CHAPTER 23

GASTROINTESTINAL ALTERATIONS

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COMPETENCIES

Upon completion of this chapter, the reader will be able to:

- Describe the anatomy and physiology of the gastrointestinal (GI) system of the infant and child and how it differs from the adult GI system.
- Describe the etiology, pathophysiology, clinical manifestations, diagnosis, and treatment of common GI alterations.
- Explain how the pathophysiology is associated with the clinical manifestations of common GI alterations.
- Discuss nursing management and interventions appropriate for children requiring abdominal surgery for specific disorders.
- Identify the educational needs for families and describe appropriate content to be taught by the nurse.

Alterations of the gastrointestinal system can involve the esophagus, stomach, small and large intestine or the accessory organs, the liver, gallbladder, and pancreas. The primary function of the system is ingestion, digestion, absorption of nutrients essential for normal growth and maintenance of fluid and electrolyte balance, and elimination of waste products. These functions are vital for normal growth and development of infants and children. Gastrointestinal complaints are common in this age group. Alterations of function can be expressions of congenital anatomic abnormalities or alterations acquired after birth from disease or infection. The severity of gastrointestinal problems ranges from minor illnesses causing inconvenience to severe, life-threatening disorders such as intestinal obstruction.

This chapter presents a discussion of the anatomy and physiology of the GI system and how it compares to the adult system and the most common GI alterations and disorders. These include upper and lower gastrointestinal alterations, alterations in motility, inflammatory disorders, malabsorption disorders, poisoning and hepatic disorders.

ANATOMY AND PHYSIOLOGY

In comparison with adults, the newborn has a very ineffective gastrointestinal system because of its immaturity at birth. Sucking and swallowing are automatic reflexes initially, gradually coming under voluntary control as the nerves and muscles develop by 6 weeks of age. The newborn’s stomach capacity is only 10 to 20 ml, but expands rapidly to 200 ml by one month of
age and reaches adult capacity of 2000–3000 ml by late adolescence. **Peristalsis**, the coordinated, rhythmic, serial contraction of the smooth muscle of the GI tract, is greater in the infant than in the older child. The emptying time of the stomach increases from 2 to 3 hours in the newborn to 3 to 6 hours by one to two months of age. These factors, the small stomach capacity, increased peristalsis, and increased stomach emptying rate, result in the need for small, frequent feedings. The infant's metabolic rate is faster than an adult's, thus requiring approximately 100 calories per kilogram of body weight compared with 30 to 40 for an adult. Regurgitation is common in the infant because the lower esophageal sphincter tone is decreased or relaxed.

The length of the small intestine is proportionately greater in an infant than an adult: six times the body length in infancy as opposed to four times the height of the adult. However, the infant's intestine is supplied with an adult's proportion of functional secretory glands per unit of area; therefore, an infant secretes proportionately more fluids and electrolytes into the intestine than does an adult. Similarly, the infant's small intestine has a larger surface for absorption relative to body size than does an adult's. Therefore, if diarrhea develops, more electrolytes will be lost from the intestinal secretions. In contrast, the large intestine of the infant is proportionately shorter than an adult's, resulting in less epithelial lining available for absorption of water from the feces. These two characteristics, more secretions and less absorption, are responsible for the soft, frequent stools of infants. Liver functions are also immature at birth; therefore, toxic substances are inefficiently detoxified and medications are inefficiently processed. Hence, the therapeutic dosage of drugs must be adjusted during the first few months of life to prevent them from reaching toxic levels. The processes of gluconeogenesis, deamination, plasma protein and ketone formation, and vitamin storage are immature during the infant's first year.

The infant is deficient in several digestive enzymes that are usually not sufficient until 4–6 months of age. The pancreatic enzyme **amylase**, responsible for the initial digestion of carbohydrates, is insufficient resulting in an intolerance of starches. If cereals are given before 4–6 months, the infant may develop gas and diarrhea. The enzyme **lactase** breaks down or hydrolyzes lactose, the primary source of carbohydrates in infant formula and breast milk. Lactase levels are low in the preterm infant, increase in infancy, and decline after early childhood. This initial decreased level results in incomplete absorption of lactose, which can cause gas, abdominal distention, and diarrhea. Digestion and absorption of fats is impaired because of low levels of the enzyme **lipase**. Fat in breast milk is absorbed more readily than in formula because human milk contains lipase. Protein digestion and absorption are fairly efficient in the newborn and infant. The infant intestine is more permeable to proteins than the older child or adult, thus allowing passage into the bloodstream of cow's milk protein and other potential allergens. Therefore, infants ingesting formula instead of breast milk are more susceptible to food protein allergens. Breastfed infants receive protective immunoglobulin proteins from human milk whereas formula-fed infants do not. Figure 23-1 illustrates the gastrointestinal tract of the child.

### Upper Gastrointestinal Alterations

Upper gastrointestinal alterations commonly found in children include hypertrophic pyloric stenosis, cleft lip and cleft palate, and esophageal atresia and tracheoesophageal fistula.

#### Hypertrophic Pyloric Stenosis

Hypertrophic pyloric stenosis (HPS) is the most common intra-abdominal condition requiring surgery during the neonatal period. Figure 23-2A illustrates a normal pyloric opening; Figure 23-2B shows pyloric stenosis.

#### Incidence and Etiology

HPS affects 1 to 3 per 1,000 live births and four to five times as many males as females. Caucasian males, especially first born, are the group most commonly affected. It is more common among Caucasians of Northern European ancestry, less common among African-Americans, and rare in Asians. The exact cause is not known; however, several theories have been proposed to explain the etiology. Environmental

### Reflections from Families

My son, Robert, is three weeks old and had been healthy since he was born. I am breastfeeding him, and he feeds vigorously and has gained weight. All that changed two days ago when he began vomiting after every feeding. The vomiting has become more forceful, and it looks like curdled milk. He always seems hungry and eagerly sucks when I feed him. He feeds for about five minutes before he starts vomiting. He cried almost all night, and his diaper was dry this morning. I’m so upset and worried that something is seriously wrong with him. I feel so guilty because I can’t console him for long. I can’t even satisfy his hunger. What have I done to cause him to be so sick?
CHAPTER 23 Gastrointestinal Alterations

Figure 23-1 Gastrointestinal Tract of a Child

Figure 23-2 (A) Normal Pyloric Opening; (B) Pyloric Stenosis
factors, allergies, pylorospasm, and muscle enzymes are just a few of the unproven etiologies. Genetic predisposition seems to increase the risk of HPS (Cook, Lopez, & Manfredi, 1996).

Pathophysiology
The pylorus is the opening through which food passes from the stomach to the intestines. This opening is surrounded by a muscular ring, the pyloric sphincter. In HPS the pyloric sphincter hypertrophies and increases to four times its normal width, resulting in a narrowed opening and gastric outlet obstruction (Cook, et al., 1996). This obstruction prevents gastric contents from emptying into the duodenum.

Clinical Manifestations
Symptoms usually develop during the third and fourth weeks of life. Nonbilious vomiting beginning between the second and fourth week of life is the initial symptom. Because of the progressive nature of the obstruction, the vomiting increases in frequency and eventually becomes projectile with vomitus being propelled up to several feet. The emesis is not bile stained because the obstruction occurs above the outlet of the bile duct. The infant is hungry in spite of the vomiting and will usually feed again. Because food does not pass through the pylorus, bowel movements are small. As vomiting continues, there is loss of fluid, leading to dehydration, and hydrogen and chloride ions are lost, leading to hypochloremic metabolic alkalosis. Serum potassium levels are usually maintained, but there may be a total body potassium deficit. The infant has poor weight gain or experiences weight loss and becomes increasingly irritable and lethargic as dehydration and electrolyte imbalances worsen.

Diagnosis
Diagnosis may be made on history and physical identification of the hypertrophic pylorus, which can usually be palpated as an olive-shaped mass in the epigastrium, above and to the right of the umbilicus. If the olive-shaped mass is felt, the diagnosis is confirmed. However, in many cases the enlarged muscle cannot be felt and prompt diagnostic imaging is required for further evaluation. The diagnosis may be confirmed with a barium upper gastrointestinal (UGI) series or an abdominal ultrasound. The UGI, if positive, will reveal a delay in gastric emptying and a narrow, elongated pyloric channel, referred to as a “railroad track” sign (two narrow channels) or a “string sign” (one narrow channel). Ultrasonography is becoming the diagnostic method of choice because it is highly accurate (direct visualization of the muscle hypertrophy and the pyloric channel) and lacks the ionizing radiation inherent in a radiologic procedure such as the upper gastrointestinal contrast series (Deluca, 1993).

Treatment
A surgical procedure called a pyloromyotomy is the treatment of choice in which the circular muscle fibers are released opening the passage from the stomach into the duodenum. Preoperatively, a nasogastric (NG) tube may be inserted, and the stomach emptied. Fluid, acid-base, and electrolyte losses must be corrected for 24 to 48 hours before surgery. Intravenous fluids and electrolytes are administered until the infant is rehydrated and the serum bicarbonate concentrations are less than 30 mEq/dl, indicating that the alkalosis has been corrected.

Postoperatively, the NG tube should be removed, unless there is a reason to keep it in place, such as injury to or perforation of the duodenum. The blood glucose, electrolytes, and complete blood count (CBC) should be monitored. Intravenous glucose should be continued until the infant is able to feed normally. Gastric motility is delayed for up to 24 hours following anesthesia. Therefore, feeding should begin slowly and advance cautiously. The surgical treatment for HPS has a high success rate and is considered curative.

Nursing Management
Assessment
In the nursing history the relationship of feeding to vomiting is determined, and the frequency, color, and amount of emesis is documented. Strict intake and output records are
essential to assess the status of the infant’s hydration. Signs of dehydration are noted such as inelastic skin turgor, crying without tears, dry mucous membranes, a depressed anterior fontanel, urine output <1 cc/kg/hr, increased pulse, decreased blood pressure, and weight loss. The infant is observed for evidence of pain or discomfort, which does not occur except that of chronic hunger.

**Nursing Diagnoses**

Nursing diagnoses for the infant with HPS include:

1. Deficient fluid volume related to the effects of frequent vomiting.
2. Imbalanced nutrition: Less than body requirements related to vomiting and gradual reintroduction of feedings.
3. Pain related to surgical trauma.
4. Risk for infection related to surgical incision.
5. Deficient knowledge (caregivers) related to care of infant after discharge.

**Outcome Identification**

1. The infant will demonstrate improved fluid and electrolyte balance.
2. The infant will tolerate feedings and will demonstrate adequate nutrition by maintaining or regaining pre-admission weight.
3. The infant will experience minimal postoperative pain.
4. The infant's surgical incision will remain free of infection as evidenced by decreased swelling without redness or purulent discharge.
5. Caregivers will verbalize an understanding of incision care, feeding techniques, and signs and symptoms of complications (recurrent vomiting, wound infection, failure to gain weight).

**Planning/Implementation**

Preoperative nursing care focuses on rehydration and correction of the electrolyte imbalance. Daily weights obtained at the same time of day using the same scale are the best indicator of extracellular deficient fluid volume. Because vomiting will continue until surgical correction, the infant is given nothing by mouth; thus, maintaining a patent intravenous infusion is essential. Monitoring the infusion, intake and output, and urine specific gravity are important nursing activities in fluid replacement. Family members need to be reminded to save diapers for weighing to measure urine output. If NG suction is used to decompress the stomach preoperatively, the nurse’s responsibility is to maintain its patency and record the amount, color, and type of drainage. Laboratory data are assessed for electrolyte abnormalities. The nurse continually assesses the infant's hydration status.

Postoperative care includes maintaining fluid and electrolyte balance by (1) monitoring intravenous infusion until oral fluids are tolerated; (2) monitoring infant’s response to feedings by mouth; and (3) assessing for signs of dehydration. Appropriate analgesics are given for pain. The incision site is monitored for signs of infection, such as redness, inflammation, purulent drainage, or temperature of 101°F or higher. Most surgeons remove the NG tube immediately after surgery and begin feeding within 4 to 6 hours if bowel sounds are normal. Initial feedings consist of small amounts of an electrolyte solution such as Pedialyte, and the volume is gradually increased. If greater volumes are tolerated without vomiting, formula or breast milk is offered. Most infants experience some vomiting in the first 24 to 36 hours after surgery; therefore, intravenous fluids are administered until full feedings are tolerated.

**Evaluation**

Evaluation of nursing care is based on how effectively the identified outcomes were met. When feedings are resumed, the infant should be able to tolerate feedings without vomiting, and weight should be gained to the pre-illness amount. The surgical incision should heal without signs of infection. The caregivers need to be able to demonstrate correct care of the incision, state plans for feeding and caring for the infant at home, and verbalize the signs and symptoms of complications and when to contact their health care provider.

**Family Teaching**

Caregivers often feel ineffective because their baby has been hungry and, yet, they have not been able to satisfy this hunger. They may believe they have done something wrong. Nurses can support them by explaining that they are not at fault and the condition is caused by a structural defect. They need to be encouraged to be involved in caring for the baby before and after surgery. Prior to surgery the infant is irritable, hungry, and cries often. Caregivers can be involved by holding, rocking, and cuddling their baby. A pacifier may satisfy the infant's sucking needs.

Nurses should instruct caregivers about the care of the incision (if any is required) and signs of infection. The infant’s response to feedings should be observed. Vomiting may still be present; however, if it persists beyond 48 hours, the health care provider should be notified.

**Cleft Lip (CL) and Cleft Palate (CP)**

A cleft is a fissure or elongated opening. A cleft of the lip, palate, or both is one of the most common congenital anomalies of newborns. Most afflicted will have both cleft lip and palate; some have only a cleft of the lip and others only of the palate. Any type of cleft interferes with the development
of the normal anatomic structures of the lips, nose, muscles, and palate. The degree to which these structures are incomplete or malformed depends on the type, placement, and severity of the cleft(s).

Incidence and Etiology
The incidence of cleft lip and/or palate (CL/CP) is 1.5 in 1,000 births (Czeizel, Timar, & Sarkozy, 1999). The incidence is highest in Asians, followed by Caucasians, and is lowest in African-Americans. Clefts of the lip with or without cleft palate are more common in males, while clefts of the palate alone are more common in females.

Possible etiologies include genetic and environmental factors. If there is a family history of a cleft, the risk of other children also having a cleft is higher. Environmental factors have also been identified as a possible etiology of CL/CP including parental age, maternal intake of excessive alcohol, maternal drug exposure to phenytoin (Dilantin) or diazepam (Valium), and dietary factors such as folic acid and vitamin deficiencies.

Pathophysiology
The hard palate is the bony front part of the roof of the mouth. The soft palate lies behind the hard palate and is composed of muscle and fibrous tissue. The flap of mucosa that hangs down from the soft palate is the uvula. Cleft lip is caused by a failure of the nasal and maxillary processes to fuse between the 5th and 8th week of gestation. The lip and palate develop independently; therefore, it is possible to have either a cleft of the lip or the palate separately or together. Cleft palate is caused by the failure of the palatine plates to fuse between the 7th and 12th weeks of gestation.

Clinical Manifestations
Cleft lip can occur as either unilateral (only on one side) or bilateral (both sides) and can vary from a slight notch in the red portion of the lip to a complete separation extending into the nostril. Cleft palate can occur in the hard or bony palate and/or in the soft palate, with or without a cleft lip being present (Figures 23-3A and 23-3B).

Diagnosis
Cleft lip, and in most cases, cleft palate are obvious at birth. Even a small cleft of the palate can be detected by visual inspection and palpation. When cleft palate is not diagnosed at birth, formula coming from the nose may be the first sign. Both of these defects can be diagnosed in utero by ultrasound, and if present, the family will be referred to a multidisciplinary team at a cleft palate, craniofacial, or orofacial center.

Treatment
The treatment for a child with a cleft lip and palate is complex and involves many specialists, including a plastic surgeon, neurosurgeon, orthodontist, otolaryngologist, pediatrician, nurse, speech pathologist, and audiologist. Reconstruction begins in infancy and can continue through adulthood. Wide variations exist in the timing and technique for surgical repair. Closure of the lip is usually performed when the infant is approximately 3 months of age or 12 pounds. The goal of surgery is to close the cleft so scarring...
will be minimal, and the face will have an increased chance to develop normally. Clefts of the hard and/or soft palate are surgically closed at approximately one year of age to assist feeding and to promote speech and language development. Additionally, good nutritional status and general health are essential factors influencing timing for surgery. Long-term consequences of cleft lip and palate may include speech difficulties, malocclusion problems (abnormal tooth eruption pattern), and hearing problems from recurrent otitis media caused by abnormalities of the eustachian tube.

**Nursing Management**

**Assessment**

A cleft of the lip and usually the palate are observable at birth. During the newborn assessment the nurse examines the palate by visualization and palpation with a gloved finger. A description of the location and extent of the defects is documented. The neonate’s ability to suck, swallow, and feed are also noted. Nurses must also assess the caregiver’s reactions as the birth of a baby with a cleft may be devastating.

