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Poor weight gain in children younger than two years in resource-abundant settings: Etiology and evaluation

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INTRODUCTION

Poor weight gain, also called "weight faltering," "faltering growth," or "growth deficit," refers to failure to gain weight appropriately; in more severe cases, linear growth and head circumference also may be affected. Although the term "failure to thrive" has been used for poor weight gain, we avoid it because it is ambiguous [1]. Poor weight gain is a sign that describes a particular problem rather than a diagnosis. The underlying cause of poor weight gain is "always insufficient usable nutrition," although a wide variety of medical and psychosocial stressors can contribute ([table 1](#)) [2].

Poor weight gain that results in severe malnutrition can cause persistent short stature, secondary immune deficiency, and permanent damage to the brain and central nervous system [2]. Early identification and expeditious treatment may help to prevent long-term developmental deficits [3]. (See "[Poor weight gain in children younger than two years in resource-abundant settings: Management](#)", section on 'Prognosis'.)

The etiology and initial evaluation of poor weight gain in children younger than two years in resource-abundant settings will be described here. The management of poor weight gain in children in resource-abundant settings and the evaluation and management of poor weight gain and undernutrition in children from resource-limited settings are discussed separately.

- Resource-abundant settings:

- (See ["Poor weight gain in children younger than two years in resource-abundant settings: Management"](#).)
- (See ["Poor weight gain in children older than two years in resource-abundant settings"](#).)
- Resource-limited settings:
 - (See ["Malnutrition in children in resource-limited settings: Clinical assessment"](#).)
 - (See ["Management of moderate acute malnutrition in children in resource-limited settings"](#).)
 - (See ["Management of uncomplicated severe acute malnutrition in children in resource-limited settings"](#).)
 - (See ["Management of complicated severe acute malnutrition in children in resource-limited settings"](#).)

DEFINITIONS

Poor weight gain — A consensus definition for poor weight gain, including duration of concern about growth, is lacking [4-7].

- **Working definition** – We define poor weight gain as:
 - Weight less than the 2nd percentile for gestation-corrected age and sex when plotted on an appropriate growth chart ([figure 1A-B](#)) ([calculator 1](#)) **and**
 - Decreased velocity of weight gain that is disproportionate to growth in length ([figure 2A-B](#)) ([calculator 2](#)) [8]

Weight below the 2nd percentile is approximately equivalent to a Z-score of -2; the Z-score is a value that represents the number of standard deviations from the mean value. (See ["Measurement of growth in children", section on 'Use of Z-scores'](#).)

Weight gain may not be poor among children growing along a curve with a normal interval growth rate, even if their weight is <2nd percentile [9,10]. Weight gain also may not be poor among infants and young children with genetic short stature, constitutional growth delay, prematurity, or intrauterine growth restriction who have appropriate weight-for-length

and normal growth velocity [11,12]. (See "Normal growth patterns in infants and prepubertal children", section on 'Variants of normal'.)

- **Other definitions** – Other indices of poor weight gain that are sometimes used include [2,8,12-16]:
 - Weight below the 2nd percentile for gestation-corrected age and sex on more than one occasion ([calculator 1](#)); special growth charts for prematurity and selected genetic syndromes should be used when indicated (eg, for children with achondroplasia, Turner syndrome, etc). (See "Growth management in preterm infants", section on 'Normative growth data' and "Normal growth patterns in infants and prepubertal children", section on 'Growth charts for special populations'.)
 - Weight <80 percent of ideal weight-for-age, using a standard growth chart ([figure 3](#))
 - Depressed weight-for-length (ie, weight age less than length age, weight-for-length <10th percentile ([figure 2A-B](#)) ([calculator 2](#))).
 - A rate of weight change that causes a decrease of two or more major percentile lines ([figure 1A-B](#)) (90th, 75th, 50th, 25th, 10th, and 5th) over time (eg, from 75th to 25th). (See "Normal growth patterns in infants and prepubertal children", section on 'Determinants of normal growth'.)
 - A rate of daily weight gain less than that expected for age ([table 2](#)).

Severity of malnutrition — Alternative methods of classifying the severity of malnutrition in routine clinical practice include ([table 3](#)) [17]:

- **Weight-for-length Z-score** ([calculator 2](#)) – Mild malnutrition -1 to -1.9, moderate malnutrition -2 to -2.9, severe malnutrition ≤ -3 . This is the main parameter used to classify poor weight gain in resource-abundant settings.
- **Mid-upper arm circumference (MUAC) Z-score** – Mild malnutrition -1 to -1.9, moderate malnutrition -2 to -2.9, severe malnutrition ≤ -3 .
- Decline in weight-for-length Z-score ([calculator 2](#)) – Mild malnutrition ≥ 1 , moderate malnutrition ≥ 2 , severe malnutrition ≥ 3 [17].
- MUAC – Moderate wasting MUAC 115 to 125 mm, severe wasting MUAC <115 mm (for children 6 to 59 months). These flat cutoffs can be used as field measures in place of Z-scores, particularly in low-resource settings [18]. In severe acute malnutrition, MUAC predicts mortality better than weight for length [19].

