ألجامعة المستنصرية / كلية الطب
ألمرحلة الرابعة / طب الأطفال
ا.م. د. نبيهة النقيب
2025-2026

Feeding Problems During The 1st Year of Life

1.Underfeeding:

is suggested by: Restlessness, crying, and Failure to gain weight adequately.
Result from: failure to take a sufficient quantity of food even when offered.
All the following should be considered
* The frequency of feedings,
* The mechanics of feeding,
* The size of the holes in the nipple of feeding bottle,
* The adequacy of eructation of air,
* The possibility of abnormal mother-infant "bonding.
* Possible systemic disease in the infant

The extent and duration of underfeeding determine the clinical manifestations:
* Constipation, failure to sleep, irritability, excessive crying is to be expected.
* Weight gain may be slow, or there may be an actual loss of weight.

In the latter case:
* Skin becomes dry and wrinkled.
* Subcutaneous tissue disappears.
* The infant assumes the appearance of an "old man.
* Deficiencies of vitamins A, B, C, and D as well as of iron and protein may be responsible for the characteristics of clinical manifestations.

Treatment of underfeeding include:
*Increasing nutrient intake
*Correcting any deficiencies of vitamins and/or minerals
*Instructing the caregiver in the art and practice of infant feeding
*If an underlying systemic disease, child abuse or neglect, or a psychological problem is responsible, specific management of that disorder is necessary.

2.Overfeeding

As a rule, postprandial discomfort from excessive intake limits the amount of food an infant voluntarily ingests, but there are exceptions.
If intake is excessive, regurgitation and vomiting are the most frequent symptoms.

Diets that are too high in fat delay gastric emptying, cause abdominal distention and discomfort, and may cause excessive weight gain. Diets that are too high in carbohydrate are likely to cause undue fermentation in the intestine, resulting in distention and flatulence as well as more rapid weight gain than desirable.

3.Regurigitation and Vomiting

Regurgitation is the effortless movement of stomach contents into the esophagus and mouth. It is not associated with distress, and infants with regurgitation are often hungry immediately after an episode. The lower esophageal sphincter prevents reflux of gastric contents into the esophagus. Regurgitation is a result of gastroesophageal reflux through an incompetent or, in infants, immature lower esophageal sphincter. This is often a developmental process, and regurgitation or "spitting" resolves with maturity.

Regurgitation is a natural occurrence, especially during the 1st several months of life and reduced to a negligible amount by:

* Adequate eructation of swallowed air during and after eating, by gentle handling,
*Avoiding emotional conflicts,
*Placing the infant on the right side for a short time immediately after eating.

Regurgitation should be differentiated from vomiting, which denotes an active reflex process with an extensive differential diagnosis. Regurgitation, the result of gastroesophageal reflux, occurs commonly in the1st yr of life. Effortless regurgitation can dribble out of an infant's mouth but also, may be forceful. In an otherwise healthy infant with regurgitation, volumes of emesis are commonly approximately 15-30 mL but occasionally they are larger. Most often, the infant remains happy, although possibly hungry, after an episode of regurgitation. Episodes can occur from one to several times per day. Regurgitation gradually resolves in 80% of infants by 6 mo of age and in 90% by 12 mo. If complications develop or regurgitation persists, gastroesophageal reflux is considered pathologic rather than merely developmental and deserves further evaluation and treatment.

Complications of gastroesophageal reflux include

1.Failure to thrive,
2.Pulmonary disease (apnea or aspiration pneumonitis),
3.Esophagitis with its sequelae

Regurgitation is a result of gastroesophageal reflux through an incompetent or, in infants, immature lower esophageal sphincter. This is often a developmental process, and regurgitation or "spitting" resolves with maturity. Regurgitation should be differentiated from vomiting, which denotes an active reflex process with an extensive differential diagnosis

Vomiting Define: violent expulsion of gastric and sometimes intestinal contents.

vomiting caused by obstruction of the GIT is probably mediated by intestinal visceral afferent nerves stimulating the vomiting. If obstruction occur below the second part of the duodenum, vomitus is usually bile stained. With repeated vomiting in the absence of obstruction, duodenal content is refluxed in to the stomach and emesis may become bile stained. Vomiting is one of the most common symptoms in infancy and may be associated with a variety of disturbances both trivial and serious. Its causes should always be investigated

Causes of vomiting

GIT

- Chalasia
- Achalasia
- Hiatal hernia
- Peptic esophagitis
- F.B
- Intussusceptions
- Pyloric stenosis
- Gluten enteropathy
- Diaphragmatic hernia
- Food allergy
- Hirsch sprung disease
- Appendicitis
- Gastroenteritis
- Hepatitis
- Volvulus
- Duodenal ulcer
- Malrotation
- Duplication

EXTRA GIT:

- Sepsis
- Pneumonia
- Otitis media
- UTI
- Meningitis
- Brain tumor
- Adrenal insufficiency
- Inborn error

Common causes:

Common IN infants	**Common in child**
GE	Pertussis
Esophageal reflux	GE
Over feeding	Systemic infection
Anatomical obstruction	Toxic ingestion
syndrome	Pertussis
Systemic infection	Medication
Medication	Reflux

