 G894g]

RJ446.G78 1983 618.9'23 81-48621

ISBN 0-7216-4329-9 AACR2

Listed here is the latest translated edition of this book together with the language of the translation and the publisher.

Italian (*1st Edition*)—Edizioni Medico Scientifiche, Torino, Italy

Gastrointestinal Problems in the Infant ISBN 0-7216-4329-9

Last digit is the print number: 9 8 7 6 5 4 3 2 1

Contributors

JOEL M. ANDRES, M.D.
Associate Professor
University of Florida College of Medicine
Department of Pediatrics
Division of Gastroenterology
Gainesville, Florida

STANLEY A. COHEN, M.D.
Clinical Assistant Professor
Medical College of Georgia;
Director, Gastroenterology, Nutrition and Pediatric Research
Scottish Rite Hospital for Children
Atlanta, Georgia

KRISTY MARIE HENDRICKS, M.S., R.D.
Research Dietitian
Pediatric Gastrointestinal and Nutrition Unit
Massachusetts General Hospital
Boston, Massachusetts

RONALD ELLIS KLEINMAN, M.D.
Assistant Professor of Pediatrics
Harvard Medical School;
Assistant in Pediatrics
Gastrointestinal and Nutrition Unit
Massachusetts General Hospital
Boston, Massachusetts

ALAN M. LAKE, M.D.
Assistant Professor of Pediatrics
The Johns Hopkins University School of Medicine;
Division of Pediatric Gastroenterology and Nutrition
The Johns Hopkins Hospital
Baltimore, Maryland

SUSAN H. LARAMEE, M.S., R.D.
Instructor
MGH Institute of Health Professions;
Department of Dietetics
Massachusetts General Hospital
Boston, Massachusetts

REBECCA NILOFF, M.D., C.M.
Clinical Fellow in Pediatrics
Harvard Medical School;
Fellow in Pediatric Gastroenterology and Nutrition, Children's Service
Massachusetts General Hospital
Boston, Massachusetts

Preface

In this second edition of the textbook, we have revised the format to allow readers more convenience in addressing gastrointestinal problems that develop during infancy. The textbook is divided into three major parts. The *first* deals with an approach to the evaluation of common gastrointestinal signs and symptoms presenting in infants. In these chapters we attempt to differentiate between developmental, self-limiting problems, such as neonatal jaundice and regurgitation, and potentially life-threatening conditions that must be identified and managed with expedience.

In the *second* part, we have provided an approach to managing the more complicated gastrointestinal problems that result in failure to thrive. This section provides a practical guide to parenteral and enteral nutritional support, which is based on the editors' experience over the decade. In many instances, the consequences of the gastrointestinal disorders are more dangerous to the young infant than the actual problems. Nutritional support to prevent damage to the developing brain or secondary infections due to inadequate host defenses ultimately may be more beneficial to the young infant than an aggressive approach to establishing the primary diagnosis. In fact, in many instances, such as with intractable diarrhea of infancy, the primary basis for the insult is never completely established, and nutritional support represents the most important approach to the problem.

In the *third* part, specific gastrointestinal problems unique to or also encountered in infancy are described. These conditions, as in the first edition, are presented according to location within the gastrointestinal tract. A considerable effort has been made to deal primarily and comprehensively with the most common disorders encountered and yet be as complete as possible. Extensive attempts to update the expanding literature in this area have been made, and chapters are referenced with the most recent articles appearing in the medical literature.

Finally, an expanded appendix has been provided to facilitate the access of practicing physicians to important objective data for interpreting diagnostic tests and for managing patients with specific problems.

As with the first edition of this textbook, the editors have tried to be as complete as possible in providing the most recent information on gastrointestinal problems in infants. However, the tenet of this edition is also clinical and directly primarily toward house officers, practicing pediatricians, and gastroenterologists. We have attempted to answer questions that we ourselves have asked. We are hopeful that this expanded edition will provide an appropriate reference text to be kept in your office for perusal when

you encounter a young infant with symptoms or signs relevant to the gastrointestinal tract. Much of what is written comes from our own collective experience in diagnosing and managing infants in our own practices of pediatric gastroenterology. We hope that it is of benefit to you in your practice.