EPIDEMIOLOGY

Poor weight gain is common. It occurs in approximately 5 to 10 percent of children in primary care settings and 3 to 5 percent of those in the referral setting [20].

Medical risk factors for poor weight gain include prematurity (particularly when associated with intrauterine growth restriction), developmental delay, congenital anomalies (eg, cleft lip and/or palate, genetic or chromosomal disorders), intrauterine exposures (eg, alcohol, anticonvulsants, infection), lead poisoning, anemia, and any medical condition that results in inadequate intake, increased metabolic rate, maldigestion, or malabsorption [2,21-23]. Virtually every organic disease process may contribute to poor weight gain.

Psychosocial risk factors for poor weight gain include poverty, certain health and nutrition beliefs (eg, fear of obesity or cardiovascular disease, prolonged exclusive breastfeeding), social isolation, life stresses, poor parenting skills, disordered feeding techniques, substance abuse or other psychopathology, violence, and abuse [2,23-27].

CAUSES

Poor weight gain is caused by insufficient usable nutrition secondary to inadequate nutrient intake, inadequate nutrient absorption, increased urinary or intestinal losses, increased nutrient requirements, or ineffective metabolic utilization ([table 1](#)) [2]. Medical, nutritional, developmental/behavioral, and psychosocial factors may all contribute [16,28]. (See 'Evaluation' below.)

The majority of cases in primary care practice are secondary to inadequate dietary intake, usually related to psychosocial factors or disturbance in feeding behavior [29]. Insufficient dietary intake is also a common cause of poor weight gain among infants referred to specialty clinics.

EVALUATION

Goals and process — The goal of the evaluation of a child with poor weight gain is to identify the potential contributing factors (medical, nutritional, psychosocial, and developmental/behavioral) that can be addressed in the management [16,30].

Ancillary providers (eg, dietitian, occupational or speech therapist, social worker, developmental and behavioral pediatrician) can facilitate information gathering and formulation of the

management plan. Throughout the evaluation, it is important to work in partnership with and to support the caregivers, who may feel guilty about the child's poor weight gain. (See "[Poor weight gain in children younger than two years in resource-abundant settings: Management](#)", section on 'Adjunctive interventions'.)

The evaluation involves a thorough history and physical examination and basic laboratory tests. Additional laboratory testing and diagnostic imaging are guided by the findings from the initial evaluation.

- Mild malnutrition – The initial evaluation of children with mild malnutrition ([table 3](#)) focuses on the age of onset ([table 4](#)), associated symptoms ([table 5](#)), and possible feeding problems ([table 6](#)) [31]. (See 'Severity of malnutrition' above and 'Age of onset' below and 'Medical history' below and 'Diet and feeding' below.)
- Moderate to severe malnutrition – The initial evaluation of children with moderate to severe malnutrition ([table 3](#)) requires more intensive investigation of medical ([table 5](#) and [table 7](#)), nutritional ([table 6](#)), and social factors. (See 'Severity of malnutrition' above and 'Medical history' below and 'Diet and feeding' below and 'Psychosocial' below.)

Virtually every organic disease process may contribute to poor weight gain. Most organic diseases are suggested by information gleaned from a thorough history, review of systems, and examination. However, some medical conditions that contribute to poor weight gain are clinically subtle; it is important to ask questions to elicit symptoms or risk factors for these conditions ([table 5](#)) and to look for signs of them during the examination ([table 7](#)) [16].

History — Important aspects of the history in a child with poor weight gain include detailed information regarding the age of onset, dietary intake and feeding practices, psychosocial factors, the child's environment, and the child's behavior and development.

Age of onset — Causes of poor weight gain also can be loosely grouped according to the age of onset ([table 4](#)) [9,31-33].

Medical history — Important aspects of the medical history include [34]:

- Pre- and perinatal history – Low birth weight, intrauterine growth restriction, perinatal stress, and prematurity are important predisposing factors to poor weight gain. Prenatal exposures (eg, anticonvulsants, alcohol) may compromise growth and/or be risk factors for impaired caregiver-child interactions [11]. (See "[Risks associated with epilepsy during pregnancy and the postpartum period](#)", section on 'Effects of ASMs on the fetus and child')

and ["Fetal alcohol spectrum disorder: Clinical features and diagnosis"](#), section on 'Clinical features'.)