**Nursing Diagnoses**

Nursing diagnoses for the infant with cleft lip and/or palate include:

**Preoperative:**

1. **Imbalanced nutrition: Less than body requirements related to feeding difficulties.**

2. **Altered parenting related to interruption in the bonding process.**

**Postoperative:**

3. **Risk of injury and infection to the surgical site related to surgical procedure.**

4. **Pain related to surgical correction of clefts.**

5. **Deficient knowledge related to the condition, treatment, and long-term care.**

**Outcome Identification**

1. Infant will consume adequate nutrients.

2. Caregivers will demonstrate feeding techniques that provide adequate nutrients.

3. Caregivers will begin to adjust and bond to their infant.

4. Infant will maintain optimum comfort.

5. Infant's incision will heal without disruption or infection.

6. Caregivers will verbalize understanding of treatment plan, feeding and restraint techniques, surgical site care, and need for possible later surgeries and speech therapy.

**Planning/Implementation**

Preoperatively, nursing care focuses on providing support for the caregivers, preventing aspiration and infection, and ensuring adequate nutrition. The birth of a child is usually a time of joy and celebration; however, the birth of a child with craniofacial anomaly has potentially devastating effects on a family. The initial reactions are shock, grief, feelings of isolation, feelings of failure or inadequacy. Shock is usually followed by anger, guilt, frustration, and depression. Caregivers may become preoccupied with the baby’s appearance and experience negative feelings toward the infant, which may disrupt or delay attachment. Nurses working with these families must realize that these are normal reactions and that they can aid in the bonding process by demonstrating acceptance of the baby and by encouraging the caregivers to hold and touch their infant. Fears may be allayed by seeing before and after photographs of successful surgical repairs. Providing an opportunity to talk with other families who have a child with a cleft is also important. Agencies such as the Cleft Palate Foundation provide information and support for children and their families (see Resources).

Once the initial shock has been dealt with, the caregivers usually have many questions pertaining to the child’s condition. The four most frequently asked are:

1. **Why did this happen?**
2. **Is this hereditary?**
3. **What can be done? Can anything be done right away?**
4. **What about our baby’s future? Will my child be normal?**

Feeding problems, such as poor or inadequate suction, prolonged feeding time, frequent nasal regurgitation, and inadequate weight gain, can be a frustrating and exhausting experience for many caregivers. Early teaching by nurses about the anatomy and functioning of the palate and successful feeding techniques can decrease caregiver anxiety. When an infant sucks, the soft palate rises up closing off the nasopharynx from the oropharynx, thereby creating negative pressure. This mechanical vacuum draws liquid into the

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**Nursing Tip:**

Caring for families whose child has a cleft lip/palate

Help caregivers to understand this condition by explaining that:

1. Clefting occurs by the 35th day after conception, which is often before a woman knows she is pregnant.

2. The mother needs reassurance that she did nothing wrong during the pregnancy.

3. Many caregivers feel guilty about having a child with this disorder. Counsel caregivers appropriately.

4. Nothing is missing from their child’s face. The pieces just need to be put together.
mouth and delivers it to the back of the throat where it is swallowed.

Breastfeeding an infant with a CL/CP is one option for the mother. The infant with only a CL will probably have no more difficulty than other babies in achieving effective breastfeeding. The breast itself tends to fill the opening in the lip because it has the capacity to mold to the shape of the oral cavity. It may be possible to breastfeed an infant with both CL and CP; however, if this method is unsuccessful, a breast pump may be used to express the milk and bottle feeding with special nipples should be used.

If the method chosen is bottle feeding, it is important for caregivers to initially try a regular nipple and bottle as some infants with small clefts may feed satisfactorily without special adaptations. One method using readily available standard nipples and bottles that is inexpensive and convenient is the Enlarge, Stimulate, Swallow, and Rest (ESSR) method. Enlarging the nipple hole by making a cross cut allows the infant to receive formula in the back of the throat for swallowing, thus bypassing the sucking problem. The next step, stimulate, refers to stimulating the sucking reflex by rubbing the nipple on the lower lip. The nipple is inserted into the mouth, and then the bottle is inverted. The infant swallows the fluid normally. The last step is a rest.

Shortly before infants choke or gag, their facial expression will signal a need for a short break to finish swallowing formula already in their mouth. The signal consists of elevating eyebrows and wrinkling of the forehead. The nipple should be removed slowly and gently from the mouth. Frequent burping is needed. These steps are repeated until the infant has consumed normal amounts of formula, 3 or 4 ounces, in a normal amount of time, 15 to 30 minutes (Richard, 1991). Figure 23-4 illustrates a cross-cut nipple.

If standard nipples are ineffective for feeding, a variety of special nipples, for example, soft, “preemie,” or elongated, are commercially available. If the infant is unable to ingest adequate milk using any of these types of nipples, an asepto syringe with a rubber tip may be effective.

The major emphasis after surgery for cleft lip repair is the protection of the operative area. A small metal strip called a Logan bow or a butterfly adhesive may be placed over the upper lip and taped to the infant’s cheeks to prevent tension on the suture line (Figure 23-5). The infant should be placed only on the back or side and arm or elbow restraints applied to prevent touching or pulling the site. These restraints should be removed periodically to exercise the arms (Figure 23-6). Adequate pain medication needs to be administered to minimize crying and stress on the suture line.

Evaluation

Evaluation is based on how effectively the outcomes of nursing management are met. The infant consumes adequate nutrients and gains weight along a normal growth curve. Caregivers demonstrate increased feelings of confidence with feeding techniques and routine. Caregivers begin to

Figure 23-4 Cross-cut in Nipple. When the nipple is squeezed, the hole can be seen to have been enlarged slightly. From Golding-Kushner, K. J. (2001). Therapy techniques for cleft palate, speech, & related disorders. San Diego, CA: Singular Thomson Learning.
bond with their infant by stroking, touching, and nurturing appropriately. The infant appears content while resting and displays behaviors consistent with comfort. Caregivers verbalize understanding of CL/CP pathology, treatment plan, home care, and long-term care.

Family Teaching
Family teaching includes information about feeding techniques and care of the operative site. Nurses should instruct caregivers to clean the suture line after feeding and as necessary with cotton tipped applicators dipped in diluted hydrogen peroxide. Small amounts of water should be offered after feedings to rinse away any milk residue that could lead to bacterial growth. The elbow restraints should be removed one at a time several times each day for about 10 minutes. If the infant had a cleft lip repair, a side or back lying only position should be used. Nurses should discuss the possibility of additional surgeries on the lip, nose, and palate as the child grows and matures.

Esophageal Atresia and Tracheoesophageal Fistula
Esophageal atresia (EA) and tracheoesophageal fistula (TEF) are congenital defects of the esophagus. They can each occur as a single entity, but usually occur together. EA is characterized by incomplete formation of the esophagus so it terminates before reaching the stomach. It is usually associated with a fistula between the trachea and the esophagus (TEF). Many anatomic variations of EA with or without TEF have been described and are illustrated in Figure 23-7. The five types are (1) esophageal atresia with distal tracheoesophageal fistula (upper segment of the esophagus ends in a blind pouch; lower segment is connected to the trachea by a fistula) (87%); (2) isolated or pure esophageal atresia (blind pouch of upper and lower segments of the esophagus without a connection to the trachea) (8%); (3) tracheo-esophageal fistula without esophageal atresia (intact esophagus with fistula between the esophagus and trachea; “H-type”) (4%); (4) esophageal atresia with proximal tracheoesophageal fistula (blind pouch at each end of the esophagus with a fistula from the trachea to upper segment of the esophagus) (<1%); (5) esophageal atresia with proximal and distal tracheoesophageal fistula (both upper and

Nursing Alert:
Avoiding Hard Objects in the Oral Cavity
Hard objects such as thermometers, tongue depressors, straws, and forks should not be allowed in the child’s mouth until healing has adequately progressed after repair of a cleft palate.
lower segments of esophagus connect to the trachea) (<1%) (Herbst, 1996).

Incidence and Etiology
Esophageal atresia with TEF occurs in 0.2 in 1,000 births, with an equal incidence in the sexes (Clark, 1999). The birth weight of infants with this anomaly is significantly lower than average. Esophageal atresia has been associated with prematurity. Associated congenital anomalies occur in approximately one-half of these infants. The presence and severity of these anomalies are thought to be the most important factor influencing mortality. Cardiac anomalies, such as ventricular septal defect, patent ductus arteriosus, and tetralogy of Fallot are encountered in approximately 30% of all cases. Refer to Chapter 25 for a discussion of these anomalies. Gastrointestinal anomalies including imperforate anus and malrotation may occur in 25% of these infants. Musculoskeletal defects are also common and include vertebral malformations. The acronym VACTERL has been used to describe the condition of multiple anomalies in infants with tracheoesophageal defects:

V—Vertebral defect
A—Anorectal malformation
C—Cardiac defects
T—Tracheoesophageal fistula

Research Highlight

Short Stature in Children with Orofacial Clefts (Cleft Lip/Cleft Palate)

Study Purpose
To assess stature in children with orofacial clefting to determine whether this population is at risk for short stature. In subjects with growth failure (less than the 5th percentile in height), the purpose was to assess hypothalamic-pituitary function and ascertain whether growth failure or hypothalamic dysfunction was related to age, sex, or type of cleft.

Methods
Forty children, ranging in age from 3 to 12 years, with orofacial clefts were measured. Those who demonstrated growth failure were to have further evaluation to determine if hypothalamic-pituitary dysfunction was the cause of the short stature. Data were also collected on age, sex, and type of cleft.

Findings
The group of children with orofacial clefting contained significantly more individuals than expected whose heights were less than the 10th percentile for age and sex. Five children in this study were less than the 5th percentile for height. The parents of four of the five children with growth failure refused further evaluation. The one child who was evaluated had normal hypothalamic-pituitary function. Growth failure was not related to age or type of cleft; however, it was related to sex. More girls exhibited growth failure than boys.

Implications
The high rate of growth failure in this population emphasizes the need for nurses caring for these children to incorporate measurement of growth into their assessment. If growth failure is demonstrated, these children should be referred for evaluation of the etiology of their short stature. Additionally, all health care providers should monitor growth as a component of a child’s assessment, regardless of the health care setting. Short stature should not be ignored or minimized in populations of children having other significant health care problems.

Citation
Pathophysiology
The esophagus and trachea derive from the common primitive foregut (embryonic digestive tube from which the pharynx, esophagus, stomach, and duodenum form) during the fourth and fifth weeks of embryonic development. This foregut lengthens and separates the esophagus from the trachea during the sixth to eighth week. EA and TEF are caused by defective separation. EA as an isolated anomaly occurs rarely. The atresia is attributable to failure of the recanalization of the esophagus.

Clinical Manifestations
Typically, the neonate with EA/TEF presents with copious, fine, frothy bubbles of mucus in the mouth and sometimes the nose. These secretions may clear with aggressive suctioning but eventually return. The infant may have rattling respirations and episodes of coughing, choking, and cyanosis. These episodes may be exaggerated during feeding. If a fistula between the esophagus and the trachea is present, abdominal distention develops as air builds up in the stomach.

Diagnosis
A history of maternal polyhydramnios, an excessive amount of amniotic fluid, should suggest the possibility of a high gastrointestinal obstruction, which prevents the fetus from swallowing and absorbing the fluid. The inability to identify the fetal stomach bubble on a prenatal sonogram in a mother with polyhydramnios makes the diagnosis of EA more likely. If it is suspected, after birth a radiopaque nasogastric or feeding tube should be passed through the nose to the stomach. In infants with atresia, the tube typically stops at 10–12 cm. The normal distance is 17 cm (Clark, 1999). The type of esophageal abnormality is further determined by radiographic studies. When present, curling of the tube in the upper esophageal segment is shown on radiography. If TEF is present, air will be seen in the stomach because of the connection between the esophagus and trachea. The absence of air in the stomach indicates EA without TEF.

Treatment
Before the performance of the first successful repair in 1939, this condition was fatal. However, over the past 50 years, refinements in neonatal surgical technique, preoperative support, anesthesia, and neonatal intensive care have improved the outcome (Clark, 1999). Treatment is aimed at preventing aspiration pneumonia until surgical repair of the defect is completed. Healthy infants without pulmonary complications or other major anomalies usually can undergo surgery in the first few days of life. The type of surgical correction depends on the esophageal abnormality. A one-stage repair to connect both ends of the esophagus and close the fistula is preferred in all infants with TEF.

Occasionally, the infant’s condition (preterm, low birth weight, pneumonia, other major anomalies) requires that surgery be performed in stages. The first is closing of the fistula and inserting a gastrostomy tube for feeding. The second stage involves anastomosis (surgical connection of two tubular structures) of the two ends of the esophagus. Eight to ten days after this procedure, oral feedings are begun and usually tolerated.
Nursing Management
The goals preoperatively are prevention of aspiration of secretions from the upper esophageal pouch and prevention of regurgitation of stomach contents through the fistula into the trachea. Nursing care initially includes maintaining hydration status by allowing nothing by mouth and administering intravenous fluids. The infant is positioned with the head elevated to decrease pressure against the thoracic cavity and minimize reflux of gastric secretions into the trachea and bronchi. The patency of intermittent or continuous suction of the esophageal segment, if ordered before surgery, is essential.

In the postoperative period the nurse’s goals are to maintain a patent airway and prevent trauma to the anastomosis. Suctioning must be performed gently to avoid trauma to the tissues to maintain the airway. The nurse observes the infant for early signs of airway obstruction, such as an anxious expression on the infant’s face, tachypnea (increase in respiratory rate), and the presence of abnormal breath sounds. In the immediate postoperative period the gastrostomy tube is elevated to allow gastric secretions to flow into the small intestine and air to escape. A pacifier is offered to meet the infant’s sucking needs and to prepare for oral feeding. The infant remains fluid restricted (NPO) until bowel sounds return and there is no danger of disturbing the surgical site. Nutrients are obtained through intravenous fluids. When the infant is begun on gastrostomy feedings, glucose water is given, and if tolerated, followed by formula or breast milk.

Family Teaching
Infants who have the single-stage repair need to be observed for signs of esophageal stricture. The nurse explains and provides a written list of these and instructs the caregivers to contact their health care provider if any occur. Signs include dysphagia, inability or difficulty swallowing, increased drooling, and frequent coughing and choking that appear to be related to swallowing. The family of infants who require multiple stage surgery need to learn how to perform the necessary procedures for gastrostomy feeding and care and oral feeding.

LOWER GASTROINTESTINAL ALTERATIONS
Lower gastrointestinal alterations in infants and children include obstructive disorders in which nutrients and secretions are unable to pass through the GI tract, and elimination disorders. The alterations that will be presented include intussusception, Hirschsprung’s disease, and anorectal malformations.

Intussusception
Intussusception is a common pediatric condition that occurs when one segment of the bowel telescopes into the lumen of an adjacent segment of intestine.

Incidence and Etiology
Intussusception is the most frequent cause of intestinal obstruction in infants and young children. The incidence is 1–4 in 1,000 live births with the peak in the third to ninth month of life and occurring two times more frequently in boys compared with girls (Birkhahn, Fiorini, & Gaeta, 1999). In most cases the cause cannot be identified. In a minority of cases, a specific lesion, such as a polyp or foreign body, or a viral infection can be identified as a possible trigger. Figure 23-8 illustrates intussusception.

Pathophysiology
As one segment of the bowel telescopes or invaginates into another, the walls of the bowel press against each other and compromise the blood and lymph flow. The involved intestine becomes inflamed and edematous and bleeding occurs resulting in blood and mucus in the stool. Eventually, complete bowel obstruction develops producing abdominal distention and vomiting. If untreated, it may progress to necrosis and perforation.

Clinical Manifestations
Four signs and symptoms are classically described in the infant with intussusception: colic, intermittent abdominal pain, vomiting, and currant jelly-like stools. However, these are present in fewer than one-half of infants with the disease.
(Kuppermann, O’Dea, Pineckney, & Hoecker, 2000). Characteristically, a previously healthy, thriving infant has a sudden onset of severe and intermittent abdominal pain. Non-bilious vomiting is the predominant sign in neonates and is usually seen early in the illness. In the later phase the vomitus becomes bile stained. Blood and mucus appear in the stool, resulting in the red, currant jelly-like appearance. The abdomen is tender, and a sausage shaped mass may be felt in the right upper quadrant. As the intussusception progresses, the infant becomes listless and lethargic. Eventually a shock-like state may develop with a weak and thready pulse, shallow respirations, and a marked elevation of body temperature.