Red Flag' clinical features in the vomiting child

1. Bile-stained vomit: Intestinal obstruction
2. Hematemesis: Esophagitis, peptic ulceration, oral/nasal bleeding.
3. Projectile vomiting, in first few weeks of life: Pyloric stenosis
4. Vomiting at the end of paroxysmal coughing: Whooping cough (pertussis)
5. Abdominal tenderness/abdominal pain on movement: Surgical abdomen
6. Abdominal distension: Intestinal obstruction, including strangulated inguinal hernia
7. Hepatosplenomegaly: Chronic liver disease
8. Blood in the stool: Intussusception, gastroenteritis – salmonella or campylobacter
9. Severe dehydration, shock: Severe gastroenteritis, systemic infection (urinary tract infection, meningitis), diabetic ketoacidosis
10. Bulging fontanelle or seizures: Raised intracranial pressure
11. Failure to thrive: Gastroesophageal reflux, coeliac disease and other chronic gastrointestinal conditions

Approach to child with vomiting

Physical examination should include assessment of the child's hydration status, including examination of capillary refill, moistness of mucous membranes, and skin turgor. The chest should be auscultated for evidence of rales or other signs of pulmonary involvement. The abdomen must be examined carefully for distention, organomegaly, bowel sounds, tenderness, and guarding. A rectal examination and testing stool for occult blood should be considered.

Laboratory evaluation of vomiting should include serum electrolytes, tests of renal function, complete blood count, amylase, lipase, and liver function tests. Additional testing may be required immediately when history and examination suggest a specific etiology. Ultrasound is useful to look for pyloric stenosis, gallstones, renal stones, hydronephrosis, biliary obstruction, pancreatitis, malrotation, intussusception, and other anatomical abnormalities. CT may be indicated to observe structures that cannot be visualized well by ultrasound. Barium studies can show obstructive or inflammatory lesions of the gut and can be therapeutic, as in the use of contrast enemas for intussusception.

Complications of Vomiting

COMPLICATION	PATHOPHYSIOLOGY	HISTORY, PHYSICAL EXAMINATION, AND LABORATORY STUDIES
Metabolic	Fluid loss in emesis	Dehydration
	HCl loss in emesis	Alkalosis; hypochloremia
	Na, K loss in emesis	Hyponatremia; hypokalemia
	Acidosis	Dehydration
Nutritional	Emesis of calories and nutrients Anorexia for calories and nutrients	Malnutrition; “failure to thrive”
Mallory-Weiss tear	Retching → tear at lesser curve of gastroesophageal junction	Forceful emesis → hematemesis
Esophagitis	Chronic vomiting → esophageal acid exposure	Heartburn; Hemoccult + stool
Aspiration	Aspiration of vomitus, especially in context of obtundation	Pneumonia; neurologic dysfunction
Shock	Severe fluid loss in emesis or in accompanying diarrhea	Dehydration (accompanying diarrhea can explain acidosis?)
	Severe blood loss in hematemesis	Blood volume depletion
Pneumomediastinum, pneumothorax	Increased intrathoracic pressure	Chest x-ray
Petechiae, retinal hemorrhages	Increased intrathoracic pressure	Normal platelet count

Treatment needs to address the consequences and the causes of the vomiting. Dehydration must be treated with fluid resuscitation. This can be accomplished in most cases with oral fluid-electrolyte solutions, but intravenous (IV) fluids may be required. Electrolyte imbalances should be corrected by appropriate choice of fluids. Underlying causes should be treated when possible.

The use of antiemetic medications is controversial. These drugs should not be prescribed until the etiology of the vomiting is known, and then only for severe symptoms. For reflux, Dopamine antagonist like Metoclopramide 0.1-0.2 mg/kg PO or IV qid.

Anticholinergics (e.g., scopolamine) and antihistamines (e.g., dimenhydrinate) are useful for prophylaxis and treatment of motion sickness. Drugs that block serotonin 5-HT3 receptors, such as ondansetron 0.15-0.3 mg/kg IV or PO tid, are frequently used for viral gastroenteritis and can improve tolerance of oral rehydration therapy. They are helpful for chemotherapy- induced vomiting, often combined with dexamethasone. No antiemetic should be used in patients with surgical emergencies or when a specific treatment of the underlying condition is possible. Correction of dehydration, ketosis, and acidosis is helpful to reduce vomiting in most patients with viral gastroenteritis.

Supportive and Nonpharmacologic Therapies for Vomiting

DISEASE	**THERAPY**
All	Treat cause • Obstruction: operate • Allergy: change diet (±steroids) • Metabolic error: Rx defect • Acid peptic disease: H2-receptor antagonists, proton pump inhibitors
Complications	
Dehydration	IV fluids, electrolytes
Hematemesis	Transfuse, correct coagulopathy
Esophagitis	H2-receptor antagonists, proton pump inhibitors
Malnutrition	NG or NJ drip feeding useful for many chronic condition
Meconium Ileus/ DIOS	Gastrografin enema
Intussusception	Barium enema; air reduction enema
Reflux	Positioning; dietary measures (infants: rice cereal, 1 tbs/oz of formula)

4.LOOSE OR DIARRHEAL STOOLS

Diarrhea is best defined as excessive loss of fluid and electrolyte in the stool. Acute diarrhea is defined as sudden onset of excessively loose stools of >10 mL/kg/day in infants and >200 g/24 hr in older children, which lasts <14 days. When the episode lasts longer than 14 days, it is called persistent diarrhea.