- Past medical history – Chronic diseases of any type (eg, celiac disease, cystic fibrosis, giardiasis) may affect nutritional intake, absorption, or needs; frequent recurrent illnesses may indicate immunodeficiency; frequent injuries may indicate inadequate supervision. Report of problems in ≥ 5 organ systems, ≥ 5 food allergies, and absence of serious congenital anomaly or confirmed genetic disorder suggest the possibility of medical child abuse [35]. (See ["Approach to the child with recurrent infections"](#) and ["Child neglect: Evaluation and management"](#), section on 'Evaluation'.)
- Family history – Family history should include the height and weight of parents and siblings and history of developmental delay, constitutional delay of growth and puberty, and any illnesses that may contribute to slow growth. Shorter parental height and higher parity may be associated with slow weight gain in infants [36]. (See ["Causes of short stature"](#).)
- Review of systems – The review of systems should include symptoms of medical conditions that could contribute to poor weight gain ([table 5](#)).

Diet and feeding — Detailed information regarding nutritional intake and feeding should be obtained, including the duration of mealtimes, the type of food, the amount of food consumed, and feeding behaviors (particularly picky eating) ([table 6](#)) [31,37-39]. Feeding questionnaires may provide meaningful information about feeding difficulties in children [40,41]. The dietary history should be interpreted with caution, particularly if psychosocial problems are suspected, because caregiver guilt may result in inaccuracies in reporting. (See ["Dietary history and recommended dietary intake in children"](#), section on 'Dietary history'.)

Specific areas of inquiry include [31,42,43]:

- Vomiting, diarrhea, and rumination (ie, chewing without swallowing), which increase nutrient losses
- Feeding problems including picky eating, food refusal, inappropriate self-feeding for age, anorexia, and caregiver responses to the child's eating patterns
- Structural abnormalities of the face, oral cavity, or aerodigestive system, neuromuscular dysfunction (see ["Neonatal oral feeding difficulties due to sucking and swallowing disorders"](#) and ["Aspiration due to swallowing dysfunction in children"](#), section on 'Evaluation')

- Food preferences – Avoidance of foods with certain textures may indicate underlying oral motor dysfunction or autism spectrum disorder (ASD); otherwise unexplained avoidance of specific foods may indicate food intolerance or allergy for which the young child cannot verbalize symptoms (eg, nausea, abdominal discomfort) or ASD [38] (see "[Clinical manifestations of food allergy: An overview](#)", section on 'Anaphylaxis' and "[Autism spectrum disorder in children and adolescents: Clinical features](#)", section on 'Atypical responses to sensory stimuli')
- Excessive consumption of artificially sweetened beverages or fruit juice, which often causes functional diarrhea and may result in loss of nutrients due to fructose and/or [sorbitol](#) malabsorption [10]
- Deficient protein or vitamin intake in some vegetarian diets (see "[Vegetarian diets for children](#)")
- Dietary restrictions related to perceived food allergies or dietary beliefs and practices (eg, fear of cardiovascular disease, vegetarianism)

In children with moderate to severe malnutrition quantitative assessment of intake (eg, 24-hour food recall, three-day food record) may be helpful. (See '[Severity of malnutrition](#)' above and "[Dietary history and recommended dietary intake in children](#)", section on 'Dietary diary'.)

Psychosocial — The psychosocial history is critical in the evaluation of children with poor weight gain; poor weight gain can be an indicator of serious social or psychological problems in the family [34]. Psychosocial stressors are the predominant cause of insufficient intake in children of all ages [44]. The material and emotional resources of the caregiver(s) may not be available for the care of the child for a variety of reasons (eg, poverty, depression, substance abuse, family discord, maladaptive parenting style, etc) [16,45-48]. Many caregivers of children with poor weight gain lack self-esteem; the health care provider should work to identify the strengths of the family that will encourage development of a nurturing environment, rather than focus blame.

The psychosocial history should include an assessment of the child's caregivers and family/household composition, employment status, financial state, degree of social isolation (eg, absence of a telephone or of nearby neighbors), and family stress. If there are multiple caregivers, it is important to determine whether they have similar or disparate views of the growing and eating problem [16]. The psychosocial history should also explore how the child's poor weight gain affects the caregivers and family (eg, guilt, stress, family conflict).

The psychosocial history should specifically address:

- **Poverty and food insecurity** – In the United States, assessment of poverty may include eligibility for the Supplemental Food Program for Women, Infants, and Children (WIC). An attempt should be made to determine if adequate food is available in the home [16,49].
 - Asking the single question, "In the past month, was there any day when you or anyone in your family went hungry because you did not have enough money for food?" appears to be a reliable and accurate method of identifying food insecurity in families with limited resources [50].
 - Similarly, endorsement of one or both of the following statements as often or sometimes true rather than never true accurately identifies households at risk for food insecurity [49,51-53]:
 - "Within the past 12 months we worried whether our food would run out before we got money to buy more."
 - "Within the past 12 months the food we bought just didn't last, and we didn't have money to get more."