JOYCE GRYBOSKI
ALLAN WALKER

Acknowledgments

We wish to thank all the individuals who contributed to the quality of this edition. This includes Dr. Ronald Kleinman, who contributed to Part II, on Nutritional Support of the Newborn with Gastrointestinal Problems; Dr. Rebecca Niloff, who contributed to the section on Pancreatic Disease States; Dr. Stanley Cohen, who contributed in Part I on Chronic Diarrhea and in Part III on Infectious and Miscellaneous Causes of Diarrhea; Dr. Alan Lake, who made major contributions to the chapter on Inherited and Metabolic Disorders; Dr. Joel Andres, who contributed to Symptoms of Liver Dysfunction in Part I and Diseases of the Liver in Part III; and Ms. Susan Laramee and Ms. Kristy Hendricks, who diligently prepared much of the Appendix material for this edition.

Finally, we would like to express our gratitude to the Saunders staff: Mary Cowell, Associate Medical Editor, with whom we worked closely and Bill Preston of the Production Department for both his patience and his skill.

Contents

PART I / SIGNS, SYMPTOMS, AND EVALUATION

Chapter One

INTRODUCTION

THE MEDICAL HISTORY

In medicine one is taught that a complete and careful history almost always points to the correct diagnosis. This is no less true in pediatrics, although the fact that the parent — not the patient himself — is the historian may make diagnosis more challenging. The history should cover both prenatal and perinatal events and should include complete dietary information. A history of allergy or gastrointestinal disease in other family members should be sought.

PHYSICAL EXAMINATION

A few minutes spent in simply watching the infant is a rewarding investment of time. One can determine the degree of activity and awareness, state of illness, nutritional status, responsiveness, and whether there is any preferred position of comfort. The physican examination must be complete, and certain points are stressed by the gastroenterologist. The vital signs are recorded, height and weight are plotted on standard linear growth and growth velocity charts. Skin lesions are often associated with intestinal diseases, and café-au-lait spots, hemangiomas, telangiectases, bullae or dystrophic lesions, excoriations, ulcerations, erythema, and edema should be noted. Head size and shape and any peculiarities of facial appearance are recorded. Palatal and pharyngeal function must be evaluated. Examination of the neck includes assessment of the degree of mobility and detection or exclusion of masses and thyroid enlargement. The chest is examined for bowel sounds as well as for pneumonitis and atelectasis. During quiet observation of the abdomen, one notes whether it is distended or scaphoid and whether there are dilated loops of bowel, peristaltic waves, dilated periumbilical veins, granulomas or sinus tracts in the umbilicus, or bulging of hernias. Auscultation of all four quadrants reveals the quality of the bowel sounds and the presence or absence of a bruit. Percussion demonstrates areas of tympany or dullness. Palpation must be gentle so that the infant does not tighten the abdominal muscles; it indicates whether the abdomen is soft or tense, contains a mass, or has any area of localized tenderness. The size and consistency of liver, spleen, and kidneys are noted. The perineum and genitalia are ex-

amined for fistulas, ectopia, or bulging. By rectal examination one looks for pelvic masses and feels for sphincter tone and the presence of stool in the ampulla. Stool on the examining glove is routinely tested for occult blood and pH. Examination of the extremities stresses evaluation of muscle tone and the presence or absence of clubbing.

An estimate of motor development and growth is noted and related to the chronological age (see Chapter Four, in which the causes of growth retardation are discussed).

COMMON GASTROINTESTINAL SYMPTOMS

VOMITING

Vomiting represents the forceful evacuation of gastric contents and is accompanied by gastric and abdominal contractions. Most babies will vomit at some time during infancy for no apparent reason. In some, vomiting may be caused by overfeeding or disturbed mother-child relationships, while in others it is a reaction to systemic illness, urinary tract or respiratory tract infection. Metabolic disorders, such as adrenal insufficiency, renal tubular acidosis, uremia, and particularly hyperammonemia from urea cycle enzyme deficiencies, are associated with intractable and occasionally projectile vomiting. Central nervous system lesions that lead to increased intracranial pressure typically cause vomiting without any preceding abdominal discomfort. The vomiting is most frequent in the morning but may occur at any time. Infants with the diencephalic syndrome remain contented and unconcerned as they vomit to the point of emaciation. Vomiting associated with headache should arouse suspicion of a brain tumor even in the presence of a negative CAT scan, for this may be normal early in the course of disease.[4a, 22a] Cyclic vomiting may be psychologic or may be related to urea cycle enzyme deficiencies, pyloric channel ulcer, or Meniere-like disease due to congenital abnormalities of lymphatics in the inner ear (Table 1–1). Morning dizziness and nystagmus are of help in making this diagnosis.[4a, 22a]