Normally, a young infant has approximately 5 mL/kg/day of stool output; the volume increases to 200 g/24 hr in an adult. The greatest volume of intestinal water is absorbed in the small bowel; the colon concentrates intestinal contents against a high osmotic gradient. The small intestine of an adult can absorb 10-11 L/day of a combination of ingested and secreted fluid, whereas the colon absorbs approximately 0.5 L. Disorders that interfere with absorption in the small bowel tend to

produce voluminous diarrhea, whereas disorders compromising colonic absorption produce lower-volume diarrhea. Dysentery (small-volume, frequent bloody stools with mucus, tenesmus, and urgency) is the predominant symptom of colitis.

The number, color, and consistency of stools can vary greatly in the same infant and between infants of similar age, without apparent explanation. The earliest stools after birth consist of meconium, a dark, viscous material that is normally passed within the 1st 48 hr of life. With the onset of feeding, meconium is replaced by green-brown transition stools, often containing curds, and, after 4-5 days, by yellow-brown milk stools. Stool frequency is extremely variable in normal infants and can vary from none to 7 per day. Breastfed infants can have frequent small, loose stools early (transition stools), and then after 2-3 wk can have very infrequent soft stools. Some nursing infants might not pass any stool for 1-2 wk and then have a normal soft bowel movement. The color of stools has little significance except for the presence of blood or absence of bilirubin products (white gray rather than yellow-brown). The presence of vegetable matter, such as peas or corn, in the stool of an older infant or toddler ingesting solids is normal and suggests poor chewing and not malabsorption. A pattern of intermittent loose stools, known as toddler's diarrhea, occurs commonly between 1 and 3 yr of age. These otherwise healthy growing children often drink excessive carbohydrate-containing beverages. The stools typically occur during the day and not overnight. The volume of fluid intake is often excessive; limiting sugar and unabsorbable carbohydrate-containing beverages and increasing fat in the diet often lead to resolution of the pattern of loose stools.

The stool of the breast-fed infant is naturally softer than that of the formula-fed infant. From about the 4th to the 6th day of life, the stools of the breast-fed infant go through a transitional stage of being loose, greenish-yellow in color and containing mucus to the typical “milk stool. Subsequently, the use of laxatives or the ingestion of certain foods by the mother may be temporarily responsible for a breast-fed infant's loose stools.

Excessive intake of breast milk may also increase the frequency and water content of the stool.

Actual diarrhea from overfeeding, however, is unusual; thus, diarrhea should be considered infectious until proven otherwise. Although the stools of formula-fed infants tend to be firmer than those of breast-fed infants, loose stools also may result from artificial feeding.

Overfeeding may cause loose, frequent stools, particularly during the 1st 2 wk or so of life. Later, formulas that are too concentrated or too high in sugar content, especially in lactose, may result in loose, frequent stools.

Non-digestive Tract Causes of diarrhea in Children

Infection: otitis media, urinary tract infection

Uremia

Medications: antibiotics, cisapride.

Tumors: neuroblastoma

Pericarditis

Adrenal insufficiency

Toddler's diarrhea — (also known as chronic nonspecific diarrhea of childhood) is described as a benign, self-limited condition that commonly affects children between 1 and 3 years of age. Toddler's diarrhea is a functional gastrointestinal disorder characterized by chronic, recurrent loose stools in otherwise healthy, thriving toddlers, with no evidence of infection, malabsorption, or systemic disease.

Clinical Features

- Occurs most commonly between 6 months and 5 years, peaking around 2–3 years of age.
- Stools: 3–6 loose or watery stools per day, often containing undigested food particles.
- No blood, pus, or mucus in the stool.
- Growth and development remain normal.
- No systemic symptoms — the child is otherwise healthy and active.

Pathophysiology

The diarrhea is thought to result from a combination of:

- Excessive intake of fruit juices, especially those containing sorbitol and fructose.
- Low fat intake, which reduces intestinal transit time.
- Increased intestinal motility.
- Sometimes mild carbohydrate malabsorption.

Diagnosis: is clinical, no extensive investigations are required if the presentation is typical.based on:

- Normal growth and physical examination.
- Normal laboratory tests (stool studies, growth parameters).
- Absence of red flags such as blood in stool, weight loss, or systemic illness.

Management

- Reassurance to parents — the condition is self-limited and usually resolves by 4–5 years of age.
- Dietary modification:
    - Reduce fruit juice and other high-sugar beverages.
    - Ensure adequate dietary fat.
    - Increase fiber intake to normalize stool consistency.
- No pharmacologic treatment is needed.

Prognosis

Excellent. Most children outgrow the condition by school age without any long-term consequences.