(See ["Screening tests in children and adolescents"](#), section on 'Screening for poverty'.)

- **Parenting skills and nutrition knowledge** – The feeding history described above typically provides some information about the caregivers' nutrition knowledge. If this raises concern, a more detailed assessment and/or referral to a dietitian is warranted. (See ["Introducing solid foods and vitamin and mineral supplementation during infancy"](#), section on 'What to feed and how to advance' and ["Dietary recommendations for toddlers and preschool and school-age children"](#), section on 'Dietary composition'.)
- **Psychosocial stressors and access to resources** – Evaluation of psychosocial stressors may include questions about the frequency of changes of home address, changes to household composition, and whether the caregiver feels safe at home. Caregivers initially may avoid mentioning psychosocial problems such as marital discord or spousal abuse; discussions of such issues should take place over the course of several visits and are crucial to addressing growth holistically [54]. These conversations should be conducted in a nonthreatening manner, demonstrating concern and compassion. (See ["Intimate partner violence: Childhood exposure"](#), section on 'The process of asking about caregiver intimate partner violence'.)

In addition to identifying psychosocial stressors, it is important to identify the family's strengths and access to resources that can be used in formulating a management plan

[11]. The involvement of a social worker can be helpful in this regard.

- **Maternal factors** – Maternal factors relating to the pregnancy and postnatal period may be significant. They include whether the pregnancy was planned or unplanned, maternal age, use of medications for illness, alcohol or substance use, physical or mental illness (including history of feeding/eating disorder), postpartum depression, or inadequate breast milk [48,55]. The mother also should be questioned about possible abuse as a child or an eating disorder [38,56].

Mothers who are under stress may use breastfeeding to comfort themselves as well as their infants [57]. In infants who are older than six months of age, this may result in more frequent breastfeeding, refusal of complementary foods, and poor weight gain.

- **Child neglect** – Child neglect is responsible for poor weight gain in a minority of children [30,58-60] but should be considered in children if there is any history of [61]:
 - "Intentional withholding of food from the child;
 - Strong beliefs in health and/or nutrition regimes that jeopardize a child's well-being; and/or
 - Family that is resistant to recommended interventions despite a multidisciplinary team approach."

In such cases, as well as in those where there is evidence of associated physical or emotional abuse, a report should be made to Child Protective Services. (See "[Child neglect: Evaluation and management](#)", section on 'Notification of child protective services'.)

Examination — The goal of the physical examination of a child with poor weight gain is to identify signs of genetic disorders or medical conditions contributing to undernutrition, malnutrition (eg, vitamin deficiencies), and child abuse or neglect.

General examination — Important aspects of the examination are listed in the table ([table 7](#)) [8,11,12,34,62].

Measurement of growth — Accurate measurement of the child's weight, recumbent length ([figure 4](#)), and head circumference is essential. In the child younger than two years, the recumbent length, rather than the standing height, should be obtained. These measures, along with the child's weight-for-length, should be plotted on a standardized growth chart and monitored with repeated measures over time. (See "[The pediatric physical examination: General principles and standard measurements](#)", section on 'Growth parameters' and "[Measurement of growth in children](#)".)

We generally use the [World Health Organization \(WHO\) growth standards](#) to assess the growth of children younger than two years, as recommended by the Centers for Disease Control and Prevention [63]:

- Males – Weight-for-age, length-for-age, head circumference-for-age, and weight-for-length ([figure 1A, 2A, 2C-D](#))
- Females – Weight-for-age, length-for-age, head circumference-for-age, and weight-for-length ([figure 1B, 2B, 2E-F](#))

The WHO growth standards are appropriate for children of all races and ethnicities. Specific genetic disorders (eg, achondroplasia, Williams-Beuren syndrome) may have individualized growth charts. (See "[Achondroplasia](#)" and "[Normal growth patterns in infants and prepubertal children](#)", section on 'Growth charts for special populations'.)

Differences between the WHO growth standards and the [Centers for Disease Control and Prevention/National Center for Health Statistics \(CDC/NCHS\) growth charts](#) are discussed separately. (See "[Measurement of growth in children](#)", section on 'Growth references and standards'.)

- **Correcting for prematurity** – When assessing the growth of former premature infants, it is important to correct growth parameters for gestational age (by subtracting the number of weeks the child was premature from the child's postnatal age at the time of evaluation) [64]. However, there is no consensus about how long to continue such correction. Studies that provide definitive guidance are lacking [65]. The rate and duration of "catch-up growth" may vary depending upon gestational age, birth weight, race/ethnicity, and other factors.

We generally follow the traditional approach of correcting for weight through 24 months of age, for stature through 40 months of age, and for head circumference through 18 months of age [16]. However, the [2009 United Kingdom-WHO growth charts](#) suggest correction of all three parameters until age two years for children born before 32 weeks' gestation and at least until age 12 months for children born between 32 and 36 weeks' gestation [66]. (See "[Growth management in preterm infants](#)", section on 'Monitoring of growth'.)