Table 1–1 Causes of Vomiting and Regurgitation

Esophageal	Chalasia Hiatus hernia Achalasia Stricture or stenosis
Gastric	Antral web Pyloric stenosis Antral spasm Gastric ulcer Pyloric channel ulcer
Metabolic	Hyperammonemia Familial protein intolerance Renal tubular acidosis Adrenal insufficiency
Central nervous system or psychologic	Rumination Merycism Increased intracranial pressure Diencephalic syndrome
Lower gastrointestinal tract	Malrotation Duodenal obstruction

Persistent vomiting in the absence of the aforementioned lesions indicates upper gastrointestinal obstruction until proved otherwise. Emesis containing unchanged formula denotes esophageal obstruction, whereas that which is curdled and sour signifies gastric obstruction. Bile-stained vomitus indicates an obstructive lesion below the ampulla of Vater.

REGURGITATION

Regurgitation implies an effortless flow of esophageal contents from the infant's mouth; it may occur during sleep when the infant is prone or when he is awake after feedings. Most infants "spit up" to some degree after feeding, simply to clear residual formula from their mouths. Often formula accompanies eruption of air as a "wet" burp or bubble. If the physician verifies that only small quantities of formula are lost, that weight gain is adequate, and that there is no evidence of feeding problems, reassurance

to the mother is all that is required. If symptoms progress, gastroesophageal reflux must be suspected and investigated.

ABDOMINAL MASS

The time of development of an abdominal mass does not establish whether it is malignant or benign in nature. The consistency is more helpful, for masses that are soft more often represent an abscess or a cystic lesion than a tumor. As noted in Table 1–2, the location of the mass may be a clue to its diagnosis. Although each of the gastrointestinal lesions will be discussed in appropriate sections of the text, it is timely in this section to review the diagnostic techniques in current use.

A flat film of the abdomen is of value in determining a large intra- or extraluminal lesion of the gastrointestinal tract. Localized air-fluid levels or distention of a viscus suggest intrinsic obstruction, whereas displaced loops of bowel suggest an extrinsic mass. The presence of abnormal calcification suggests a teratoma. Liver, spleen, and kidney size may be evaluated from this film.

Ultrasonography is a rapid technique for determining consistency of an intra-abdominal mass and will easily differentiate fluid-filled from solid lesions.[8b] In the upper quadrants it will reveal the texture, consistency, and size of the solid organs, such as pancreas, liver, spleen, and kidneys, and ascertain the size of the external bile ducts and gallbladder (Figs. 1–1 and 1–2).[8b, 37]

Further studies, such as intravenous pyelography and radiologic examination of the gastrointestinal tract, are performed as indicated.

Table 1–2 Abdominal Mass in Infancy

Left upper quadrant	Splenomegaly
	Enlarged kidney
	Wilms' tumor
	Neuroblastoma
	Gastric distention
	Gastric duplication
	Gastric hematoma
	Gastric teratoma
	Pancreatic pseudocyst
	Enlarged left lobe of liver
	Volvulus of small bowel
Right upper quadrant	Hepatomegaly
	Hydropic gallbladder
	Choledochal cyst
	Mesenteric cyst
	Neuroblastoma
	Enlarged kidney
Epigastrium	Left lobe of liver
	Choledochal cyst
	Gastric tumor
	Gastric or duodenal duplication
	Pancreatic pseudocyst
Left lower quadrant	Fecal impaction
	Ovarian tumor or cyst
	Appendiceal abscess and malrotation of colon
Right lower quadrant	Appendiceal abscess
	Lymphangiomatous cyst
	Mesenteric cyst
	Ovarian cyst or tumor
Suprapubic or midabdominal	Fecal impaction
	Omphalomesenteric abnormalities
	Sacral teratoma
	Anterior meningocele
	Distended bladder
	Ectopic kidney

GASEOUSNESS

The usual cause of increased belching or passage of flatus is air-swallowing due to excessive sucking of a nipple with poor flow. Crying, improper feeding techniques, and poor burping are additional factors in accumulation of gastrointestinal air.

Disaccharide intolerances, particularly lactose intolerance, may cause significant gas production within the colon, but in infants are usually accompanied by diarrhea. A simple examination of the stool pH will determine the presence or absence of sugar malabsorption.