**Diagnosis**

An X ray of the abdomen is non-specific in the diagnosis; however, it will reveal intraperitoneal air if present, which indicates bowel perforation. The definitive test for diagnosing intussusception has been the barium or air contrast enema. Contrast enema is a safe procedure with minimal risk of bowel perforation. Nonetheless, it is invasive and presents the potential risk of radiation exposure. In addition, this test may be unnecessary if a less invasive technique can be used to accurately rule out intussusception. In recent years, abdominal ultrasound has been found to be a reliable and noninvasive screening tool for this disease (Harrington, et al., 1998).

**Treatment**

The treatment of choice for intussusception is non-surgical hydrostatic reduction using barium, a water-soluble contrast agent, or air enema. The water-soluble contrast and air insufflation (blowing air into a cavity) are believed to be safer than barium, with less risk of bowel perforation. Successful reduction rates have been reported as high as 90% for air and 65–85% for barium or the water-soluble contrast agent (Birkhahn, et al., 1999). If there is evidence of intestinal perforation, peritonitis, or shock or if hydrostatic reduction using barium, a water-soluble contrast agent, or air enema. Contrast enema is a safe procedure with minimal risk of bowel perforation. Nonetheless, it is invasive and presents the potential risk of radiation exposure. In addition, this test may be unnecessary if a less invasive technique can be used to accurately rule out intussusception. In recent years, abdominal ultrasound has been found to be a reliable and noninvasive screening tool for this disease (Harrington, et al., 1998).

**Nursing Management**

Because the onset of this disorder is so abrupt, most caregivers need much reassurance regarding the effectiveness of treatment and excellent prognosis. Preparation for hydrostatic reduction involves placing the infant on NPO status, inserting a nasogastric tube that is connected to low suction, and administering intravenous fluids. The nurse monitors the infant’s vital signs for changes that might indicate perforation, peritonitis, or shock, assesses for worsening abdominal pain, and examines and records all stools. The passage of normal stool may indicate spontaneous resolution of the obstruction. For a few hours after reduction, the child should remain in the hospital and be observed for the passage of stool and barium or water-soluble contrast, as indicated, and for the recurrence of the intussusception. Recurrence develops in about 10% of children following hydrostatic reduction. If hydrostatic reduction is unsuccessful, surgical treatment is indicated. For the child undergoing surgery, postoperative care is similar to that described in the nursing care plan for a child having an appendectomy.

**Family Teaching**

When the child is discharged, the nurse should instruct the caregivers to observe for signs of intestinal obstruction and recurrence. These include increasing abdominal pain, abdominal distention, blood in the stools, bile stained vomiting, and decreased or absent stools, all of which should be reported to their health care provider.

**Hirschsprung’s Disease**

Hirschsprung’s disease (HD), also called congenital aganglionic megacolon, is a motility disorder of the bowel caused by the absence of parasympathetic ganglion cells in the large intestine. This absence prevents peristalsis and causes feces to accumulate proximal to the defect, leading to bowel obstruction. It is the most common cause of distal bowel obstruction in the newborn; however, it may not be diagnosed until infancy or childhood.

**Incidence and Etiology**

The incidence is 0.2 in 1,000 live births, with males affected three to four times more often than females; the racial distribution is equal. HD is not a hereditary condition, but an inherited predisposition is relatively strong. A family history can be obtained in about 7% of cases, and the incidence in siblings is about 3.5% (Rudolph & Benaroch, 1995). Other associated congenital anomalies include imperforate anus, urinary tract abnormalities, cardiac defects, seizure disorders, and Down syndrome (Quinn & Shannon, 1996).

**Pathophysiology**

The disease is caused by an absence of parasympathetic ganglion cells in the colon. The aganglionic segment is most
frequently located in the rectosigmoid area. Defecation is controlled by the parasympathetic nervous system (the ganglion cells), to which the lower colon, the internal and external anal sphincters, and the anus respond in a coordinated manner. The affected bowel (absence of ganglion cells) is unable to transmit coordinated peristaltic waves and to pass fecal contents along its length, resulting in an accumulation of fecal material and distention proximal to the defect. The normal portion of the bowel becomes hypertrophied and dilated, hence, the name megacolon (Allen, 1995). Figure 23-9 illustrates the bowel in HD.

**Clinical Manifestations**

In the newborn the primary manifestations are failure to pass meconium (the first feces of the newborn) within 24 to 48 hours after birth, abdominal distention, bile stained vomitus, refusal to feed, and intestinal obstruction. In older infants and children, the initial symptom is chronic constipation. Abdominal distention, episodes of explosive passage of stools, inadequate weight gain, ribbon-like or pellet shaped, foul-smelling stools, vomiting, and an easily palpable fecal mass are also present.

The most ominous presentation is enterocolitis, inflammation of the small intestine and colon. An otherwise well infant who has a history of constipation has an abrupt onset of foul-smelling diarrhea, abdominal distention, and fever. The illness may progress rapidly, with perforation of the bowel and sepsis, and may occur before, during, or after surgery. Enterocolitis and sepsis remain the major causes of death in HD, occurring in about 30% of cases (Rudolph & Benaroch, 1995).

**Diagnosis**

Hirschsprung’s disease is diagnosed in 15% of infants within the first month of life, in 60% by the third month, and in 80% by 1 year of age (Rudolph & Benaroch, 1995). It can present with symptoms varying from complete intestinal obstruction with enterocolitis to simple constipation. In the neonate who does not pass meconium and has abdominal distention the diagnosis of HD is suspected. In older infants and children a history of chronic constipation should raise the question of HD. A rectal examination reveals the absence of stool in the rectum, and the internal anal sphincter is tight. A barium enema documents a transition zone between the narrowed aganglionic segment of the colon and the dilated, hypertrophied section. This sign may be absent in the first few weeks of life because it takes some time for normal ganglionic bowel to dilate with stool; therefore, the barium enema may not be diagnostic in newborns. For a definitive diagnosis, a rectal biopsy is required. The absence of ganglionic cells in the tissue confirms the diagnosis.

**Treatment**

The surgical treatment is usually performed as a two-stage procedure. In the first stage a temporary colostomy is created in the normal bowel. The purpose of the colostomy is to provide a means for the infant to defecate, to allow the bowel to rest and the infant to gain weight (Figure 23-10).
The second stage involves a pull-through procedure in which the affected, aganglionic segment is resected or removed and normal bowel is anastomosed to the rectum. The temporary colostomy is also closed at this time. This definitive surgical repair is performed when the infant is between 6 and 15 months of age or weighs between 18 and 20 pounds (George, Hammes, & Schwartz, 1995).

In recent years, the treatment has been changing from a two-stage surgical repair to a one-stage pull-through without a temporary colostomy with excellent results. The timing of the definitive procedure also has been changing from approximately 12 months to early neonatal surgery. Benefits of the one-stage correction include avoidance of multiple operations, reduction in the number of hospital admissions and the cost of treatment, elimination of the problems in colostomy care, and completion of treatment at an earlier age (Ramesh, Ramanujam, Yik, & Goh, 1999).

Another advancement in the treatment is a procedure called the laparoscopic-assisted pull-through. The laparoscope allows surgeons to enter the child's body through the anus and pull the affected segment of bowel through the opening, thereby eliminating major abdominal surgery. Because the laparoscope only requires a few small incisions, the length of hospital stay is decreased, the scar is minimal, and complications are fewer (Ramesh, et al., 1999).

Figure 23-10 Child With Colostomy After the First Stage of Surgical Repair of Hirschsprung’s Disease

The second stage involves a pull-through procedure in which the affected, aganglionic segment is resected or removed and normal bowel is anastomosed to the rectum. The temporary colostomy is also closed at this time. This definitive surgical repair is performed when the infant is between 6 and 15 months of age or weighs between 18 and 20 pounds (George, Hammes, & Schwartz, 1995).

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Nursing Management
Preoperative assessment of the infant’s fluid and electrolyte status is essential because preparation for surgery involves extensive bowel cleansing with repeated saline enemas. The infant is NPO, and an NG tube is inserted. Intravenous fluids and electrolytes are administered to prevent dehydration and correct electrolyte deficiencies if they occur. Oral administration of antibiotics may be ordered in conjunction with antibiotic enemas to reduce intestinal flora.

Postoperative nursing care includes routine post-abdominal surgery interventions, such as maintaining patency of the NG tube, monitoring for abdominal distention, and assessing for return of bowel sounds. The nurse measures and records the amount of colostomy and NG drainage. An enterostomal therapist (ET) fits the ostomy with an appropriate appliance and begins education with the family about colostomy care.

Family Teaching
The nurse explains to the caregivers about the need for the surgery and the temporary colostomy and how to care for it. Instructions include skin care, appliance application, and information about community resources for obtaining supplies. Referral to an ET is important for assistance if problems occur with the appliance or stoma. The nurse teaches the family signs and symptoms of complications such as enterocolitis and leaks or strictures at the site of the anastomosis. Signs of leaks are abdominal distention and irritability, and signs of strictures are constipation, vomiting or diarrhea. These indications of complications should be reported to their health care provider. Families will usually require encouragement, understanding, and support, especially with the idea of the colostomy. They should be informed that with proper management their child can return to a normal lifestyle in a short time.

Anorectal Malformations
Anorectal malformations are defined as an arrest of rectal descent resulting in absence of an anal opening and occur during the 4th–16th week of gestation. Examples of these malformations include anorectal agenesis (imperforate anus), rectal atresia, and anal agenesis.

Incidence and Etiology
Anorectal malformations occur in 0.2 in 1,000 live births and are more common in males. The etiology is unknown; however, these defects are associated with several other congenital anomalies of the urinary tract, esophagus, and intestines.

Pathophysiology
The origin of the anus and rectum is an embryonic structure called the cloaca, which is the precursor of the anorectal and
genitourinary structures. Both the rectum and the urinary structures become completely separated by the 7th week of gestation. Any abnormality in the development of these systems results in anorectal and genitourinary malformations. Depending on the week of gestation when the embryonic development is disrupted and on the level to which the rectal pouch has descended, the anomaly will be either low or high. Low malformations occur between the 10th and 12th week; high ones occur during the 4th week. In low anomalies the rectal pouch has descended below the rectal sphincter muscle complex. For rectal continence to occur the rectum must descend to this point. In high defects the rectum terminates above the sphincter muscle complex, making continence more difficult to establish (Quinn & Shannon, 1996).

**Clinical Manifestations**

Anorectal malformations are usually obvious at birth. Low defects vary from a normal appearing anus, to a thin translucent anal membrane, to a deep anal dimple. The anal dimple will demonstrate strong muscular contractions when pricked with a pin. High defects present as a flat perineum, absence of an anal dimple, and no muscular contraction to a pin prick. If meconium is noted in the urine, a fistula is present between the bowel and the urinary tract.

**Diagnosis**

Diagnosis is made by physical exam of the anal features and by radiologic imaging of the abdomen. The level of the defect and presence and location of any fistulas are determined by these tests. During the abdominal X rays the neonate is held in an inverted position for a few minutes to allow air to fill the blind colonic pouch and permit identification of the level of the defect. The presence of gas in the bladder or urethra during imaging indicates a fistula between these structures and the bowel.

**Treatment**

Treatment depends on the extent of the malformation. Anal stenosis is managed with repeated manual dilatation of the anus. All other defects require surgery. Low defects are corrected by creating an anal opening, followed by anal dilation to prevent stenosis. High malformations are treated with a two-stage repair, the first involving creation of a temporary colostomy. The second stage includes closure of the colostomy and a pull-through procedure in which the blind pouch of the rectum is anastomosed to the anus (Quinn & Shannon, 1996).

**Nursing Management**

During the newborn examination, the nurse assesses the patency of the anus by inserting a rectal thermometer. The defect is obvious when there is an absence of a normal anal opening. Nursing observations that should be documented and reported include failure of the neonate to pass meconium within the first 24 hours of life, inability to insert a rectal thermometer, and the presence of an anal dimple. Nursing care depends on the type of lesion corrected. For low defects with the anoplasty, the main focus is on preventing infection of the perineal and anal wounds. Because of the location of the surgical incisions, they are at high risk for infection from urine and stool; therefore, meticulous skin care is essential. For high lesions, postoperative care initially includes colostomy care, perineal wound care, IV fluid management, and NG tube maintenance. Oral feedings are begun when stooling has started through the stoma. Oral feedings following the pull-through are begun when peristalsis resumes, stooling occurs through the anus, and initial healing has taken place.

**Family Teaching**

Caregivers are instructed in colostomy care, perineal wound care, and anal dilations as appropriate. Prevention of constipation is stressed in family education. Adequate fluid, dietary fiber, and stool softeners or bulk agents help the child to achieve normal bowel activity. It is important to advise caregivers that toilet training may be delayed and children may have difficulty with this developmental task. Their patience and understanding of their child is essential. Encouraging the family and child during this stressful time is a key nursing intervention.

**ALTERATIONS IN MOTILITY**

Disorders discussed in this section include gastroesophageal reflux and constipation.

**Gastroesophageal Reflux**

Gastroesophageal reflux (GER) is the most common esophageal disorder of infants and the most frequently referred condition to a pediatric gastroenterologist (Orenstein, Izadnia, & Khan, 1999). GER is defined as the return of gastric contents into the lower esophagus through the lower esophageal sphincter (LES). The LES is a distinct area formed by the union of the muscle fibers from the esophagus and stomach (Figure 23-11).

Physiological GER is a common occurrence in many healthy infants. Improvement is usually seen between 6 and 12 months of age as the infant matures. The esophagus elongates and the LES moves down below the diaphragm decreasing the chance of reflux. Pathologic GER is reflux that manifests as respiratory disorders, esophagitis or its complication (strictures), and malnutrition.

**Incidence and Etiology**

GER is known to occur in 5 in 1,000 live births. Boys are affected 3 times more frequently than girls. GER is more
common in premature infants and those with neurologic impairment. The exact cause is unknown although it is thought to result from the delayed maturation of the lower esophageal neuromuscular function or impaired local hormonal control mechanisms.

Pathophysiology
The LES acts as a physiological barrier to reflux of stomach contents into the esophagus. It is innervated by the vagal nerves, so a defect in this nerve transmission may result in inappropriate relaxation of the LES. This allows the reflux of gastric contents into the esophagus. Delayed gastric emptying also occurs in infants caused by hypomotility or retrograde peristalsis of the duodenum (Ault & Schmidt, 1998).

Clinical Manifestations
Vomiting and regurgitation are the most common symptoms. The regurgitated matter is typically non-bilious and consists of undigested formula and mucus. The infant displays excessive crying and irritability from esophagitis, which is caused by recurrent reflux of acidic gastric contents into the esophagus. Refusal to feed may develop in infants with esophagitis as they learn to associate feeding with pain. Esophagitis can cause bleeding in the gastrointestinal tract, which produces anemia and is seen as blood in the stools. Insufficient caloric intake resulting from caregivers’ hesitancy to feed infants who are repeatedly spitting up and nutrient losses in emesis contribute to malnourishment. Complications of GER include apnea, choking spells, recurrent aspiration pneumonia, and frequent respiratory infections.

Critical Thinking
What’s the Difference Between Gastroesophageal Reflux and Hypertrophic Pyloric Stenosis?
Vomiting is a classic symptom of GER and HPS. Based on the clinical manifestations of each disorder, how would you distinguish between the two?

Diagnosis
The diagnosis of GER is established by taking a history, performing a physical examination, observing the infant’s feeding habits, and conducting several diagnostic tests. The first goal is to rule out other possible causes for the symptoms such as gastrointestinal tract obstructions, neurologic disease, or metabolic disorders. Several diagnostic tests exist to help confirm GER. Initially an upper GI barium series is performed to eliminate anatomic abnormalities (esophageal stricture, pyloric stenosis, and intestinal malrotation) from consideration. Upper GI endoscopy allows direct visualization of the esophageal mucosa to detect inflammation and ulceration, which are indicative of esophagitis. Another test is the esophageal pH probe study. A small catheter is placed through a nostril into the distal third of the esophagus and is left in place for 18 to 24 hours. It measures the pH of the distal esophagus, indicating the...
number of reflux episodes and the time it takes for acid to clear from the esophagus. Although a sensitive test, the pH probe is not needed for routine diagnosis. Because of the cost and the need for hospitalization, this test is best used for infants who have complicated courses or who present diagnostic dilemmas.

**Treatment**

Medical management involves dietary modifications, positioning, and medications. Small, frequent feedings are recommended because of the probable relationship between gastric volume and reflux. This feeding method decreases the duration of reflux episodes. However, increased frequency causes more frequent stimulation of stomach acids. Therefore, the advantages must be weighed against the disadvantages. Many infants have benefited from this traditional approach. Another dietary adjustment that is recommended is the thickening of formula with cereal. This approach has major benefits for the regurgitating infant, particularly when there has been poor weight gain. These include increased caloric density, decreased time spent crying, and decreased episodes of emesis.