Growth trajectory and proportionality — If possible, the growth trajectory should be plotted from birth. The timing of changes in the slopes of the weight, length, or head circumference trajectories is particularly important in identifying potential contributors to poor weight gain

(eg, initiation of complementary foods, onset of diarrhea, psychosocial stressor) [11]. (See ['Age of onset'](#) above.)

Proportionality is assessed by determining the weight-for-length percentile ([calculator 2](#)) ([figure 2A-B](#)) or the median age for the child's weight (weight age), length (length age), and head circumference (head circumference age).

Growth trajectory and proportionality may provide clues to the etiology of diminished weight:

- Wasting – Decreased weight in proportion to length ("wasting") reflects inadequate nutritional intake.

Normal growth parameters at birth with subsequent deceleration in weight, followed (weeks to months later) by deceleration in stature (referred to as "stunting") and finally deceleration in head circumference, is characteristic of inadequate nutritional intake ([figure 5](#)). As stunting develops, the weight-for-length may return toward normal [31].

- Stunting – Decreased length with a proportionate weight may be nutritional (if long-standing), genetic, or endocrine in origin [16,31]. Information from the family history, growth trajectory, and calculation of the mid-parental height may help to distinguish between these possibilities. (See ["Causes of short stature"](#) and ["Diagnostic approach to children and adolescents with short stature"](#).)

Normal growth parameters at birth with simultaneous deceleration in length and weight before two years of age and normal growth velocity after two years of age is suggestive of genetic short stature or constitutional growth delay. These normal growth patterns are often confused with poor weight gain. (See ["Normal growth patterns in infants and prepubertal children"](#), section on ["Variants of normal"](#) and ["Causes of short stature"](#) and ["Diagnostic approach to children and adolescents with short stature"](#).)

- Decreased head circumference – When head circumference is impaired as much as, or more than, weight or length, intrauterine infection, teratogenic exposures, congenital syndromes, and other causes of microcephaly should be considered. (See ["Microcephaly in infants and children: Etiology and evaluation"](#), section on ["Etiology"](#) and ["Overview of TORCH infections"](#), section on ["Clinical features of TORCH infections"](#).)

Deceleration of head circumference before deceleration in weight or length is suggestive of a neurologic disorder (eg, neonatal encephalopathy) [31]. (See ["Microcephaly in infants and children: Etiology and evaluation"](#), section on ["Monitoring head growth"](#).)

The growth trajectories of preterm and small for gestational age (SGA) infants are discussed separately. Premature and SGA infants have increased risk for subsequent undernutrition, but many infants with these conditions have catch-up growth and subsequent normal growth velocity. (See "[Growth management in preterm infants](#)" and "[Fetal growth restriction \(FGR\) and small for gestational age \(SGA\) newborns](#)", section on 'Catch-up growth'.)

Caregiver-child interaction — During the examination, the clinician can observe the warmth, caring, and responsiveness of the caregiver to the child's cues, as well as the extent to which the child looks to the caregiver for comfort and support [11].

Observation of the child being fed when they are hungry may disclose a variety of factors contributing to insufficient intake [16,30,67]. Observation also may provide opportunities to provide positive reinforcement to the caregivers (eg, by pointing out how the child looks to the caregiver for comfort and support or how the caregiver appropriately responds to the child's cues). Such observation may occur in the office, hospital, or by an experienced clinician visiting the child's home environment. Another option is to ask the child's primary caregiver to videotape the child eating a meal; the tape can then be reviewed with the family, with specific advice offered based upon the behaviors noted on the tape [23].

Important aspects of the feeding observation include [12,16,67]:

- Is the child adaptively positioned to eat (eg, in a high chair)?
- For bottle-fed infants, does the caregiver cuddle the infant or merely "prop" the bottle?
- Are the child's cues of hunger and satiety clear? Does the caregiver respond appropriately?
- Is there sufficient time for feeding?
- Does the child have oral motor or swallowing difficulties (eg, does the child have difficulty with foods of certain textures? Is feeding prolonged?)
- Does the caregiver permit age-appropriate autonomy and messiness?
- What is the tone of the feeding interaction for the child? For the caregiver (eg, pleasant? relaxed? stressful? hurried?)
- Is the child easily distracted during feeding?
- Is the child fed while they are watching television or the television is on in the background?
- Is the caregiver irritable, punitive, depressed, disengaged, or intrusive?

- Is the child apathetic, irritable, noncompliant, or provocative?