The most effective diagnostic tool is observation of the infant during feeding and burping. Silicone-containing preparations are occasionally given to diffuse the air bubbles but are generally not required.

PAIN

Pain is a difficult symptom to interpret in the infant. Normal periods of irritability may represent pain to the insecure or inexperienced mother, and in such instances, observation and examination of the infant

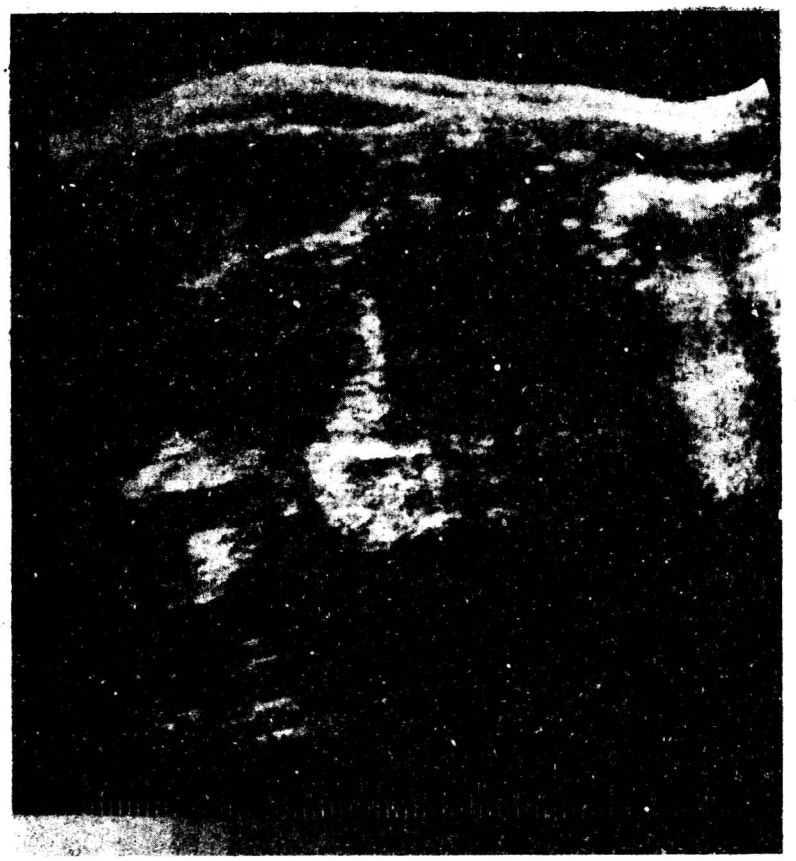

Figure 1–1 Ultrasonographic cross section of abdomen showing duodenal hematoma (*A*) and edematous pancreas (*B*). Skin surface (top) is shown for orientation.

during an attack of pain are crucial to the diagnosis. Some characteristic patterns, may, however, be discerned from a careful history. A cry after a brief period of staring may represent a seizure. The infant with esophageal pain arches his back in an effort to propel a nutrient bolus through an irritated or partially obstructed esophagus and is usually without symptoms between feedings. The pain of pancreatitis and peritonitis is of such severity that the infant lies quite still, has grunting respirations, and resists any efforts to examine his abdomen or to move him.

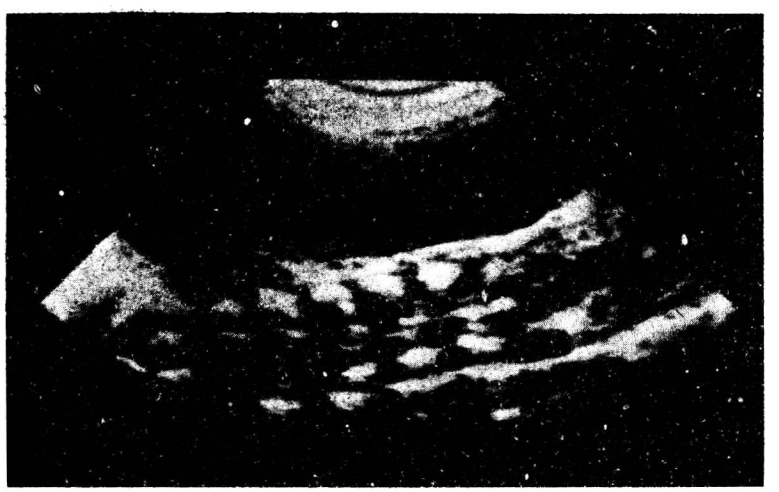

Figure 1–2 Suggested ultrasound study of 800-gm one-month-old infant with hepatomegaly and right lower quadrant mass. The view is such that orientation shows upper abdomen (left) and lower abdomen (right). Mottled opaque material (upper left) represents liver and the large lucent area, an enlarged gallbladder.