Controversy surrounds positioning therapy in the medical treatment for GER. Recommendations for positioning have changed from the upright or seated position prior to the early 1980s, to the 30 degrees prone or head elevated prone in the middle 1980s, to the flat prone in the early 1990s (Orenstein, 1990). One aspect of the controversy involves positioning to prevent sudden infant death syndrome (SIDS). The American Academy of Pediatrics (1996) recommends that infants be placed in the supine (on their back) position for sleep. Most of the SIDS deaths in the prone position are attributable to suffocation because of puffy bedding materials, such as comforters. If these materials are eliminated from the infant's bed, the superiority of the prone position for GER is clear and has been recommended by the American Academy of Pediatrics (Orenstein et al., 1999). Currently either the flat prone or head elevated prone position is suggested for infants with GER.

Medications may also be used to treat GER. Antacids act to buffer existing gastric acids that may irritate the esophageal mucosa. Adverse effects of antacids include diarrhea (with magnesium based products, Mylanta) and constipation (with aluminum based products, Amphogel). Prokinetic drugs are often used before acid suppression therapy in infants who have no evidence of esophagitis. Prokinetic agents increase gastric motility and LES pressure, and enhance gastric emptying. Cisapride (Propulsid) has become the first choice prokinetic for infants because of its minimal side effects such as transient diarrhea and increased psychomotor activity. Metoclopramide (Reglan), also a prokinetic, has the same action as cisapride; however, its adverse side effects are common and include restlessness, insomnia, and extrapyramidal movements. Acid suppression medications are added if esophagitis is suspected or demonstrated. This action is achieved with histamine-2 receptor antagonists such as cimetidine (Tagamet) or ranitidine (Zantac). There are few adverse effects with these medications, primarily diarrhea or constipation, headache, and rash. Some infants likely to require the more complete acid suppression achievable with proton-pump inhibitors are those with chronic respiratory disease (cystic fibrosis, steroid-dependent asthma), or neurologic disabilities (cerebral palsy). Omeprazole (Prilosec) has minimal side effects that are similar to those of H-2 receptor antagonists.

The role of surgical treatment has decreased as pharmacotherapy has improved. Repeated episodes of pneumonia, failure to gain weight, recurrent esophagitis with stricture, severe apnea, and failure to respond to 4 to 6 weeks of medical management are indications for surgery. The Nissen fundoplication in which the fundus of the stomach is wrapped around the lower part of the esophagus is the procedure of choice. A temporary gastrostomy tube may be inserted to allow for venting of the stomach and initial feedings. The success rate with surgery is high, but recurrences are common. Less invasive fundoplication performed laparoscopically is available in a limited number of hospitals.

**Nursing Management**

**Assessment**

An in-depth assessment of the infant’s feeding pattern should include the amount, type, and frequency of feedings, and the timing of emesis afterwards. The nurse inquires about the positioning of the infant during feeding and the frequency of burping. It is important to obtain height, weight, and head circumference measurements and to plot them on a growth chart to assess current and potential growth problems. Infants with GER are at high risk for aspiration; therefore, assessment of a baseline respiratory status is imperative, such as lung sounds, respiratory rate, and effort.

**Nursing Diagnoses**

1. Risk for aspiration related to vomiting and reflux of gastric contents into the esophagus
2. Imbalanced nutrition: Less than body requirements related to reduced nutrient intake and vomiting
3. Deficient knowledge related to infant's condition and care including feeding, positioning, and home management

**Outcome Identification**

1. Infant maintains normal respiratory status (respiratory rate appropriate for age, oxygen saturation within normal limits, clear, bilateral breath sounds).
2. Infant will maintain normal growth pattern and will ingest adequate number of calories.
3. Caregivers will verbalize and/or demonstrate understanding of GER, feeding and positioning of infant, and home care.

Planning/Implementation
Nursing management focuses on caregiver education including dietary modifications, positioning, medication administration, and developmental needs of the infant, and on perioperative care if surgery is performed. Dietary modifications include small, frequent feedings, thickened feedings, and avoidance of foods that irritate the GI tract. Small, frequent feedings can cause additional stress on caregivers; therefore, they need to know that higher volume and less frequent feedings can be tolerated as the infant grows. The infant should be burped frequently during feeding. Thickening of formula with rice cereal increases consistency and retention, and supplies needed calories for the infant who vomits frequently. If the health care provider has recommended the head elevated prone position after feedings and during sleep, maintaining this can be challenging for caregivers. This position can be achieved by using a wedge, sling, harness, and towel rolls, some of which are commercially available.

Nursing interventions related to the treatment of GER with pharmacotherapy include caregiver education about dosages, proper administration and scheduling, and potential side effects. Verbal information should be augmented by written material to assure proper understanding. The nurse can support the caregivers by encouraging them to identify and verbalize their fears and concerns. They often feel guilt and inadequacy because of the frequent vomiting of feedings and weight loss of the infant. They may feel overwhelmed with doubt and anxiety about their ability to adequately care for the child. Nurses can be instrumental in forming a network of caregivers of children with GER to provide support, share experiences, and foster confidence.

Evaluation
The effectiveness of the nursing interventions is evaluated by the infant experiencing no respiratory difficulty or aspiration. A decrease in the frequency of vomiting and improvement in growth and development are also evaluated. Most important is the caregivers’ comfort with the diagnosis, treatment plan, and their confidence in being able to care for the infant at home.

Family Teaching
Family teaching includes an explanation of the physiology of GER so caregivers understand their feeding technique is not the cause of the frequent vomiting. In order to accommodate the thickened feedings, the nurse should demonstrate how to enlarge the hole in the nipple. Caregivers need information about spicy and acidic foods and beverages to avoid feeding their infant because they increase secretion of gastric acid. These include citrus fruits and fruit drinks and tomato products such as tomato juice. Esophageal irritants such as chocolate and caffeine (tea, coffee, and colas) for older infants and children should also be avoided. Because of the infant’s limited mobility, adequate stimulation becomes essential. Bright and colorful objects, wrist rattles, mobiles, and mirrors are all appropriate. Touching and stroking the infant provide tactile stimulation. Family teaching should also include an explanation about avoiding vigorous playing with their infant after feeding to prevent reflux.

Constipation
Constipation is the difficult passage of stool or infrequent passage of hard stool, associated with straining, abdominal pain, or withholding behaviors. Children vary widely in the frequency with which they have a bowel movement; therefore, frequency alone is not a good diagnostic criterion.

Incidence and Etiology
Constipation is common in children, accounting for 3% of office visits to pediatricians and 25% of pediatric gastroenterologist’s visits (Van der Plas, et al., 1996). It is more common in males during early childhood; however, during adolescence it is seen more frequently in females. The cause of constipation can be organic or non-organic, also called functional. Organic causes include:

1. Dietary (e.g., low fiber, inadequate fluid intake, excessive dairy intake)
2. Structural disorders of the gastrointestinal tract (e.g., Hirschsprung’s disease, intestinal strictures)
3. Metabolic and endocrine disorders (e.g., hypothyroidism, diabetes mellitus, lead poisoning)
4. Neurogenic diseases (e.g., cerebral palsy, myelomeningocele)
5. Medications (e.g., opiates, antidepressants, anticholinergics, antacids)

For the majority of children, non-organic or functional problems are the cause.

Infant Seats for Positioning
Infants with GER should not be placed in an infant seat as a mode of treatment. The reduced truncal tone in infants raises their intra-abdominal pressure and actually promotes reflux.
Additionally, a previously painful bowel movement because of a hard stool or anal fissures can result in fear of having a bowel movement. Magical thinking is a characteristic of toddlers’ cognitive development, which can result in their fear of the toilet and cause difficulty in having a bowel movement. For example, a two-year-old boy who had developed a fear of having a bowel movement, admitted he was worried that his “poo would drown.” Another child wanted to know “do poos have brains?” Fear of the toilet may be initiated by television. Commercials for toilet cleaners, for example, contain images of “germs and monsters” climbing out of the toilet. This can seem very real in a young child’s imagination.

The older toddler, preschooler, and school-aged child may develop problems when starting nursery school, kindergarten, or 1st grade. Bathrooms in these settings may lack privacy and tend not to have soft toilet paper. As a result, the urge to defecate is suppressed during school hours. Continual suppression of defecation can lead to constipation.

**Pathophysiology**

Normal defecation occurs when stool moves into the rectum, causing rectal distention and relaxation of the internal anal sphincter. The conscious awareness of rectal distention results in contraction of the voluntary muscles of the external anal sphincter. Voluntary relaxation of the external sphincter and increased intra-abdominal pressure result in defecation. Constipation tends to be self-perpetuating. As stool is retained, the simultaneous process of stretching the rectal wall and decreasing sensory feedback leads to less frequent bowel movements, which result in further stool retention and larger stools. As water is reabsorbed, the stool becomes harder, and bowel movements may become painful. As this cycle progresses, the external and internal sphincters become compromised. Sensitivity to rectal distention and control of rectal evacuation diminish, and the child soon loses the urge to have a bowel movement.

**Clinical Manifestations**

The child who is constipated will have hard, small stools that may be passed at regular intervals or large masses of stool at intervals of days to weeks. Soiling of underwear in a child who is toilet trained is possible. Abdominal pain and/or distention develop as more stool accumulates in the bowel. The child may become irritable and experience a loss of appetite. Often a palpable fecal mass is felt on physical exam.

**Diagnosis**

Diagnosis is based on the history and physical exam. When attempting to determine the cause, it is important to rule out any organic causes. A thorough dietary history is obtained. A description of stool pattern, such as frequency, consistency, and size of stools, and toilet training history is elicited. Certain medications can cause constipation; therefore, it is important to determine if the child is taking any of these. An abdominal X-ray shows a colon enlarged with stool and gas.

**Treatment**

Constipation is treated with a combination of therapies, which include cleansing the bowel, establishing a regular pattern of defecation, and modifying the diet. Cleaning the bowel of hardened or impacted stool is accomplished with enemas, oral medications, and suppositories. There are many types of oral medications that can be used. The choice depends on the child’s ability to take the medication, the ease of giving it, and how well it works. Occasionally, if feces are impacted, they may need to be removed manually.

Modification of the diet includes increasing the intake of fiber and fluids. Establishing a regular pattern for defecation is largely a matter of caregiver education about normal defecation and bowel training techniques.

**Nursing Management and Family Teaching**

Nursing intervention focuses on education. Caregivers need instruction in the appropriate way to administer an enema (See the Pediatric Nursing Skills CD-ROM). Dietary modifications include increasing dietary fiber and fluids. The nurse should teach caregivers about high fiber foods and diet planning. High fiber foods include: whole grain breads and cereals, bran, high fiber snack bars, raw vegetables, fruits, especially raisins, prunes, cherries, and apricots, beans, popcorn, nuts, and seeds.

Establishing a regular pattern of defecation is accomplished by requiring the child to sit on the toilet after a meal for a reasonable amount of time, 5–10 minutes. Positive reinforcement with star charts or small prizes can be used to reward success and adherence with the toileting schedule and taking of medications.

**INFLAMMATORY DISORDERS**

Disorders caused by chronic inflammation of the GI tract can occur at any age, newborns through adolescents. Some of these disorders are short term and readily resolved like appendicitis; others are chronic and affect growth and development. Appendicitis, inflammatory bowel disease (ulcerative colitis and Crohn’s disease), peptic ulcers, and necrotizing enterocolitis will be discussed in this section.

**Appendicitis**

Appendicitis, the inflammation of the vermiform appendix or the small sac at the end of the cecum, is the most com-
mon condition requiring abdominal surgery in children. Although appendicitis was first described over 100 years ago, the vagueness of its signs and symptoms in children poses a continuing challenge for health care providers to arrive at a timely and accurate diagnosis. Failure to diagnose appendicitis is the most frequent subject of malpractice suits and the fifth most expensive source of claims for emergency department physicians (Pisarra, 1999).

Incidence and Etiology
Appendicitis is the most common condition requiring abdominal surgery in childhood, occurring at a rate of 4 per 1,000 children younger than 14 years of age. It is more common in summer, has a higher incidence in males than females, Caucasians than non-Caucasians. Although the exact cause is poorly understood, the appendix becomes inflamed usually because of obstruction between the appendix and cecum or a systemic or enteric infection. Appendicitis is rare in third-world countries where diets are high in fiber; however, no causal relationship has been established between dietary fiber and the prevention of appendicitis (Higgenbotham & Gottlieb, 1998).

Pathophysiology
The appendix is vermiform (wormlike) in shape, with a diameter similar to a lead pencil. It rises from the wall of the cecum portion of the large intestine, below the ileocecal valve. However, its location can vary among individuals. As food passes through the cecum, the appendix also fills and empties. In 70% of cases, the lumen between the appendix and the cecum becomes obstructed with agents such as a fecalith, fecal matter that becomes petrified and stone-like, calculi, tumors, parasites, and foreign bodies. In the remaining 30% with no evidence of obstruction, the inflammation may be caused by a bacteria, virus, trauma, or postoperative fecal stasis (Pisarra, 1999).

Clinical Manifestations
Abdominal pain is the first symptom in typical cases of appendicitis. Initially, the pain may be vague and poorly localized to the periumbilical area; gradually migrating to the right lower quadrant (RLQ). Anorexia and nausea with or without vomiting may also be present, but occur after the initial symptom of pain. The most reliable information gained from the history is the sequence of symptoms. Pain nearly always precedes anorexia, nausea, or vomiting. Nausea and vomiting that precedes abdominal pain often indicates gastroenteritis. Additional clinical manifestations that may be present are constipation or diarrhea. The child’s temperature is usually normal or slightly elevated. A temperature of 101°F or higher suggests the presence of peritonitis.

Diagnosis
Appendicitis remains a diagnosis made largely on the basis of the history and physical examination. Diagnosis is challenging in children because the clinical manifestations can present atypically. Children who are misdiagnosed have an increased incidence of perforation, abscess, wound infection, and even mortality. Abdominal tenderness on palpation is a common, important, and reliable symptom. Tension of the muscles (muscle rigidity) over the tender area may be felt. Rigidity over the entire abdomen, accompanied by tense positioning and guarding (involuntary contraction of abdominal muscles caused by fear of impending pain), indicates a perforated appendix with peritonitis. Rebound tenderness describes a sensation of severe pain that occurs after deep pressure is applied and released and is indicative of peritonitis. However, many practitioners consider the elicitation of rebound tenderness to be a crude and unnecessarily painful technique. The resultant severe pain may adversely affect the element of trust with the child. Palpation with a stethoscope is a preferred method for children in order to identify areas of tenderness.

Laboratory findings do not establish the diagnosis, but there is often a moderate elevation of the white blood cell (WBC) count, seldom higher than 15,000 to 20,000/mm³, with a “shift to the left” (an increased number of immature WBCs). However, some children with appendicitis have a

Eye On:

Empacho
Empacho is a Spanish word that means indigestion, stomach pains, and abdominal cramps. Some Hispanics believe these symptoms are caused by a ball of undigested food clinging to some part of the gastrointestinal tract and are due to being forced to eat against one’s will or lying about the amount of food eaten. Treatment is to massage and gently pinch the spine. This therapy can be problematic if the cause of symptoms is something serious such as appendicitis.
normal white blood cell count. Abdominal X rays may reveal a fecolith or some other cause of obstruction, although this rarely confirms the diagnosis. Other causes of acute abdominal pain must be ruled out, including severe constipation, urinary tract infection, acute gastroenteritis, pelvic inflammatory disease, and discomfort associated with ovulation. Abdominal ultrasound has been employed in an attempt to increase accuracy of appendicitis diagnoses. However, it may be more appropriate in verifying other causes of abdominal pain than in diagnosing appendicitis (Pisarra, 1999).

Delay in diagnosing appendicitis in children is a factor contributing to perforation rates of 30% to 60%. Young children have a thinner appendiceal wall, so progress from inflammation to perforation is more rapid than in adults. Children also have a poorly developed omentum, so local perforation is not usually confineable, and peritonitis develops. The close proximity of abdominal and pelvic organs further favors the spread of peritonitis to other structures. The inflammatory process associated with perforation may lead to intestinal obstruction or paralytic ileus. The signs and symptoms of peritonitis can be remembered by using the mnemonic PERITONITIS (Box 23-1).

**Treatment**

Once the diagnosis of appendicitis has been made, surgery is required as soon as possible. In an uncomplicated appendectomy, an incision approximately 2–3 inches is made in the right lower quadrant. If perforation is suspected, has

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**Nursing Tip:**

Assessment of peritoneal irritation

One way to check for peritoneal irritation in a toddler or preschooler is to have the child jump up and down with you. If the child does so several times without bending over in pain, you can confidently rule out peritoneal irritation.

**Nursing Tip:**

Palpating the abdomen for pain

Children frequently tense their abdominal muscles when they are being examined. Warm your hands and stethoscope before touching their abdomens. Maintain eye contact with them while palpating their abdomens, and they will watch your face rather than your hands and relax. This technique provides more reliable information about the presence of pain.