Development and behavior — Children with poor weight gain should undergo formal developmental and behavioral testing for a number of reasons [16]:

- Developmental and behavioral problems may contribute to undernutrition (eg, it can be difficult to feed a child with "difficult temperament").
- Undernutrition may contribute to developmental and behavioral problems (eg, malnutrition may cause irritability). In addition, children with poor weight gain are at increased risk for developmental and behavioral problems [2]. Insufficient intake of nutrients during the vulnerable periods of most rapid development may have a persistent effect on the nervous system. Poor weight gain also appears to heighten developmental vulnerability to other adverse psychosocial factors that contribute to undernutrition (eg, poverty, maternal depression). The earlier these problems are identified, the sooner they can be addressed. (See "[Developmental-behavioral surveillance and screening in primary care](#)", [section on 'Benefits of surveillance and screening'](#).)
- Subtle neurologic deficits may interfere with the normal progression of feeding skills, limiting with the child's ability to consume adequate nutrients (eg, fine motor deficits may affect the ability to self-feed) [2]. The feeding skills of a child with such deficits may be consistent with their developmental, rather than chronologic, age.
- Evaluation of the child's behavior can provide clues to an underlying problem. Children who fail to interact appropriately with their environment and caregivers (eg, avoidance of eye contact, absence of smiling or vocalization, lack of interest in the environment, or lack of response to cuddling) may have developmental delay, hearing or vision impairment, ASD, or psychosocial or environmental deprivation. Behavioral rigidity or sensory aversions may result in self-imposed feeding restrictions. (See "[Autism spectrum disorder in children and adolescents: Clinical features](#)", [section on 'Terminology'](#).)

Diagnostic evaluation — Laboratory tests and imaging studies are unlikely to lead to the diagnosis of an underlying medical disorder in the absence of findings from the medical history or physical examination [24,30,68,69]. However, selective screening studies may provide reassurance that certain conditions are unlikely to be present.

Initial tests — The initial (baseline) tests in the children <2 years with poor weight gain may include:

- Complete blood count, C-reactive protein, and erythrocyte sedimentation rate as a screen for anemia, chronic infection, inflammation, and malignancy.
- Urinalysis and culture as a screen for protein or carbohydrate loss (eg, glucosuria in type 1 diabetes mellitus) and indolent kidney disease, such as chronic urinary tract infection or renal tubular acidosis.

These routine tests may identify consequences of malnutrition (eg, anemia) or medical conditions that may contribute to undernutrition (eg, infection, kidney disease).

Additional tests — Additional laboratory tests or imaging studies may be necessary in the initial evaluation if the history and/or examination suggest particular conditions. For example:

- Electrolytes, blood urea nitrogen, creatinine, glucose, calcium, phosphorus, magnesium, albumin, prealbumin, total protein, liver enzymes, amylase, lipase to evaluate kidney, liver, or pancreatic disease; these tests also provide an indication of nutritional status. (See ["Laboratory and radiologic evaluation of nutritional status in children", section on 'Undernourished children'.](#))
- Stool studies, including guaiac, leukocytes, routine culture, ova and parasite smears, and/or *Giardia* antigen to evaluate gastrointestinal infection. (See ["Giardiasis: Epidemiology, clinical manifestations, and diagnosis"](#).)
- Chest radiograph to evaluate cardiac and pulmonary disease.

Advanced tests — Specialized testing may be warranted if certain diagnoses are suspected or if an etiology has not been found and the child has not responded to dietary or behavior modification. Specialized testing varies with the suspected disorder; examples include:

- Food allergy – Serum immunoglobulin (Ig) E, radioallergosorbent tests, and skin tests to selected food antigens (see ["Clinical manifestations of food allergy: An overview"](#) and ["Diagnostic evaluation of IgE-mediated food allergy"](#))
- Gastrointestinal disease
 - Celiac disease – Tissue transglutaminase and serum IgA as an initial screen, with follow-up testing as indicated (see ["Diagnosis of celiac disease in children", section on 'Tissue transglutaminase antibodies'](#))
 - Malabsorption (see ["Approach to the adult patient with suspected malabsorption"](#))
 - Carbohydrates – Stool-reducing substances