Table 1–3 Causes of Abdominal Pain in Infants and Children

Lactose intolerance
Constipation
Milk allergy
Pancreatitis
Peptic ulcer disease
Esophagitis
Inflammatory bowel disease
Gastroenteritis
Duplication cysts
Meckel's diverticulum
Porphyria
Giardiasis
Psychogenic problems
Errors of rotation of the intestine
Ectopic pancreas
Annular pancreas
Spinal cord tumor

Intermittent, acute, or colicky pain may indicate intestinal obstruction or acute gastroenteritis . In the former, stools decrease, whereas in the latter, diarrhea predominates. If the pain persists in a wave-like pattern and is associated with vomiting or decreased stooling, the infant should be evaluated for intestinal obstruction. A sudden loss of consciousness or the passage of currant-jelly stools suggests intussusception. Postprandial discomfort and abdominal distention that seem to be relieved by the passage of flatus suggest disaccharide intolerance.

The causes of abdominal pain are listed in Table 1–3. Depending upon the study and the interests of the investigator,[27] one may identify the more common causes as lactose intolerance,[5, 35a] peptic ulcer disease,[29] pancreatitis,[11] constipation,[16] or a psychosomatic condition.[3] The individual disorders are discussed separately in the text.

Psychosomatic aspects of abdominal pain are less frequent in the infant and toddler than in older children. Apley notes, however, that the "gut is a psychosomatic target organ, and often the main one"[3] throughout childhood years; constipation, colic, and "irritable colon" are found to be more frequent manifestations of psychosomatic symptomatology in children under three years than is abdominal pain. After studying well over 100 children with recurrent abdominal pain, considered to be functional, Stone and Barbero[35] found a high incidence of prenatal and perinatal problems as well as abdominal complaints in other family members. In long-term follow-up, there is a 2 per cent or less incidence of actual intestinal disease.[34] Often, however, one tends to term pain "functional" when a cause cannot be identified. We have seen a teratoma of the lumbar spine and others have reported an astrocytoma of the thoracic cord[8a] in children with entirely negative gastrointestinal evaluations. Each, significantly, had unexplained and persistent scoliosis.

COLIC

Colic is a symptom complex of presumed gastrointestinal etiology that is characterized by unexplained crying or screeching. It is associated with abdominal pain and some inability to pass gas.[8, 23]

Incidence. The incidence varies directly with the criteria used for diagnosis, for these are largely subjective. Approximately 11 per cent of full-term and low birth weight infants have been estimated to suffer from colic, but in a few studies, incidences range as high as 23 to 40 per cent. Caucasians are most often affected, but there seems to be no predilection for either sex.[2, 16]

Etiology. A multitude of factors have been implicated in the quest for the etiology of this disorder. Originally, the most popular theories included family tensions, central nervous system immaturity, allergies, and the position in the family as first-born child. Later, rather extensive studies have discovered no correlation with home environment, finding most mothers to be well educated, intelligent, stable, and cheerful.[32] Indeed, colicky infants at one year appear to be more active and inquisitive than their noncolicky peers. The majority of infants have, however, proved to be first-born and, as symptoms continued over several weeks, did generate conflict in many homes as well as concerns about parental inadequacy. Postulations of soft neurologic damage or central nervous system immaturity cannot be entirely dismissed, for we have seen several such infants grow into hyperactive youngsters.

Milk protein allergy remains a consideration in the diagnosis of colic, for it may be a presenting symptom in some infants before the onset of diarrhea. In recent years, colic in breast-fed infants has been related to the passage of cow's milk protein through breast milk.[20, 25] Nursing mothers are often instructed to drink 1 to 1½ quarts of milk per day, and colic is often relieved by simply decreasing or eliminating milk protein in the diet.