**Nursing Alert:**

Treatments to Avoid in Suspected Appendicitis

Caregivers should be told to avoid giving their child laxatives or enemas and applying heat to the abdomen if appendicitis is suspected. These measures may stimulate bowel motility and increase the danger of perforation. Additionally, pain medication should be avoided because it can mask the signs of intraperitoneal inflammation.

**Kids Want To Know**

What will the scar look like after my appendectomy?

Maria, a 14-year-old girl, has been diagnosed with appendicitis and is scheduled for an appendectomy tomorrow. She asks her nurse “What will the scar look like? How big will it be? Will I still be able to wear my bikini swimming suit?” Because Maria’s appendix has not perforated prior to surgery, the nurse can explain that the surgical incision will be small, about one-inch long, and to the right and slightly below the level of the umbilicus. She will be in the hospital for approximately three days if no complications occur and should be able to wear her bikini. She can further explain that if her appendix had ruptured and peritonitis had developed before surgery, the abdominal incision would be larger, and a drain would be placed in the abscess site. The incision may be closed only through the fascia with the skin left open and the wound packed to decrease the potential for infection or problems with healing.

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**BOX 23-1 A mnemonic for peritonitis**

P Pain: front, back, sides, shoulders
E Electrolytes fall, shock ensues
R Rigidity or rebound of abdominal wall
I Immobility
T Tenderness
O Obstruction
N Nausea and vomiting
I Increasing pulse, decreasing blood pressure
T Temperature falls, then rises
I Increasing girth of abdomen
S Silent abdomen (no bowel sounds) (Shipman, 1984)
occurred, or the appendix is in an atypical position, the incision is larger. An uncomplicated appendectomy may also be performed via laparoscopy. Preoperatively, the child is managed with an NPO status, intravenous fluids to replenish fluid volume and to correct electrolyte or acid-base imbalances, and pain medication. Postoperatively in an uncomplicated appendectomy, the child will receive antibiotics for 24 hours, remain NPO until intestinal peristalsis returns, and is discharged in 2–3 days. If perforation occurred, drains may protrude from the incision site or the incision may remain open and allowed to heal by secondary intention to prevent infection. Figure 23-12 illustrates an incision healing by secondary intention after an appendectomy for a perforated appendix. Additional treatment includes IV antibiotics for 7–10 days, NPO status, NG tube suction, IV fluids, and pain medication. The child remains NPO until bowel function returns and is discharged when wound drainage is minimal and oral intake satisfactory. If a laparoscopic procedure was

Figure 23-12 Incision Healing by Secondary Intention After Appendectomy of a Ruptured Appendix

Case Study/Care Plan

A Child with Appendicitis

Andrew is a 12-year-old boy who is being seen in the emergency department with a complaint of abdominal pain, vomiting, fever, and anorexia. Four days ago he awoke complaining of a dull stomach ache and not feeling well. He went to school but returned by noon and went back to bed. By early evening the pain was sharp and he could indicate the umbilical area as hurting. He refused to eat all day but did drink water and juices. The next morning he vomited yellow emesis, and the pain was periodically severe. His temperature was 100.5°F, and he vomited several more times that day. In the middle of the night he awoke with pain so severe he was crying and would not stand up straight. He was diagnosed with acute appendicitis and admitted to the hospital for surgery.

Nursing Care Plan

Assessment Because prompt and accurate diagnosis of appendicitis is important, nurses need to assess any child presenting with abdominal pain for the early signs and symptoms. Nurses in a variety of settings, such as emergency departments, school and school-based clinics, and ambulatory care clinics, are in an ideal position to recognize these early indicators of appendicitis. The nursing assessment should include detailed information about the child’s pain (onset, location, intensity), changes in behavior (refusing to attend school or play outside, loss of appetite), and vomiting. If the symptoms of appendicitis are present, the child should be referred for further evaluation.

Nursing Diagnosis #1

Acute pain related to inflamed appendix and postoperative surgical incision (refer to Chapter 18)

Expected Outcomes

1. Andrew states and/or exhibits reduced pain

Interventions/Rationales

1. Use a pain assessment scale appropriate for child’s developmental level to determine severity of pain. The pain scale provides objective data and the child’s input.

continues
2. Assess behavioral cues (crying, movement, facial grimace) that indicate discomfort or pain. *Behavior of child provides clues to pain experience.*

3. Use non-pharmacologic approaches such as distraction with toys and games or relaxing music. *Concentration on something other than pain directs the discomfort from conscious thought.*

4. Administer pain medication by continuous infusion or every 3–4 hours around the clock as prescribed. *Pain breakthrough occurs even during sleep; pain is continuous for 1–2 days postop.*

5. Assist child to assume position of comfort. *Fowler’s position with knees flexed avoids strain on the abdomen.*

**Evaluation**

Andrew will experience no pain or reduced pain as evidenced by relaxed body posture and facial expression; heart and respiratory rate and blood pressure within appropriate range for age; naps and sleeps appropriately for age undisturbed by pain; and ambulation without undue resistance.

**Nursing Diagnosis #2**

Risk for deficient fluid volume related to inadequate intake and losses secondary to vomiting, diarrhea, and NPO status

**Expected Outcomes**

1. Andrew will regain adequate hydration status.

**Interventions/Rationales**

1. Administer IV fluids as prescribed. *Inadequate oral intake, vomiting, and diarrhea deplete total body water.*

2. Replace NG output with additional IV fluids as prescribed. *NG suction removes gastric contents that contain water, hydrochloric acid, and potassium.*

3. Maintain accurate intake and output; estimate wound drainage and include as output. *Provides an ongoing assessment of hydration status.*

**Evaluation**

Andrew has adequate fluid volume as evidenced by appropriate intake of IV and oral fluids and urine output (minimum of 1 to 2 ml/kg/hr); moist mucous membranes, elastic skin turgor, and normal vital signs for age.

**Nursing Diagnosis #3**

Risk for infection related to possible or actual rupture of appendix and surgical incision

**Expected Outcomes**

1. Andrew will be free of infection.

**Interventions/Rationales**

1. Preoperative

   a. Assess child for clinical and laboratory manifestations of appendicitis. *Successful treatment is dependent on timely diagnosis of the disorder.*

   b. Assess for and immediately report symptoms of peritonitis (initial relief of pain at time of perforation followed by increased intensity, abdominal distention, guarding, decreased or absent bowel sounds, elevated temperature, and shock-like symptoms). *Perforation occurs in a high percentage of children with acute appendicitis, which greatly increases the risk of serious complications.*

   continues
Inflammatory Bowel Disease

Inflammatory bowel disease (IBD) refers to a group of chronic disorders that cause inflammation or ulceration in the small and large intestine and include ulcerative colitis (UC) and Crohn’s disease (CD). Ulcerative colitis involves inflammation of the mucosa and submucosa of the colon and rectum, while CD is an inflammation that may involve the entire gastrointestinal tract and all layers of the bowel wall (transmural) (Orloski, 1998).

Incidence and Etiology

Once considered rare in children and adolescents, IBD is now being recognized with increasing frequency in this age group. In fact, 20% of all individuals with UC and 25–30% of those with CD present before age 20. Peak onset is in late adolescence. With increasing recognition of IBD, it has become one of the most significant chronic diseases affecting children and adolescents (Baldassano & Piccoli, 1999).

The incidence of IBD is similar in males and females, is higher among the Caucasian population of developed Western countries, and lower among African-Americans and Asians. The incidence of UC is 0.05 per 1,000; the incidence of CD is 0.04 per 1,000. The etiology of IBD is unclear; however, infectious agents, autoimmune, genetic, and environmental factors have been implicated. Current thinking suggests that a triggering factor, possibly a virus or an atypical bacterium, interacts with the body’s immune system to induce an inflammatory reaction in the intestinal wall. About 15% to 20% of individuals with IBD have a close relative with one of these diseases, suggesting a genetic factor (Baldassano & Piccoli, 1999).

Pathophysiology

The bowel responds to an environmental trigger that the immune system identifies as dangerous and causes an injury resulting in vasoconstriction. This is followed by localized release of cellular mediators, including histamine, which produce a marked vasodilation. Capillaries become distended with blood and begin to contract, causing ruptures in the walls. The swollen engorged bowel is fragile and is, therefore, inclined to ulcerate, causing a break in the mucosal barrier. Digestive enzymes and intestinal bacteria act on this exposed tissue, causing further irritation, inflammation, ulceration, and bleeding. Ulcers can become fissures as they penetrate more deeply into the intestinal wall. Fistulas can occur into the bladder and/or vagina (more common in CD). Infectious exudate consisting of plasma proteins draws more fluid into the bowel resulting in diarrhea that may be bloody. Healing lesions result in scar tissue formation and subsequent scarring of bowel may lead to strictures and bowel obstruction.

Clinical Manifestations

The most common symptoms of UC are rectal bleeding, diarrhea, and abdominal pain. Multiple patterns of presentation occur in children and adolescents. Mild disease is seen in 50–60% of cases. The onset of diarrhea is insidious, and there are no extra-intestinal or systemic signs of fever, weight loss, or hypoalbuminemia. Thirty percent present with moderate disease characterized by bloody diarrhea, abdominal cramping and tenderness, and the urgency to defecate. These individuals have associated systemic signs such as anorexia, weight loss, low-grade fever, and mild anemia. Severe cases occur in 10% of clients. Clinical manifestations in these cases are more than six bloody stools per day, abdominal tenderness, fever, anemia, leukocytosis, and...
hypoalbuminemia. Occasionally, children with UC may have predominately extra-intestinal manifestations such as growth failure, arthritis, and skin lesions (Baldassano & Piccoli, 1999).

In contrast to UC, CD may occur in any segment of the gastrointestinal tract. The clinical manifestations are determined primarily by the location and extent of disease involvement. The majority of children (50–70%) have disease involving the terminal ileum. In these children symptoms of malabsorption predominate, including diarrhea, abdominal pain, anorexia, weight loss, and growth failure. CD that occurs in the colon may be indistinguishable from UC, with symptoms of bloody diarrhea, crampy abdominal pain, and urgency to defecate. Perianal involvement includes painful defecation, bright red rectal bleeding, skin tags, hemorrhoids, fistulas, and abscesses.

Growth failure occurs more frequently in children with CD than with UC. Children with either disorder tend to reduce dietary intake below that recommended for age to diminish symptoms induced by eating, which results in growth failure, characterized by an abnormally slow growth velocity. A major consequence of prolonged reduction in growth velocity is permanent short stature, frequently seen in adults who had CD during childhood. Additionally, delayed sexual development is frequently seen. More importantly, growth failure and delayed puberty are often debilitating symptoms for adolescents, potentially affecting their self-esteem, social interactions, and school performance (Ruemmele, Roy, Levy, & Seidman, 2000). Systemic or extra-intestinal symptoms are more common in children with CD than UC. Some of these symptoms tend to be more severe in children than in adults. For example, children have higher fevers, more joint pain, nausea, and in general feel sicker than do adults with CD. See Table 23-1 for a comparison of UC and CD.

**Diagnosis**

Presenting symptoms and clinical course of UC and CD are similar enough that they often elude a differential diagnosis sometimes for years. Diagnosis is based on history and physical exam and endoscopic or radiologic examination of the colon to evaluate the character and location of lesions. Endoscopy includes either a sigmoidoscopy or a colonoscopy and biopsies of the mucosa are obtained and examined. Radiologic studies include a barium enema and an upper gastrointestinal contrast examination. Laboratory studies may indicate anemia, hypoproteinemia, fluid and electrolyte imbalances, and an elevated sedimentation rate.

**Treatment**

Treatment for inflammatory bowel disease involves pharmacologic, nutritional, and surgical approaches. The goals include controlling the disease, inducing remission and preventing relapses, providing adequate nutrition for growth and development, and assisting the child to function as normally as possible (e.g., school attendance, participation in sports). Pharmacotherapy is aimed at either decreasing inflammation or directly suppressing the immune system. Categories of medications include corticosteroids, aminosalicylates, antibiotics, and immunosuppressants. Corticosteroids are used during acute episodes for treating moderate to severe IBD. The aminosalicylate azulfidine (sulfasalazine) acts directly on the bowel mucosa to reduce inflammation. Metronidazole (Flagyl), an anti-infective, has been helpful in the treatment of perianal complications in CD. Immunosuppressive medications such as cyclosporine have been useful in children with corticosteroid-resistant CD.

The goal of nutritional support is to replace lost nutrients and to provide adequate caloric intake for growth and normal metabolic functions. While no special diet has been

**TABLE 23-1 Comparison of Ulcerative Colitis and Crohn’s Disease**

<table>
<thead>
<tr>
<th>Pathology</th>
<th>Ulcerative Colitis</th>
<th>Crohn’s Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Area of involvement</td>
<td>Colon and rectum</td>
<td>May affect entire GI tract from mouth to anus</td>
</tr>
<tr>
<td>Bowel wall involvement</td>
<td>Superficial (mucosa and submucosa)</td>
<td>All layers of bowel wall (transmural)</td>
</tr>
<tr>
<td>Distribution of lesions</td>
<td>Symmetric, continuous</td>
<td>Asymmetric, segmented (disease-free skip areas)</td>
</tr>
<tr>
<td>Clinical Manifestations</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alteration in bowel pattern</td>
<td>Severe diarrhea</td>
<td>Mild to moderate diarrhea</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>Mild, lower abdominal</td>
<td>Common</td>
</tr>
<tr>
<td>Rectal bleeding</td>
<td>Common</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Weight loss</td>
<td>Mild to moderate</td>
<td>Common</td>
</tr>
<tr>
<td>Growth retardation</td>
<td>Mild</td>
<td>Often severe</td>
</tr>
<tr>
<td>Perianal disease</td>
<td>Rare</td>
<td>Common</td>
</tr>
<tr>
<td>Fistulas</td>
<td>Rare</td>
<td>Common</td>
</tr>
</tbody>
</table>
proven effective for treating IBD, some individuals find their symptoms are aggravated by milk, highly seasoned foods, and fiber. The lactose in milk and milk products is usually poorly tolerated when IBD is active, resulting in bloating, pain, and increased diarrhea. This is a problem because dairy products constitute the largest source of calories in the diets of most children. Lactase hydrolyzed milk such as Lact-Aid can be helpful in providing extra calories for lactose intolerant children. High calorie liquid nutritional supplements such as Ensure may be recommended for children with growth failure. Vitamins and minerals are often deficient and replacements are necessary. Since fats are digested and assimilated in the small intestine, children with CD have a deficiency of the fat-soluble vitamins, A, D, E, and K. Elemental formulas, which are almost completely absorbed in the small intestine and leave little residue, have been useful in inducing remission and improving nutritional status. These formulas can be given either by mouth or nasogastric tube feeding at night. Total parenteral nutrition (TPN) in children with severe CD can help to reverse growth failure. Most individuals with IBD find that a low-fiber, low-residue diet is therapeutic.

Another approach to treatment is surgery. When children experience severe complications of UC, surgery may be indicated. Severe complications are bowel perforation, hemorrhage, and conventional treatment failure. Surgical removal of the entire colon and rectum (proctocolectomy) provides a permanent cure. A permanent ileostomy is created at the same time. Indications for surgery in cases of CD are disease that is unresponsive to medical treatment, bowel strictures, obstruction, or perforation, and intractable bleeding or diarrhea. The diseased segment of the intestine is removed or resected, and the two ends of healthy intestine are reattached or anastomosed. CD is not cured with surgery because the lesions tend to recur in other parts of the bowel.

Nursing Management
The focus of nursing care includes medication and nutritional management, emotional support, and community referrals. An area for potential strife between the child and caregivers is the medication regimen. When IBD is in remission, the child may see no reason for taking medications. The concept that the disease is still present although no symptoms are evident is often difficult for children to understand. The nurse should emphasize continuation of medications despite remission of symptoms. The nurse can provide information about medications used to treat IBD and their side effects, emphasizing that they be continued despite remissions of symptoms. The side effects of corticosteroids include increased appetite and weight gain, increased susceptibility to infections, increased risk for osteoporosis and aseptic necrosis of the hip, acne, rounding of the face, and personality changes. Side effects of the sulfasalazine (Azulfidine) include gastric upset, nausea, vomiting, allergic reactions, crystalluria (crystals in the urine), and bone marrow suppression. The total dose of the medication should be given in evenly spaced doses and after meals to minimize gastrointestinal upset. The child should be encouraged to drink a full glass of water with each dose to prevent crystalluria.

Providing emotional support is an important nursing intervention in IBD. In addition to the expected effects of chronic illness, depression, anxiety, and low self-esteem appear to be more common in children and adolescents with IBD (Mascarenhas & Altschuler, 1997). Early detection of psychological problems is invaluable because appropriate referral and psychological therapy can help prevent further psychopathology. Often both the child and the family require counseling. Support groups are also useful in helping the child and caregivers deal with the diagnosis and disease. The nurse should assess the impact of the disease as reflected by impaired social activities and school absences. School activities (e.g., gym and bathroom privileges) may need to be modified.