- Protein – Stool alpha-1 antitrypsin
- Fat malabsorption or pancreatic insufficiency – Stool elastase
- *Helicobacter pylori* – Stool antigen assay or 13C-urea breath test and possibly endoscopy (see "[Helicobacter pylori: Diagnosis and management in the pediatric patient](#)", section on 'Evaluation')
- Inflammatory bowel disease:
 - Stool studies including guaiac, leukocytes, and calprotectin (see "[Clinical presentation and diagnosis of inflammatory bowel disease in children](#)", section on 'Stool tests')
 - Upper gastrointestinal series with small bowel follow through and advanced endoscopic studies (see "[Clinical presentation and diagnosis of inflammatory bowel disease in children](#)")
- Gastroesophageal reflux disease – Esophageal pH and impedance monitoring, advance endoscopic studies (see "[Gastroesophageal reflux in infants](#)", section on 'Diagnostic tests' and "[Clinical manifestations and diagnosis of gastroesophageal reflux disease in children and adolescents](#)", section on 'Diagnostic approach')
- Swallowing dysfunction or aspiration – Swallowing function study
- Vomiting or anorexia related to an intra-abdominal or intracranial process – Abdominal ultrasound, radionuclide scans for gastric and biliary tract emptying, and abdominal and head computed tomography scans
- Cystic fibrosis – Sweat chloride test (see "[Cystic fibrosis: Clinical manifestations and diagnosis](#)")
- Inborn errors of metabolism or genetic syndromes – Testing for inborn errors of metabolism, storage diseases, or chromosomal abnormalities (eg, serum amino acids, urine organic acids, urine reducing substances, serum carnitine profile, chromosomes) (see "[Metabolic emergencies in suspected inborn errors of metabolism: Presentation, evaluation, and management](#)")
- Endocrine disorders:
 - Hyperthyroidism – Serum thyroid-stimulating hormone, free thyroxine, and triiodothyronine (see "[Clinical manifestations and diagnosis of Graves disease in children and adolescents](#)")

- Growth hormone deficiency – Insulin-like growth factor 1 and insulin-like growth factor binding protein-3 (see ["Diagnosis of growth hormone deficiency in children"](#))
- Infectious diseases:
 - HIV test if HIV infection is suspected (see ["Diagnostic testing for HIV infection in infants and children younger than 18 months"](#) and ["Screening and diagnostic testing for HIV infection in adults"](#), section on 'Testing algorithm')
 - Tuberculin skin testing if tuberculosis is suspected (see ["Tuberculosis infection \(latent tuberculosis\) in children"](#) and ["Tuberculosis disease in children: Epidemiology, clinical manifestations, and diagnosis"](#))
 - Cytomegalovirus and Epstein-Barr virus IgM and IgG (see ["Overview of cytomegalovirus infections in children"](#) and ["Clinical manifestations and treatment of Epstein-Barr virus infection"](#))
 - Hepatitis (A, B, C) panel if hepatitis is suspected (see ["Overview of hepatitis A virus infection in children"](#) and ["Clinical manifestations and diagnosis of hepatitis B virus infection in children and adolescents"](#) and ["Hepatitis C virus infection in children"](#))

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See ["Society guideline links: Poor weight gain in infants and children"](#).)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or email these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient education" and the keyword[s] of interest.)

- Basics topic (see "[Patient education: Slow weight gain in babies and children \(The Basics\)](#)")
- Beyond the Basics topic (see "[Patient education: Slow weight gain in infants and children \(Beyond the Basics\)](#)")

SUMMARY AND RECOMMENDATIONS

- **Definitions**

- Poor weight gain – A practical definition for poor weight gain is weight less than the 2nd percentile for gestation-corrected age and sex when plotted on an appropriate growth chart ([figure 1A-B](#)) ([calculator 1](#)) **and** decreased velocity of weight gain that is disproportionate to growth in length ([figure 2A-B](#)) ([calculator 2](#)). However, a consensus definition is lacking. (See '[Definitions](#)' above.)

Infants and young children with genetic short stature, constitutional growth delay, prematurity, or intrauterine growth restriction who have appropriate weight-for-length and normal growth velocity may not have poor weight gain. (See '[Definitions](#)' above.)

- Severity of malnutrition – Generally classified by weight-for-length or mid-upper arm circumference (MUAC) Z-scores ([table 3](#)). (See '[Severity of malnutrition](#)' above.)
- **Causes** – The underlying cause of poor weight gain is insufficient usable nutrition, related to inadequate intake, inadequate absorption, increased urinary or intestinal losses, increased requirements, or ineffective utilization ([table 1](#)). (See '[Causes](#)' above.)
- **Goal of evaluation** – The goal of the evaluation of a child with poor weight gain is to identify the potential contributing factors (medical, nutritional, psychosocial, and developmental/behavioral). Ancillary providers (eg, dietitian, occupational or speech therapist, social worker, developmental and behavioral pediatrician) can facilitate information gathering. (See '[Goals and process](#)' above.)
- **History** – Important aspects of the history include the age of onset ([table 4](#)), symptoms of clinically subtle medical conditions that contribute to poor weight gain ([table 5](#)), dietary intake and feeding practices ([table 6](#)), and psychosocial factors. (See '[History](#)' above.)

- **Physical examination** – Important aspects of the physical examination include assessment for genetic disorders or medical conditions to undernutrition, malnutrition (eg, vitamin deficiencies), and child abuse or neglect ([table 7](#)); observation of caregiver-child interaction; and evaluation of behavior and development. (See 'Examination' above.)