Lactose intolerance, which results in the accumulation of a large volume of colonic gas, may cause abdominal distress prior to the passage of large quantities of explosive stool. Similarly, *high lactulose concentrations* in commercial canned formulas may cause increased colonic gas, for lactulose is a synthetic carbohydrate formed from lactose and is not hydrolyzed by mammalian small intestinal mucosa.

Alprostadil (prostaglandin E_1) is used often in the neonate for the treatment of congenital cardiac defects and may cause intestinal colic and diarrhea.[32a]

Health food–additive colic is a more recently encountered form of the disorder. In this age of physical fitness and emphasis upon organic foods, we occasionally encounter an infant whose mother is supplementing his formula with natural sugars, yeast, pancreatic enzymes, and minerals.

Certain anatomic disorders may cause symptoms resembling those of colic. *Gastroesophageal reflux* may rarely cause repeated episodes of crying, fussing, and difficulty in feeding in infants without apparent regurgitation. *Anal stenosis or anal membrane* was a diagnosis made in five infants referred during the last two years for the evaluation of colic. Examination revealed a slightly anterior location of the anus in four infants and an incomplete anal membrane in the fifth. Anal stenosis was noted in rectal examination of four infants. Excision of the membrane or dilatation of the rectum was curative. *Urinary tract* obstruction, particularly in the ureteropelvic region, and even uncomplicated urinary tract infections have caused symptoms of colic in infants.[14]

Immunoglobulin abnormalities, such as transient IgA or IgG deficiency, have been associated with colic that is accompanied by or followed by chronic diarrhea. In such instances, a hypoallergenic formula will result in cessation of colic.

Feeding problems caused by inadequate nipple size or by leaving an infant with a propped bottle result in excessive air swallowing and the accumulation of large quantities of gastric and intestinal gas.

Pathophysiology. The gastrointestinal tract in true colic is hypermotile, with the stools being either loose or hard and containing mucus. There are increased nonpropulsive contractions in the rectum.

Symptoms. Typically, the onset of colic is at three weeks of age, although the condition may develop any time during the first three months of life. If the symptoms are rather mild and occur for the most part in late afternoon and early evening, the term "paroxysmal fussing" is often used. Brazelton,[6] in a classic study of crying in infancy, noted that the six-week infant had a maximal fussy period between 6 and 8 P.M. and that infants with colic had exaggerated fussiness that progressed until approximately six weeks of age and lasted for a period of four hours or longer for the next two weeks.

In its more obvious form, the attack is sudden in onset, with the infant becoming flushed and the abdomen distended. He cries constantly, often shrieking, and flexes his legs to his abdomen. The attack may last for several hours and is frequently terminated with the passage of stool and flatus. Symptoms improve spontaneously between 10 and 12 weeks, explaining the often-designated term "three months' colic."

Diagnosis. The diagnosis is often based upon a "typical history." However, a complete physical examination, urinalysis, determination of stool pH, and examination of the stool for milk precipitins are probably warranted in most infants. Observation of the infant's behavior and positioning during and after feedings is as essential as the physical examination, for feeding in the supine position and poor burping techniques are often major factors in air-swallowing.

Treatment. Folklore remedies are legion, ranging from fennel tea to a few drops of brandy. Many pediatricians begin thera-

py with antihistamines and, when these prove ineffective, move on to antispasmodics or sedatives, continuing any regime that seems effective. Results with most are often no better than with placebo.[31] Illingworth[23, 24] has found methyscopolamine to be therapeutic, but this has not been confirmed by other experiences. White has noted the most beneficial therapy to be thickened feedings.[36] The old remedies of walking, rocking, or laying the infant over the mother's lap on a warm heating pad offer some degree of solace. Most important in treatment is reassurance to the parents that they or their relationships with their baby are not the cause of his distress.

Prognosis. A number of children seen later for treatment of allergies do have a history of infantile colic. Similarly, Davidson and Wasserman[12] have noted a high incidence of colic and familial constipation or diarrhea in their series of children with the "irritable bowel syndrome."

DISORDERS OF STOOLING

In order to determine the abnormal, one must be aware of the normal frequency of stools. The majority of infants pass their first stool within 36 hours of birth.[9] Retention of stool beyond that period is considered to be abnormal and suggests Hirschsprung's disease, meconium plug, neonatal small left colon syndrome, or low intestinal obstruction.