Peptic Ulcers
Peptic ulcers occur when there is erosion of the mucosal wall of the gastrointestinal tract. They develop most often in the stomach and duodenum. They are classified as primary or secondary, gastric or duodenal. Primary ulcers occur in the absence of another underlying disease and often in individuals with a family history of the disorder. Secondary or stress ulcers are associated with severe physiological stress of an underlying systemic disease or injury such as shock, sepsis, burns, or surgery. Certain drugs also contribute to secondary ulcers. Gastric ulcers are usually located at the junction of the fundus and the pylorus on the lesser curvature of the stomach. Duodenal ulcers occur in the pylorus or duodenum. Figure 23-13 illustrates the most common sites for peptic ulcers.

Incidence and Etiology
The true incidence of peptic ulcers in children is unknown; however, with the advent of endoscopy, more cases have been detected and diagnosed. Primary ulcers are most common in
Family Teaching

Inflammatory Bowel Disease

HOME:
- Teach the child and family about medication regimen, side effects of drugs, and the need to continue drug therapy despite remission of symptoms.
- Instruct the child and family about the dietary plan. A low-fiber, low-residue diet generally provides some relief of symptoms such as diarrhea and abdominal pain.
- Teach caregivers methods to increase oral caloric intake such as (1) eating 3 meals plus 3 “mini-meals” each day; (2) including a source of protein at each meal; (3) ingesting milk products as tolerated (yogurt with live cultures and aged, hardened cheeses are tolerated by many children with lactose intolerance); (4) using extra fats as tolerated, such as butter, margarine, vegetable oil, mayonnaise, cream, and cream cheese; and (5) limiting low-calorie foods and beverages.

SCHOOL:
- Instruct the family that the child can be as active as she or he feels like being.
- Encourage caregivers to try giving the child the responsibility of determining whether she/he is able to go to school during flare-ups.
- Encourage the child to find practical ways of dealing with the illness in school, such as locating the nearest bathrooms, carrying extra underclothes, and visiting the school nurse as needed.
- Assess for school avoidance because of embarrassment of diarrhea. If the child is not active in school, some element of the program should be adjusted.
- Allow the child to make the decision whether to tell friends and teachers about the illness. If there are frequent hospitalizations or frequent absences from school, teachers and school administrators should be told about the illness.
- Have caregivers contact the school district to arrange for tutoring if extended absences from school become necessary.

COMMUNITY:
- Refer the family to the Pediatric Crohn’s and Colitis Association and the Crohn’s and Colitis Foundation of America (see Resources). Both organizations provide information and support that families may find helpful.
- Refer caregivers to social services, the visiting nurse association, and home health care agencies.

Figure 23-13 Most Common Sites for Peptic Ulcers
older children and adolescents. Up to the age of 6, most ulcers are secondary (Sondheimer & Silverman, 1995). Gastric ulcers are uncommon in children, whereas duodenal ulcers are seen most frequently. Males are affected with peptic ulcers more than females.

The exact etiology is unknown; however, several factors have been implicated. There is a close association between the bacilli *Helicobacter pylori* (*H. pylori*) and duodenal ulcers. This organism is transmitted by the fecal-oral route and is more common in lower socioeconomic areas and in developing countries (Heslin, 1997). Certain drugs contribute to peptic ulcers, for example, nonsteroidal anti-inflammatory agents such as aspirin and ibuprofen (Advil), corticosteroids, tobacco, and alcohol. For many years, diet and psychological factors were suggested as important etiologic factors; however, there is no conclusive evidence that they cause peptic ulcers.

**Pathophysiology**

The parietal cells of the stomach secrete hydrochloric acid (HCl) in the digestive process; other cells secrete pepsinogen. Pepsinogen converts to pepsin when activated by HCl, which adds to the acidity of the stomach. Gastric epithelial cells secrete a mucus-bicarbonate barrier to provide protection from the acid and pepsin. Ulcers occur when a substance stimulates excessive HCl production, damages the mucus barrier, or decreases mucus production.

**Clinical Manifestations**

The signs and symptoms of peptic ulcers in children vary depending on their age. The clinical manifestations according to age are illustrated in Table 23-2.

<table>
<thead>
<tr>
<th>Age</th>
<th>Clinical Manifestations</th>
</tr>
</thead>
</table>
| 0–3 years | Primary ulcers: anorexia, vomiting, melena, hematemesis, crying after meals  
Secondary ulcers: hemorrhage and perforation |
| 3–6 years | Primary ulcers: vomiting related to eating, periumbilical or generalized pain  
Secondary ulcers: melena, hematemesis, perforation |
| 6–18 years | Melena, hematemesis, occult bleeding, anemia |

**Diagnosis**

An upper GI barium series is often the initial test for a child suspected of having peptic ulcers. With this exam the ulcer crater is detected; however, the practitioner is not able to biopsy the mucosa to determine if *H. pylori* is present. A more definitive test is an endoscopy of the upper GI tract. The ulcer crater can be directly visualized, and a biopsy can be obtained to detect the organism. A blood test is also available that detects *H. pylori* antibodies. A stool exam for occult blood may be performed to diagnose GI bleeding.

**Treatment**

The goals of treatment are to relieve pain, hasten healing, and prevent complications. Medications are the primary method for managing ulcers. The rationale for medication therapy involves different mechanisms:

- Neutralization or buffering of gastric acid (antacids)
- Reduction of gastric acid secretions (histamine receptor antagonists)
- Suppression and blockage of gastric acid secretions (proton pump inhibitors)
- Protection of the mucus barrier (mucosal barrier fortifiers) by decreasing the activity of pepsin and HCl
- Treatment of *Helicobacter* infections (antibiotics and bismuth preparations)

Antacids decrease discomfort and pain but do not affect healing of the ulcer or prevent recurrence. A common antacid dosage schedule is 1 to 3 hours after each meal and at bedtime. The most common histamine (H) receptor antagonists for peptic ulcers are ranitidine (Zantac), cimetidine (Tagamet), and famotidine (Pepcid), all of which have few side effects, primarily diarrhea or constipation, headache, and rash. In some cases, proton pump inhibitors, which effectively suppress or block all gastric acid secretions are used in children. Omepazole (Paxil), the most commonly prescribed proton pump inhibitor, has minimal side effects that are similar to H-receptor antagonists. Mucosal barrier fortifiers such as sucralfate (Carafate) coat the stomach, adhere to the ulcer surface, and reinforce the mucosal protective coat of the stomach to prevent further digestive action of HCl and pepsin. It is administered on an empty stomach 1 hour before or 2 hours after meals and at bedtime, and constipation is the more common side effect. Cure of peptic ulcers associated with *H. pylori* requires eradication of the organism. The optimal therapeutic regimen is still undetermined; yet, in children, the antibiotics metronidazole (Flagyl) and ampicillin in combination with bismuth subsalicylate (Pepto-Bismol) is most often prescribed. Diet therapy is not indicated in the treatment of ulcers because restriction of diet does not promote or accelerate healing. Surgery is rarely needed in children but is indicated if perforation, hemorrhage, or gastric outlet obstruction occurs.
Nursing Management and Family Teaching
Because peptic ulcers are usually managed at home by caregivers, a major nursing intervention is caregiver education. Adherence with the medication regimen is important in healing the ulcer and preventing recurrences; therefore, the child and caregivers need to understand the rationale for each drug, the administration schedule, and side effects. The nurse explains that a special diet is not necessary, but the child should avoid substances that increase acid secretion such as caffeine-containing beverages (coffee, tea, cola). Any food or beverage that causes discomfort or pain needs to be avoided. Older children and adolescents need information about how alcohol and cigarette smoking cause gastric irritation and contribute to ulcer formation. It is important that caregivers know the signs and symptoms of ulcer complications, which must be reported to their health care provider immediately. Melena (black or tarry stool indicating presence of blood) or hematemesis (vomiting of blood) indicate hemorrhage; severe abdominal pain and a rigid abdomen may signal perforation.

The nurse can be instrumental in preventing secondary or stress ulcers that are due to physiological stress or certain medications by identifying infants and children who may be at risk for developing these ulcers. For critically ill individuals, maintaining gastric pH above 3.5 will help prevent ulcer formation. Thus, gastric pH values should be checked frequently and treated if too low. Additionally, histamine-2 blockers may be given as prophylaxis therapy in those identified at high risk for stress ulcers.

Necrotizing Enterocolitis
Necrotizing enterocolitis (NEC), a life-threatening condition of preterm neonates, is characterized by necrosis of the mucosa of the small and large intestine, most frequently the distal ileum and proximal colon. It is the most common surgical emergency in this age group. The necrosis may be very superficial and only detectable microscopically, or it may be through the bowel wall. Mild disease may be completely reversible, but neonates with extensive involvement may not survive.

Incidence and Etiology
NEC commonly occurs in preterm, low birthweight neonates and is rarely seen in term infants. It equally affects all races and both sexes. The incidence has been rising in recent years because of the improved survival of this high-risk group. The exact etiology of NEC is unclear; however, several factors are associated with its development. These risk factors include intestinal ischemia, bacterial colonization of the bowel, and the presence of hypertonic solutions in the intestinal lumen, usually formula (Kamitsuka, Horton, & Williams, 2000). Perinatal asphyxia, respiratory distress syn-

Pathophysiology
NEC appears to occur in preterm neonates whose bowel has experienced an injury, resulting in vascular compromise. This leads to decreased blood flow to the bowel and ischemia of the intestinal mucosa. The disruption of the intestinal mucosal barrier introduces significant vulnerability to infection. Then normal intestinal bacteria hydrolyze (to cause a substance to break down into its component parts by adding water) formula in the intestine, forming gas or air in the bowel wall called pneumatosis intestinalis. The bowel becomes edematous and distended. Progressive infiltration of the bowel wall with bacteria leads to more extensive tissue inflammation, destruction, and necrosis. Sepsis and perforation of the bowel may occur.

Clinical Manifestations
The classic clinical presentation of the neonate with NEC includes the symptom group of abdominal tenderness, distention, and erythema of abdominal wall, bloody stools, decreased bowel sounds, increased gastric residuals (feeding retained in stomach following tube feeding), and bilious vomiting after feeding. Manifestations of clinical deterioration include apnea and bradycardia, lethargy, temperature instability, decreased urine output, further abdominal distention, and evidence of shock (cool, mottled skin, pallor, decreased intensity of peripheral pulses). Hypotension is a late sign of deterioration. Acidosis, sepsis, and death may occur if NEC is not treated.

Diagnosis
Diagnosis is based on clinical findings and abdominal X rays. Radiographic findings associated with NEC are dilated bowel loops and pneumatosis intestinalis. Pneumoperitoneum, free air in the peritoneal cavity, or air in the portal circulation indicate severe disease and perforation of the bowel.

Treatment
If NEC is diagnosed in its early stages and treatment is initiated promptly to prevent perforation, the infant may improve without surgical intervention. Initial treatment includes:

- Cessation of oral feedings
- Continuous gastric drainage and decompression via an NG tube
• Maintenance of oxygenation; ventilation if necessary
• Administration of IV fluid therapy for parenteral nutrition and broad spectrum antibiotics, and to restore acid-base and electrolyte balance

Frequent monitoring of laboratory data is essential in order to detect deterioration in the infant’s condition. Commonly ordered tests include blood gases, white blood cell count, hematocrit, platelet count, electrolytes, and abdominal X rays.

Despite appropriate medical treatment, surgical intervention becomes necessary in 40% to 50% of cases (Maalouf, et al., 2000). Surgery is required if the infant demonstrates evidence of perforation, localized peritonitis, persistent metabolic acidosis, or clinical deterioration unresponsive to vigorous medical management (see Box 23-2).

Resection of the necrotic bowel is necessary, and in cases of extensive removal, intestinal diversion is performed by creating a temporary ileostomy, jejunostomy, or colostomy. Postoperative complications include intestinal obstruction secondary to stenosis of the ischemic portions of the bowel and short bowel syndrome characterized by malabsorption, malnutrition, and growth failure.

Nursing Management
The nurse has a major responsibility to be aware of and continually assess for early warning signs of NEC.

Nursing Alert:
Warning signs of NEC
• Abdominal distention
• Residual gastric contents
• Feeding intolerance
• Decreased bowel sounds
• Bloody stools

Frequent measurement of abdominal girth is performed to assess for distention. Prior to feedings gastric residual contents are measured to determine if the volume is increasing, which indicates malabsorption. The presence of bowel sounds is also noted to evaluate for decreased intestinal activity, and all stools are tested for blood. When NEC is diagnosed, nursing interventions include accurate intake and output measurements, frequent assessment of vital signs, maintenance of IV therapy, and ongoing assessment for changes in the infant’s condition. Vital signs are monitored for changes that may reveal impending sepsis or shock from perforation and peritonitis. When oral feedings are restarted, the nurse must observe the infant’s response and tolerance because NEC can recur.

MALABSORPTION ALTERATIONS

Malabsorption occurs when there is a disruption in the digestive process causing insufficient assimilation of nutrients. Common causes of problems in absorption in infants and children that will be discussed are celiac disease and lactose intolerance.

Celiac Disease
Celiac disease, also known as gluten-sensitive enteropathy, is a disorder caused by permanent intolerance to gluten, the protein component of wheat, barley, rye, and oats. It is second only to cystic fibrosis as the most common cause of malabsorption in children.

Incidence and Etiology
Celiac disease is a genetic disorder that occurs in all races but is more common in Caucasians. The incidence varies in different regions and is more common in Europe than the United States. In the U.S. it is 0.1 in 1,000 live births, and the disease coincides with the introduction of foods containing gluten (Ulsben, 1996).

Pathophysiology
The exact mechanism by which gluten damages the mucosa of the small intestine is unclear. One theory postulates that gluten toxicity results in an alteration in immunologic response. Gluten consists of two protein components, glutenin and gliadin. The harmful protein appears to be gliadin. Gliadin plays the role of antigen and causes an immune response that results in inflammation of and damage to the finger-like projections called villi in the small intestine. The villi flatten out and atrophy, leading to a decrease in the absorptive surface area. Initially, fat absorption is impaired, followed by protein, carbohydrates, and fat-soluble vitamins (A, D, E, K).
Clinical Manifestations

Early clinical manifestations of celiac disease are subtle and include anorexia, irritability, listlessness, and weight loss. As the disease progresses, abdominal distention and chronic diarrhea appear with large amounts of unabsorbed fats being excreted in the stools (steatorrhea). The stools are bulky, putty colored, foul-smelling, greasy, and often float because of the high fat content. Signs of progression include a protuberant abdomen, loss of subcutaneous fat, and muscle wasting secondary to hypoproteinemia. The child may appear pale because of anemia, and bruising may develop secondary to inadequate vitamin K absorption. Late signs include severe growth retardation, osteoporosis, and in the adolescent, delayed menses or puberty.

Diagnosis

The definitive diagnostic test is a small bowel biopsy, which will reveal atrophy of the villi and deep crypts on the intestinal mucosa. These characteristic lesions return to normal after dietary restriction of gluten, which help confirm the diagnosis. Serologic tests to detect antigliadin and antidomysial antibodies are commonly ordered. Laboratory tests may be used to evaluate malabsorption and nutritional deficiencies (Stark, 1999).

Treatment

Medical management consists of a lifelong adherence to a gluten-free diet. Education about the diet is the main goal of treatment and involves the health care provider, nurse, dietician, caregivers, other family members, and the child. All wheat, barley, rye, and oats are eliminated and substituted with rice, corn, and millet. Specific nutritional supplements may be used to correct deficiency states. The most common are supplements of iron, folate, calcium, and fat-soluble vitamins. The intolerance to gluten is permanent, and lack of adherence to a gluten-free diet can cause exacerbation of symptoms.

Nursing Management and Family Teaching

The long-term goal of nursing care is to provide dietary education and supervision. The nurse explains to the caregivers the disease process, the signs and symptoms, and the rationale behind the gluten-free diet. A dietician should be involved in diet planning and nutrition education, and serves as a resource for gluten-free foods and recipes. Families are taught to read labels of all commercially prepared foods for the presence of gluten or gluten-containing additives such as hydrolyzed vegetable protein.

Children and their families often react to the necessity of a gluten-free diet with grief and may have a hard time accepting that something so fundamental to their diet could be injuring the child. Caregivers should be forewarned that many adolescents specifically have a difficult time accepting the dietary restrictions and may experiment with foods containing gluten. They may be motivated to adhere to the restrictions by the expectation of dramatic improvements in gastrointestinal symptoms such as bloating, abdominal pain, and diarrhea, and in their general well-being (Murray, 1999). Referrals to community resources for dietary and peer support organizations are important (see Resources).

Lactose Intolerance

Lactose intolerance is the inability to digest lactose, a sugar (disaccharide) present in human and cow’s milk, standard infant formulas, and dairy products such as cheese and ice cream. Lactose is also added to many prepared foods, including bread and other baked goods, breakfast cereals, and mixes for cakes, cookies, pancakes, and biscuits. This disorder results from a deficiency or absence of lactase, an enzyme in the small intestine required for the digestion and absorption of lactose. Lactose intolerance can be congenital or acquired. In the congenital type, which is extremely rare, the newborn is born with a complete absence of lactase. Acquired lactose intolerance involves the gradual loss of lactase, is more common, and appears from early childhood to late adolescence.

Incidence and Etiology

The incidence appears to vary widely among different ethnic and racial groups. Low lactose levels are least common among northern and western Europeans and highest among populations from the Far East. In the United States the incidence is lowest in Caucasians and highest in Vietnamese-Americans, Native Americans, and African-Americans. Lactase intolerance is most often secondary to other disorders. Diarrheal disease, particularly when caused by rotavirus, other infections of the small intestine, and acquired immune deficiency syndrome (AIDS) may decrease the activity of lactase. Another cause may be mucosal damage to the small bowel induced by dietary components such as gluten sensitivity (celiac disease), and sensitivity to soy protein and cow’s milk (Castiglia, 1994).

Pathophysiology

The absence or deficiency of lactase results in the inability of the small intestine to digest lactose. Subsequently, the undigested lactose moves into the colon where GI bacteria break down the lactose and release hydrogen, methane, and carbon dioxide. This process causes excessive gas production and abdominal bloating and pain. The undigested lactose also causes an increased number of solutes in the colon, resulting in an increase in the osmotic pressure. Therefore, water is drawn into the colon causing watery diarrhea.

Clinical Manifestations

Symptoms occur in response to ingestion of lactose and include explosive, watery diarrhea, abdominal distention,
abdominal pain, and excessive flatus. Symptoms develop rapidly after the child ingests milk or foods containing lactose. Some children are able to tolerate small amounts of lactose without symptoms; yet, when larger amounts are consumed, severe symptoms occur.

**Diagnosis**

Diagnosis is usually made using a hydrogen-breath test that measures the amount of hydrogen left after fermentation of undigested and unabsorbed carbohydrates such as lactose.

**Treatment and Nursing Management**

Treatment consists of reducing or eliminating lactose from the child's diet. In most cases, total elimination is unnecessary. For infants, switching to a soy-based formula (Isomil, Nutramigen, Prosobee) is effective. For older infants and children, when fluids or foods containing lactose are consumed, a commercial lactose preparation (Lact-Aid, Dairy-Ease) can be ingested or sprinkled on the items to improve tolerance. Additionally, milk products that have been commercially pretreated with microbial derived lactase are available (McBean & Miller, 1998).

**POISONING**

A poison is any substance that harms the body and interferes with the body's normal functioning. A poisoning can occur through ingestion, inhalation, skin exposure, eye contact, or any other mode that causes adverse effects. Ingestion accounts for the majority of poisonings.

**Incidence and Etiology (Pathophysiology)**

In 1998 approximately 1.1 million cases of ingestion of a toxic substance by children less than six years of age were reported to poison control centers (Shannon, 2000). Since many children with toxic ingestions are managed without contact with poison control centers or emergency facilities, the total number of cases is much larger than the numbers reported by these centers. Poisoning is the leading cause of injury and the fourth leading cause of death in toddlers and preschoolers. In infants their tendency to explore objects with their mouth puts them at high risk for accidental ingestion of toxic substances. In toddlers and preschoolers, poisoning is more often a result of curiosity. Older children may experiment with drugs and household products to produce hallucinogenic effects (Dunn & Burns, 2000). Because ingestions in children under the age of six are most commonly accidental, they rarely ingest enough poison to cause death. In contrast, adolescents are less frequently exposed to poisons but their exposure is more commonly intentional and results in more fatalities than in younger children. The incidence of childhood poisoning has decreased significantly in the past 50 years because of federal regulation of products and product safety, child-resistant containers, and safe storage of toxic substances in the home and elsewhere (Shannon, 2000).

Although the majority of poisonings occur in the home, incidents may occur anywhere medications and toxic substances are stored. Substances commonly ingested by children less than six years of age are listed in Table 23-3.

Adolescents tend to ingest psychopharmacologic drugs such as tranquilizers, sedatives, and antidepressants.

**Clinical Manifestations**

Clinical manifestations are dependent on the specific poison ingested. Table 23-4 lists the signs and symptoms associated with toxins that are frequently ingested and/or that cause significant mortality.

**Diagnosis**

Identification of the type and amount of the exposure is important. Physical findings, a detailed history, and examination of

| TABLE 23-3 Agents Most Commonly Ingested by Children Less Than Six Years of Age |
|---------------------------------|----------------------------------|
| **Type**                        | **Examples**                     |
| Cosmetics and personal care     | Perfume                          |
| products                        | Cologne                          |
| Cleaning products               | “Household” bleach               |
| Plants                          | Pine oil disinfects              |
| Foreign bodies/toys/miscellaneous | Thermometers                     |
| Hydrocarbons                    | Bubble-blowing liquid            |

the medication containers may suggest the type of toxin. In the history the following information should be obtained: who—the child’s age and weight; what—the name and dosage of the medication or substance ingested; when—the time of ingestion; how—the route of poisoning (ingested, inhaled, absorbed, or injected); and why—whether intentional or unintentional. Information about signs and symptoms that have appeared since the poisoning, emergency care given, and whether vomiting was induced or occurred spontaneously should be determined. A detailed past medical history should also be obtained including previous poisonings, medical conditions, and medications currently taken that might affect the child or adolescent’s response to and metabolism or elimination of the toxic substance. Analysis of specimens such as emesis can be helpful in determining the type of poison. Laboratory evaluation may be performed when the poison is unknown, if the poison has the potential to produce moderate to severe toxicity, and if the ingestion was intentional (Larsen & Cummings, 1998).

**Treatment**

Treatment approaches vary with the type of poison, amount of exposure, time elapsed since exposure, and susceptibility of the child. Stabilization of the child is the first priority in managing toxic ingestions and should address the ABCs (airway, breathing, circulation). Vital body functions must be maintained regardless of the poison. Oxygen may be administered. Maintenance of respiratory function may require endotracheal intubation and/or mechanical ventilation.

Following stabilization of the individual, attention is directed toward gastric decontamination (decreasing absorption of the ingested poison from the GI tract). This includes use of emesis (syrup of ipecac), gastric lavage, an absorbent agent (activated charcoal), or a cathartic agent. Gastric emptying with an emetic or lavage should not be used routinely in all oral poisonings because it is ineffective when used at a late stage, may delay more effective interventions, and may cause complications such as aspiration (Herrington & Clifton, 1995). Yet, it is beneficial when used early in the treatment of potentially severe poisonings and is most effective when used within one hour of the ingestion (Larsen & Cummings, 1998). The stomach may be emptied by inducing emesis with syrup of ipecac or gastric lavage.

**Syrup of Ipecac**

Syrup of ipecac may be used to induce vomiting; however, the absorption of the poison is only reduced by about 30 percent when it is administered within one hour of ingestion.
Vomiting will usually occur within 20 minutes and may last for several hours. Any emesis should be inspected for pill fragments and/or saved for analysis. The child should be closely observed and positioned on the left side to prevent aspiration.

**Gastric Lavage**

In most situations, gastric lavage is preferable to administration of ipecac, particularly in emergency departments where prolonged ipecac-induced vomiting may delay more effective interventions. Lavage is used for gastric emptying in the first 1 to 2 hours after the ingestion. It is indicated when the substance ingested is highly toxic (large ingestions or substances associated with high morbidity and/or mortality); when the toxin is not well absorbed by activated charcoal (i.e., lithium, iron, lead, methanol); or in children with the potential for a jeopardized airway (e.g., altered alertness) (Phillips, Gomez, & Brent, 1993). Contraindications to gastric lavage include ingestion of corrosives and ingestions by children with depressed gag reflexes who are not intubated. Complications of lavage are aspiration and perforation of the esophagus or bronchus. The procedure involves insertion of a nasal or orogastric tube and administration of small amounts of normal saline through the tube until the fluid returned is clear.

**Activated Charcoal**

Activated charcoal is effective for most oral poisonings when given alone or following the use of ipecac or gastric lavage. The use of activated charcoal decreases the amount of the toxic agent available for absorption by the gastric mucosa by up to 75 percent. It can be given when the ingestion has occurred up to two hours prior to treatment. The main concern with activated charcoal is vomiting, which occurs in approximately 15% of children and increases the risk of aspiration and pneumothorax (Shannon, 2000).

**Cathartic Agents**

Administration of cathartic agents increases GI motility and hastens the expulsion of the toxin. Magnesium citrate and sorbitol are the two most commonly used agents.

**Antidotes**

Antidotes are available for several of the common and dangerous poisons. They are typically given once the child has been stabilized, usually within a few hours of the ingestion. Examples of antidotes for some common toxins include (1) N-Acetylcysteine (Mucomyst) for acetaminophen, (2) bicarbonate for tricyclic antidepressants, (3) deferoxamine (Desferal) for iron, (4) EDTA for lead, (5) ethanol for methanol and ethylene glycol, (6) flumazenil (Romazicon) for benzodiazepines, and (7) naloxone (Narcan) for narcotics such as opiates.

**Nursing Management**

The solution to the problem of childhood poisonings is prevention. The nurse can discuss various preventive measures. To facilitate protection of the child, the environment should be modified during infancy before she or he crawls. The nurse should teach caregivers to call the poison control center before instituting treatment if their child has been exposed to a toxic substance. Box 23-3 lists poison prevention guidelines to teach caregivers.

**LEAD POISONING**

Even though there has been a decline in the average blood lead level (BLL) among the population, children continue to be exposed to lead, and it is still a major environmental health problem that could harm their health and impair their ability to learn (CDC, 2000). Based on data from Phase II of the 1991–1994 National Health and Nutrition Survey.
(NHANES) III, the CDC estimated that 890,000 (4.4%) children between 1 and 5 years of age had elevated BLLs, above 10µg/dl (CDC, 1997). The BLL rate was 5.9% among children aged 1–2 years and 3.5% among children 3–5 years. Children between 1 and 5 were more likely to have elevated BLLs if they were of non-Hispanic, African-American heritage, were poor, or lived in older housing. 21.9% of non-Hispanic African-American children and 13% of Mexican-American children living in housing built before 1946 had higher BLLs than non-Hispanic Caucasian children (5.6%) living in similar housing (CDC, 2000).

Due to these figures, in 1997 the CDC changed its national blood lead screening recommendations to an approach that was state-based (CDC, 2000). Screening Young Children for Lead Poisoning: Guidance for State and Local Public Health Officials, (1997), suggested state health departments assess risk factors and local data on BLLs. The CDC also recommended screening children receiving Medicaid for lead unless “reliable, representative blood lead data that demonstrate the absence of lead exposure among this population” exists. Specifically, the recommendations to health care providers were to screen BLLs of all children between 1 and 2 years of age enrolled in Medicaid, refer children identified as having elevated BLLs to environmental and public health services, and provide medical management that is appropriate if the blood levels were elevated (CDC, 2000).

HEPATIC ALTERATIONS

The liver performs a wide variety of vital functions; therefore, hepatic alterations can result in life-threatening severe illness. Viral hepatitis, the most common of these disorders, will be discussed.

Hepatitis

Hepatitis is an acute or chronic inflammation of the liver caused by several viral or bacterial infections, fungal or parasitic infections, or chemical and drug toxicity. Five distinct viruses have been identified as causing hepatitis: hepatitis A virus, hepatitis B virus, hepatitis C virus, hepatitis D virus, and hepatitis E virus. In this section hepatitis A, B, and C will be discussed. Hepatitis D and E are very uncommon in children.

Hepatitis A causes only acute hepatitis, whereas hepatitis B and C cause chronic infections. Hepatitis viruses are classified as enteral or parenteral, in reference to their mode of transmission. The enteral form, hepatitis A, is transmitted by the fecal-oral route. Parenteral forms, hepatitis B and C, are transmitted via venous blood transfer or through intimate sexual contact. Currently vaccines are available to prevent hepatitis A and B.

Incidence and Etiology

Hepatitis A

Hepatitis A (HAV) is responsible for most cases of hepatitis in children and occurs most frequently in children 5 to 14 years of age. It is caused by oral ingestion of the hepatitis A virus, which is found in the stool of infected individuals. Because the virus is transmitted via the oral-fecal route, it is easily spread in areas where there are poor sanitary conditions or where good personal hygiene is not observed. Employees and children in daycare settings are at high risk for developing the disease. The risk of spread and an outbreak in this setting is related to the number of infants and children in diapers (American Academy of Pediatrics, 1997). The source of infection is either contact with an infected person or direct contact with infected fecal material that has entered food or water supplies. Outbreaks have been related to sewage-contaminated water, infected food handlers (who do not wash their hands after defecating), and shellfish caught in waters contaminated by sewage. In children, HAV is characterized by either a mild course similar to that of influenza or is asymptomatic.

Hepatitis B

Hepatitis B (HBV), previously called serum hepatitis, is spread parenterally via direct contact with infected blood or body fluids. It can be an acute and/or chronic infection and is potentially lethal. Most cases of HBV in children are acquired perinatally from an infected mother during preg-
nancy and/or delivery. The disease can also be acquired from contaminated needles, especially affecting IV drug users, through sexual activity, and from blood transfusions. The clinical course of hepatitis B may be varied. It may have an insidious onset with mild or no symptoms, which is common in children, or it may result in serious complications such as fulminant or chronic hepatitis.

**Hepatitis C**

Hepatitis C (HCV) in children has been observed most frequently after transfusion with blood and blood products; therefore, the incidence is highest in hemophiliacs. Similar to HBV, HCV can be transmitted perinatally. The average rate of HCV infection among infants born to HCV positive mothers is 5–6%. Because HCV can be transmitted through blood transfusions or perinatally, the American Academy of Pediatrics recommends screening for the following groups: (1) infants born to HCV infected mothers; (2) drug users (injecting); (3) recipients of 1 or more units of blood or blood products prior to 1990; (4) individuals receiving hemodialysis; and (5) individuals receiving clotting factor concentrates before 1987 when effective inactivation procedures were introduced. HCV can also be transmitted sexually; however, it does not appear to be acquired as easily by sexual contact as does HBV. In sexually promiscuous individuals, the risk of infection is related to the number of sexual partners (Rajan-Mohandas, 1999).

**Pathophysiology**

After exposure to the hepatitis virus, the liver becomes inflamed, causing damage to the cells. As the liver becomes edematous, bile channels from the liver into the intestine become obstructed, causing biliary stasis and further destruction of cells. In most cases the disease is self-limiting and liver cells regenerate completely within 2–3 months. However, hepatitis B and C may be associated with continued degeneration of liver cells and chronic hepatitis. Chronic hepatitis is characterized by progressive liver failure, cirrhosis, and/or liver cancer. Fulminant hepatitis, a rare but often fatal complication of HBV and HCV, can also occur. It results from failure of the liver cells to regenerate, causing massive hepatic necrosis. Death can occur within 1–2 weeks.

**Clinical Manifestations**

The manifestations of viral hepatitis are similar. Generally, children have mild, nonspecific symptoms without jaundice or are asymptomatic. Initially, the child experiences nausea and vomiting, anorexia, slight fever, fatigue, headache, and abdominal pain in the epigastrium or upper right quadrant. These flu-like symptoms last approximately 1 week and may be so mild that they go unnoticed in infants and young children. Following this period, jaundice may develop, beginning with darkening of the urine and gray-colored stools, followed by yellowing of the skin and sclera. However, many children with acute hepatitis never develop jaundice. The liver usually is enlarged and tender to palpation. Children with HBV and HCV may also present with dermatologic symptoms such as rashes and pruritus or severe itching. Refer to Table 23-5 for a comparison of hepatitis A, B, and C.

**Diagnosis**

Diagnosis of hepatitis is based on history, specifically exposure to the hepatitis virus, physical examination, serologic testing for markers of hepatitis A, B, and C, and liver function tests. Diagnosis is confirmed by the presence of antigens or antibodies formed in response to specific hepatitis viruses. In hepatitis, liver enzymes are elevated, specifically ALT, AST, and serum total bilirubin, indicating liver damage.

**Treatment**

There is no specific treatment for hepatitis, which is generally supportive. The management for children is based on measures to rest the liver, promote cellular regeneration, and prevent complications. Rest is an essential focus of treatment to reduce the liver’s metabolic demands and increase its blood supply. Treatment is aimed at maintaining comfort and providing adequate nutrition.

Once the diagnosis of hepatitis is made, attention should be directed to prevention. Vaccines have been developed to prevent HAV and HBV. Children who have been exposed to a person with HAV should receive standard immune globulin (IG) within 2 weeks of exposure. Immune globulin when given in this time period is 80–90% effective in preventing the disease. Hepatitis A vaccine is approved for children at risk aged 2 through 18 years of age. The vaccine is routinely recommended for children living in communities with high HAV rates or periodic outbreaks of infection (AAP, 1997).