CONSTIPATION

Constipation is a term describing the consistency of the stool rather than its frequency. Hard, firm, or pellet-like stools indicate constipation, whether they are passed several times a day or once a week. Indeed, infrequent stools are not unusual in the breast-fed infant. In babies, the gastrocolic reflex is most pronounced and the stools are typically passed during or just after feeding. With increasing age and variation in diet, stools become more firm and fewer. The transit time in infants of three to five days is 3 to 13 hours, but by 45 days this increases to longer than ten hours in breast-fed infants but remains less than ten hours in formula-fed ones.[18] Stool frequency, weight, and water content are noted in Table 1–4, showing that although stool frequency decreases and weight increases with age, there is actually little variation in water content of stools passed by infants one week to one year of age. By three years, most children have learned voluntary control and can withhold their movements until they reach the potty chair or toilet.

Etiology. Physiologic constipation is a major cause of this disorder and may well be familial, since one or both parents will have a history of constipation as a child or young adult. It is likely that this is due to an exaggerated water-absorptive mechanism in the colon. Symptoms often become quite marked when the infant is given regular milk containing 4 per cent lactose after taking infant formula containing 7 to 7.5 per cent sugar.

Sensory or motor impairment can cause abnormalities in defecation and result in fecal retention.[19] Those conditions often responsible are spina bifida and myelodysplasia, Hirschsprung's disease, Chagas' disease, neurofibromatosis of the colon, immaturity of the ganglion cells as in prematurity, porphyria, the lipoidoses, and hypothyroidism. A Hirschsprung-like disease due to atrophic myositis of the colon has

Table 1–4 Stool Frequency and Weight in Normal Infants

	1 WEEK	8–28 DAYS	1–12 MONTHS	13–24 MONTHS
No. stools/24 hours	4	2.2	1.8	1.7
Weight	4.3 gm	11 gm	17 gm	35 gm
Water content	72%	73%	75%	73.8%

been reported in Bantu children. One metabolic disorder that has received little attention as a cause of constipation is idiopathic hypercalcemia, or Williams' disease. Anatomic lesions such as anal stenosis or ectopic anus may cause constipation as well as colic symptoms.[21, 26]

The most common cause of constipation, however, is anal fissure, which results from the passage of hard stool and is associated with such pain that it leads to withholding. A vicious cycle is established, for as the fecal bolus remains in the rectum, it becomes even firmer. Rarely, fecal impaction leads to gastrointestinal bleeding from local ulceration or to partial intestinal obstruction. Intermittent or paradoxical diarrhea is frequent in the chronically constipated child and results from the overflow of soft material around the impacted bolus. With increasing colonic distention, the sensation of a distended rectum, needed to initiate defecation, disappears.

In older infants, improper or traumatic toilet training can be the origin of chronic constipation. The use of infant seats that attach over the adult toilet are to be condemned. They not only are frightening, they also are physiologically inappropriate, for they provide no means for the child to brace his feet, a position necessary for adequate abdominal muscle contraction.

Pathophysiology. Physiologically, in constipation due to irritable bowel there is a predominance of contractions that are increased in both number and amplitude.[1, 13] Davidson, in 1956, described a nonpropulsive pattern of colonic motility in normal children and an increased propulsive activity in diarrheal states. He noted that mecholyl caused a relaxation of the phasic activity of the distal colon. In 50 per cent of children with Hirschsprung's disease, this relaxation occurred only in normally innervated distal colons but was absent from the aganglionic segment. Davidson further described three children with "achalasia of the distal rectal segment" who had normal ganglion cells.[14]

Since it is imperative to differentiate Hirschsprung's disease, particularly the short-segment form, from physiologic constipation, considerable research has been done on anorectal function.[33] Internal sphincter relaxation has been demonstrated in most term infants and in some premature ones, the youngest being 34 weeks' gestational age. The sphincter response depends upon postnatal age as well as upon the actual presence of ganglion cells and develops with daily age. Increased anal canal pressure and decreased sensitivity of the internal sphincter relaxation have been noted as early as the first day of life. Cyclic rhythmic contractions in the anal canal, often seen in those infants with Hirschsprung's disease, have been, for some, a normal finding in the neonate.[10]

Meunier et al.[30] studied a variety of rectal responses in constipated children. The rectoanal inhibitory reflex threshold (internal sphincter relaxation in response to transient rectal distention) was increased in 6.2 per cent of constipated children. Anal hypertony was present in 46 per cent and decreased rectal sensitivity to distention, in 68 per cent. Abnormally large amounts of air may be required to fill the rectal balloon in order to initiate internal sphincter relaxation in extremely constipated children. If this is not recognized, Hirschsprung's disease may be erroneously diagnosed. A low mean resting tone of the anus, thought by some to represent a weak internal sphincter, may simply be a result of chronic impaction, since the pressure normalizes after treatment.[29a]

Symptoms. The truly constipated infant has a history of fretfulness, poor appetite, intermittent abdominal pain, and distention. Weight gain may be impaired. Frequently the child has been treated for one or more urinary tract infections. The stools are pellet-like and hard or massive in size. Older infants sometimes perform what Davidson[12] has termed the "duty dance," in which they tighten their gluteal muscles and wiggle in a dance-like motion in order to withhold stool. This may be misinterpreted by the parent as an episode of acute abdominal pain.

Diarrhea or partial intestinal obstruction may be the initial symptom, indicating the need for a rectal examination as an integral part of every physical examination. Stool is

palpable above the pubis and in the left colon. In some, the entire colon, filled with firm masses of stool, is palpable. The abdomen is often distended and tympanitic to percussion. Rectal examination reveals hard stool within the ampulla. Rarely, if the patient is examined after a movement, the ampulla may be empty, a finding typical of Hirschsprung's disease.

Differential Diagnosis. Metabolic and physiologic disorders must be evaluated by physical findings and laboratory studies. Ectopic anus and anal membrane must be ruled out. Thyroid studies and analyses of serum carotene, calcium and phosphorus rule out the common metabolic disorders and celiac disease, which may present with constipation. Of these, celiac disease has been most often found by our group.

Diagnosis. The history and physical examination will often confirm the diagnosis of physiologic constipation, other family members and even a partner twin having constipation.[4] If rectal examination confirms the presence of hard stool in the ampulla, Hirschsprung's disease is unlikely, although the short-segment form cannot be eliminated. Barium enema is usually of no assistance, for it reveals only a dilated, redundant colon, and its use in the infant is not warranted. If there is anything atypical in the history, anorectal manometry is the most fruitful diagnostic tool in differentiating functional constipation from Hirschsprung's disease.

Treatment. The young infant is most easily treated by increasing the carbohydrate content of his diet, using Karo syrup, dextrins, maltose, or sucrose. If a large fecal impaction is present, this is best relieved by a mineral oil retention enema, followed by a normal saline or Fleet pediatric enema (3 ml/kg). Rarely, phosphate enemas cause problems owing to their high osmolarity and tendency to decrease serum calcium. They should therefore be used with caution or not at all in those infants suspected of having Hirschsprung's disease. In older children, addition of fiber to the diet in the form of bran cereals and breads and fresh fruits and vegetables will soften and provide increased bulk to the stools. Of course, adequate fluid intake is essential for all age groups.

If dietary means are not sufficient, mineral oil, 3 to 5 ml/per kg/per day, will, after several days, soften the stools. The initial dose may be adjusted upward if necessary to guarantee several large, loose stools each day to ensure emptying of the colon. The dosage is gradually adjusted downward until the child is passing one large, soft stool per day. Staining of oil or the passage of small quantities of oily stool usually denotes inadequate mineral oil dosages. Several weeks to months of therapy may be required before the child is confident that stooling is without pain and until the sensation for defecation has returned. Lactulose, a nonabsorbable sugar, is also an effective stool softener and bypasses the staining problems of oil. A dosage for children, however, is not yet established.

Proper toilet training is essential to establishing patterns of defecation and should imply a happy, rewarding experience for both parent and child. Training is best not initiated before two years of age and only at a time when the child is responsive. He should be placed in the potty chair at a time when he is most likely to stool and for no longer than ten minutes. If this is not successful, attempts to train should be discontinued for several weeks. Training is facilitated by the use of training pants and clothing that the child may remove easily.

DIARRHEA

Diarrhea describes the consistency rather than the frequency of stools and implies an increased water content. Stools therefore may vary from soft and mushy to liquid. Occasionally, a central core of stool is surrounded in the diaper by a large water ring. The patient's history is extremely important, for what might seem to be a single episode of diarrhea is actually an acute manifestation of a more chronic disorder, such as food allergy, celiac disease, sugar intolerance, or cystic fibrosis. An infectious etiology is most likely if other members of the household are or have been ill. A family