Hepatitis B vaccine is recommended for all newborns as part of the routine childhood immunization schedule. All children who have not received the vaccine previously should be immunized by or before 11 to 12 years of age. Additionally, administration of hepatitis B immune globulin (HBIG) is recommended for individuals exposed to HBV. If given within 2 weeks of exposure, HBIG is effective in preventing the infection (AAP, 1997).

**Nursing Management and Family Teaching**

Nursing care is directed toward supportive care and education of the family about prevention measures. Most children with mild or uncomplicated hepatitis are cared for at home. Because fatigue and listlessness can last for weeks, children usually limit their own activity during the early stages of the disease. Anorexia is common; therefore, small, frequent
meals and snacks are tolerated well. The nurse should instruct caregivers to contact their health care provider prior to administering over-the-counter medications since normal doses of many drugs may be toxic. A primary focus of education is prevention of the spread of the infection as delineated in Box 23-4. Additionally, the nurse should educate the family about the method of transmission of hepatitis and about the availability of immunoprophylaxis after exposure and of vaccines for HAV and HBV.

**ADDITIONAL GASTROINTESTINAL DISORDERS**

The following disorders are rare in children; therefore, they are discussed briefly.

**Abdominal Wall Defects: Gastroschisis and Omphalocele**

Gastroschisis and omphalocele are congenital malformations in which a defect in the abdominal wall allows portions of the abdominal contents to herniate outside the abdominal cavity. Their incidence is 0.1 to 0.3 in 1,000 live births (Howell, 1998). In gastroschisis the defect in the abdominal wall permits extrusion of the abdominal contents, primarily the small and large intestines, without involving the umbilical cord. The defect is usually to the right of the umbilicus, and there is no protective sac covering the intestines. The etiology is unclear, although one theory explains gastroschisis as resulting from an incomplete lateral infolding of the embryonic disc, which allows herniation of the bowel.

An omphalocele is centrally located, includes the umbilical cord, and the abdominal viscera and are covered by a protective sac. Omphalocele results from failure of the intestines to re-enter the abdominal cavity at approximately the 7th week of gestation. The size of the defect is variable,

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**TABLE 23-5 Comparison of Hepatitis A, B, and C**

<table>
<thead>
<tr>
<th>Type</th>
<th>Incubation Period</th>
<th>Mode of Transmission</th>
<th>Prevention</th>
<th>Possible Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>15–30 days</td>
<td>Fecal-oral through contaminated food or water</td>
<td>Immunization</td>
<td>Potential chronicity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Poor hygiene</td>
<td>Education on proper food handling</td>
<td>Cirrhosis</td>
</tr>
<tr>
<td>B</td>
<td>45–180 days</td>
<td>Perinatally</td>
<td>Immunization</td>
<td>Potential chronicity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Unsafe sex</td>
<td>Education to prevent exposures to blood and body fluids</td>
<td>Cirrhosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Poor hygiene</td>
<td>Needle exchange program</td>
<td>Liver cancer</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Blood transfusions</td>
<td>Identification of carriers</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Body secretions</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Contaminated needles</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>2 weeks–6 months</td>
<td>Blood transfusions</td>
<td>Education to prevent exposure to blood and body fluids</td>
<td>Potential chronicity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IV drug use</td>
<td>Needle exchange program</td>
<td>Cirrhosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Identification of carriers</td>
<td>Liver cancer</td>
</tr>
</tbody>
</table>

Clinical Manifestations
In gastroschisis the bowel eviscerates into the amniotic cavity, and exposure to the amniotic fluid results in thickened, beefy-red, edematous intestines. The bowel is normal in appearance in the neonate with omphalocele; however, the abdominal cavity is small and underdeveloped.

Treatment
Goals of initial management of the newborn with either of these disorders are to prevent hypothermia, maintain a sterile environment, and maintain tissue perfusion. Two accepted surgical techniques for these defects are a primary and a staged repair. Primary repair is the procedure of choice if the exposed abdominal contents will fit into the abdominal cavity. If not, a staged repair is performed. A synthetic material is used to create a sac to cover the abdominal contents. The bowel is then gradually returned to the abdomen over 7–10 days. The abdominal wall is closed in the second surgery.

Biliary Atresia
Biliary atresia is characterized by congenital absence or obstruction of bile ducts outside the liver (extrahepatic), thus preventing flow of bile from the liver to the intestines. There is no known cure for the disease. Females appear to be slightly more at risk for developing the disease than males. It is the single most frequent indication for liver transplantation in children. The cause is unknown; however, one theory postulates that a viral or other injury affected the developing bile duct system in utero or immediately after birth (Yoon, Bresee, O'he, James, & Khoury, 1997).

Clinical Manifestations
The newborn with biliary atresia is asymptomatic at birth; however, between 2 weeks and 2 months of age, jaundice appears. The infant’s urine is tea colored because of the excretion of bilirubin and bile salts. Stools are light in color because of the absence of bile pigments. Hepatomegaly may be present from the pathologic processes occurring, such as fibrosis of the liver. Failure to thrive and malnutrition eventually develop.

Treatment
Treatment involves surgery to correct the obstruction and allow for drainage of bile from the liver directly into the intestines. Hepatic portoenterostomy, such as the Kasai procedure, may be performed, thereby attempting to slow the pathologic processes that occur in the biliary duct. This surgery is not a cure, and not all surgeries are successful in delaying biliary duct injury. Complications of liver disease continue to develop and eventually result in end stage liver disease. A liver transplant is required at this point.

Cirrhosis
Cirrhosis is a pathologic condition of the liver that occurs secondary to liver disease or inflammation. Viral hepatitis, inborn errors of metabolism (galactosemia), congenital anomalies of the bile ducts (biliary atresia), and chronic diseases such as cystic fibrosis are the main disorders that cause severe liver disease and cirrhosis in infants and children. Cirrhosis is rare in the pediatric population. Fibrotic scar tissue develops in the liver as a result of chronic inflammation or disease, and the organ assumes an irregular, nodular appearance.

Clinical Manifestations
Clinical manifestations vary depending on the cause of cirrhosis. When the etiology is viral hepatitis, inborn errors of metabolism, or chronic disease, initially the child demonstrates vague symptoms of GI dysfunction, including lethargy, anorexia, and nausea. Steatorrhea is frequently present caused by disordered fat metabolism. In cases caused by biliary anomalies, ascites (accumulation of fluid in the peritoneal cavity) and portal hypertension develop. The most important sign of portal hypertension is splenomegaly, which produces anemia, leukopenia, thrombocytopenia, and often esophageal varices. The child may demonstrate easy bruising or epistaxis (nose bleeds), or GI hemorrhage. Jaundice and dark urine, and pruritis are other symptoms that occur with biliary malformations.

Treatment
Medical management focuses on preventing and treating the complications of cirrhosis. Nutritional treatment of malabsorption problems consists of a low-fat, low-protein diet and supplemental vitamins, especially fat-soluble ones. Ascites is treated with fluid restriction, decrease in sodium content of food, and diuretics. Hepatic encephalopathy is treated with reduction of protein intake and administration of lactulose (to control increased ammonia levels) and an antibiotic such as neomycin. Bleeding complications may necessitate administration of blood and blood products. Definitive treatment for cirrhosis and end stage liver disease is a liver transplant.

Umbilical Hernia
An umbilical hernia results from incomplete closure of the umbilical ring, which allows the intestines to protrude through the defect, especially during crying or straining. It is most common in African-American low birth weight females.
Clinical Manifestations
The size of the defect varies from less than 1 cm in diameter to as much as 5 cm; however, large ones are rare. It appears as a soft swelling covered by skin.

Treatment
The use of binders, tape, or other materials to flatten the protrusion do not aid in closing the defect. Most umbilical hernias disappear spontaneously by 3 to 4 years of age. If the hernia persists beyond this age; if it becomes strangulated; or if it grows larger, it is surgically corrected.

Congenital Diaphragmatic Hernia
Congenital diaphragmatic hernia (CDH) involves herniation of the abdominal contents through a defect in the diaphragm into the chest cavity and usually develops on the left side. All degrees of protrusion of the abdominal viscera through the diaphragmatic opening into the thoracic cavity may occur. The extent of herniation determines the severity and timing of the symptoms. The incidence is 0.2 in 1,000 live births (Hartman, 1996). Separation of the developing thoracic and abdominal cavities is accomplished during the 8th week of gestation by closure of the pleuroperitoneal (opening between the chest and abdomen) canal. CDH occurs when this canal fails to close.

Clinical Manifestations
Newborns will have severe respiratory distress, cyanosis, tachypnea, and retractions at birth because the lung on the side of the defect is usually hypoplastic or underdeveloped. Breath sounds are decreased or absent on the affected side, and the chest is barrel-shaped. Heart sounds are shifted to the right. Bowel sounds may be heard over the chest. The abdomen is scaphoid.

Treatment
Mortality rate is high (40% to 60%) despite advances in current treatment modalities (Hartman, 1996). Some fetuses are diagnosed prenatally by ultrasound in which case surgical repair is performed in utero. If not diagnosed and repaired at this time, the newborn is stabilized before surgery. Ventilatory support is required to manage respiratory compromise. Metabolic acidosis is corrected with the administration of bicarbonate. If stabilization is not possible, extracorporeal membrane oxygenation is required in most cases. The surgery involves repositioning the abdominal contents into the abdomen and closing the defect.

Malrotation and Volvus
Malrotation is the incomplete normal rotation of the midgut during fetal development as it returns from the umbilical pouch to the abdominal cavity. During early gestation the midgut grows extensively and protrudes into the umbilical cord pouch until it lies completely outside the abdominal cavity. Eventually this cavity enlarges and the midgut returns to the intra-abdominal position. Malrotation occurs when the bowel fails to rotate normally as it returns to the abdominal cavity. Volvus, a complication of malrotation, occurs when the incompletely rotated bowel twists on itself, leading to arterial obstruction, ischemia, and necrosis.

Clinical Manifestations
Most infants with this anomaly experience symptoms of bowel obstruction, abdominal distention, and bilious vomiting in the first year of life. Diarrhea may be an early symptom in infants under the age of six months. If volvus occurs, bloody stools may be followed by perforation and peritonitis. Older children may have intermittent abdominal cramping, pain, vomiting, and diarrhea or constipation.

Treatment
Treatment for malrotation is surgical. The intestine is rotated and placed into the abdominal cavity with the cecum in the left lower quadrant. If volvus and bowel necrosis are present, the affected area is removed.

Meckel’s Diverticulum
Meckel’s diverticulum, the most common congenital malformation of the GI tract, is a blind sac or pouch protruding from the wall of the ileum. It results when a duct connecting the embryonic yolk sac to the primitive gut fails to atrophy. It occurs in 2–3% of the population and is usually asymptomatic (Sondheimer & Silverman, 1995).

Clinical Manifestations
Most symptomatic cases appear within the first 2 years of life. Painless rectal bleeding is the most common clinical manifestation. Bleeding occurs because the tip of the pouch of the ileum contains ectopic gastric mucosa rather than ileal mucosa. The gastric mucosa secretes acid and pepsin, causing irritation, ulceration, and eventually lower GI bleeding. Rectal bleeding is massive and dark or bright red in color.

Treatment
Treatment is surgical removal of the diverticulum or pouch to prevent hypovolemic shock from hemorrhage. In most cases, intestinal resection is not required, and the child recovers rapidly.

Short Bowel Syndrome
Short bowel syndrome (SBS) is a disorder characterized by inadequate surface area of the small intestine and usually occurs after surgical resection of the intestine in cases of
necrotizing enterocolitis, volvus, or Crohn’s disease. The small intestine may be congenitally short in conditions such as gastroschisis, omphalocele, and intestinal atresia. SBS may not be a permanent disorder because the intestine can grow and adapt. This process of adaptation is gradual, requiring months to years.

Clinical Manifestations
The most common clinical manifestations are malabsorption, malnutrition, and diarrhea. Carbohydrate malabsorption and steatorrhea also occur. Fluid and electrolyte losses may lead to dehydration, hyponatremia, hypokalemia, and acidosis. Vitamins and minerals are lost and deficiencies occur. Skin irritation and breakdown on the buttocks and perineum are caused by the frequent loose, watery stools. Bacterial overgrowth in the remaining small intestine is common and occurs when the ileocecal valve is absent or when there is impaired motility in the bowel and stasis. This overgrowth leads to increased diarrhea and intestinal gas.

Treatment
Medical management focuses on maintaining optimum nutrition and preventing complications. Nutritional therapy initially includes total parenteral nutrition (TPN) via a central line and enteral feedings via an NG or gastrostomy tube. The main purpose of enteral nutrition is to stimulate the adaptive growth of the small intestine. Oral feedings are given when tolerated so the infant can learn to suck and swallow. Additionally, to maintain an interest in oral feeding and to stimulate sucking a pacifier may be used. When enteral and oral feedings are increased, TPN is gradually decreased proportionately. Several complications may occur as a result of long-term use of TPN, including central catheter infection, occlusion, and thrombosis; liver disease; and cholestasis (interruption in the flow of bile). Therefore, when TPN is initiated and regularly used throughout therapy, certain laboratory values are obtained. These include liver and renal function tests, liver enzymes, calcium, magnesium, and phosphorus.

Key Concepts

- The gastrointestinal system of the infant and child is immature compared to an adult; therefore, feeding must be in smaller amounts, more frequent, and consist of a greater number of calories per kilogram of weight.
- Pyloric stenosis is characterized by projectile vomiting without loss of appetite, poor weight gain, dehydration, and a palpable olive-shaped mass in the epigastrium.
- Clefts of the lip and palate are some of the most common congenital anomalies. Initial reactions of caregivers to an infant with this defect include shock, grief, feelings of failure, inadequacy, and isolation.
- Nursing management for the infant with a cleft palate or lip focuses on adapting feeding methods, providing preoperative and postoperative care, educating caregivers, and providing emotional support.
- The typical presentation of an infant with esophageal atresia includes copious, fine, frothy bubbles of mucus in the mouth and sometimes the nose.
- Intussusception is one of the most common causes of intestinal obstruction in infancy and presents with severe abdominal pain, vomiting, and blood and mucus in stools.
- Hirschsprung’s disease, the most common cause of distal bowel obstruction in the newborn, is treated with surgical removal of the aganglionic portion of the bowel.
- Anorectal malformations are usually noted at birth, and after surgical repair, these infants may have difficulty with toilet training.
- Nursing care for gastroesophageal reflux is directed toward teaching caregivers methods to prevent or reduce reflux by feeding and positioning.
- For the child with chronic constipation nursing management is directed toward cleansing the bowel, diet therapy, and establishing a regular elimination pattern.
- Signs and symptoms of appendicitis include abdominal pain that begins in the periumbilical area and migrates
to the right lower quadrant, low-grade fever, nausea, and sometimes vomiting.

- Prompt and accurate diagnosis of appendicitis is essential to prevent perforation and peritonitis, which are common in children.
- Inflammatory bowel disease includes ulcerative colitis and Crohn’s disease and is characterized by persistent diarrhea, abdominal pain, and growth failure. Treatment focuses on reducing the symptoms with medications, nutritional therapy, and often surgery.
- Nursing management for a child with a peptic ulcer includes teaching caregivers about medication therapy and dietary modifications.

- The nurse has a significant role in early detection of NEC, assessing for signs of complications, and providing emotional support for the family.
- For the child with celiac disease, nursing management focuses on education about the gluten-free diet and referral to community resources for emotional and dietary support.
- For a child with lactose intolerance, treatment and nursing management focuses on educating the child and caregivers about dietary needs.
- Nursing management for the child with hepatitis is directed toward teaching about dietary needs, infection control, and signs and symptoms of severely impaired liver function.

Review Questions

1. Differentiate between hypertrophic pyloric stenosis and gastroesophageal reflux.
2. Outline a teaching plan for caregivers related to feeding an infant born with bilateral CL/CP.
3. List the clinical manifestations of an infant with esophageal atresia and tracheoesophageal fistula.
4. Discuss the non-surgical treatment for intussusception.
5. Explain the pathophysiology of Hirschsprung’s disease.
6. Describe the nurse’s role in detecting an anorectal malformation in a newborn infant.
7. Explain the American Academy of Pediatrics recommended position for the infant with gastroesophageal reflux.
8. Discuss why appendicitis in children frequently progresses to perforation.
9. List the clinical manifestations of appendicitis.
10. Compare ulcerative colitis and Crohn’s disease in the following areas: a) pathologic changes in intestine and (b) clinical manifestations.
11. Outline a plan for teaching the child with celiac disease and the child’s family.
12. Identify strategies to enhance compliance with dietary restrictions for the child with celiac disease.
13. Compare the methods of transmission and clinical manifestations of hepatitis A, B, and C.

References


**Suggested Readings**