Assessment of the growth trajectory and proportionality may provide clues the etiology of diminished weight (see 'Growth trajectory and proportionality' above):

- Normal growth parameters at birth with subsequent deceleration in weight, followed (weeks to months later) by deceleration in stature (referred to as "stunting") and finally deceleration in head circumference, is characteristic of inadequate nutritional intake.
 - Decreased length with a proportionate weight may be related to nutritional (if long-standing), genetic, or endocrine factors. The family history, growth trajectory, and calculation of the mid-parental height may help to distinguish between these possibilities.
 - When head circumference is affected as much or more than weight or length, intrauterine infection, teratogenic exposures, congenital syndromes, and other causes of microcephaly are more likely than inadequate intake.
- **Laboratory tests and imaging studies** – Laboratory tests and imaging studies are unlikely to lead to the diagnosis of an underlying medical disorder in the absence of findings from the medical history or physical examination. Our approach to the diagnostic evaluation is summarized in the tables ([table 8A-B](#)). (See 'Diagnostic evaluation' above.)

ACKNOWLEDGMENT

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REFERENCES

1. Huh SY, Duggan CP. Case 31-2017: A 19-Month-Old Girl with Failure to Thrive. *N Engl J Med* 2018; 378:685.
2. Perrin E, Frank D, Cole C, et al. Criteria for Determining Disability in Infants and Children: Failure to Thrive. Evidence Report/Technology Assessment No. 72. AHRQ Publication NO. 03-E

026. Agency for Healthcare Research and Quality, Rockville, MD, March 2003.
3. Corbett SS, Drewett RF. To what extent is failure to thrive in infancy associated with poorer cognitive development? A review and meta-analysis. *J Child Psychol Psychiatry* 2004; 45:641.
 4. Peterson KE, Chen LC. Defining undernutrition for public health purposes in the United States. *J Nutr* 1990; 120:933.
 5. Olsen EM, Petersen J, Skovgaard AM, et al. Failure to thrive: the prevalence and concurrence of anthropometric criteria in a general infant population. *Arch Dis Child* 2007; 92:109.
 6. Spencer NJ. Failure to think about failure to thrive. *Arch Dis Child* 2007; 92:95.
 7. Hughes I. Confusing terminology attempts to define the undefinable. *Arch Dis Child* 2007; 92:97.
 8. Casey PH. Failure to thrive. In: *Developmental-Behavioral Pediatrics*, 4th ed, Carey WB, Crocker AC, Coleman WL, et al (Eds), Saunders Elsevier, Philadelphia 2009. p.583.
 9. Emond A, Drewett R, Blair P, Emmett P. Postnatal factors associated with failure to thrive in term infants in the Avon Longitudinal Study of Parents and Children. *Arch Dis Child* 2007; 92:115.
 10. Maggioni A, Lifshitz F. Nutritional management of failure to thrive. *Pediatr Clin North Am* 1995; 42:791.
 11. Bithoney WG, Dubowitz H, Egan H. Failure to thrive/growth deficiency. *Pediatr Rev* 1992; 13:453.
 12. Zenel JA Jr. Failure to thrive: a general pediatrician's perspective. *Pediatr Rev* 1997; 18:371.
 13. Wright JA, Ashenburg CA, Whitaker RC. Comparison of methods to categorize undernutrition in children. *J Pediatr* 1994; 124:944.
 14. Wright CM. Identification and management of failure to thrive: a community perspective. *Arch Dis Child* 2000; 82:5.
 15. Edwards AG, Halse PC, Parkin JM, Waterston AJ. Recognising failure to thrive in early childhood. *Arch Dis Child* 1990; 65:1263.
 16. Kistin CJ, Frank DA. Failure to thrive. In: *Zuckerman Parker Handbook of Developmental and Behavioral Pediatrics for Primary Care*, 4th ed, Augustyn M, Zuckerman B (Eds), Wolters Kluwer, Philadelphia 2019. p.256.
 17. Becker P, Carney LN, Corkins MR, et al. Consensus statement of the Academy of Nutrition and Dietetics/American Society for Parenteral and Enteral Nutrition: indicators recommended for the identification and documentation of pediatric malnutrition (undernutrition). *Nutr Clin Pract* 2015; 30:147.

18. World Health Organization: Note on terminology of "wasting" and "acute malnutrition" (February 2023). Available at: https://fscluster.org/sites/default/files/documents/note_on_terminology_acute_malnutrition_and_wasting_who_feb_2023.pdf (Accessed on March 28, 2024).
19. Chiabi A, Mbanga C, Mah E, et al. Weight-for-Height Z Score and Mid-Upper Arm Circumference as Predictors of Mortality in Children with Severe Acute Malnutrition. *J Trop Pediatr* 2017; 63:260.
20. Schwartz ID. Failure to thrive: an old nemesis in the new millennium. *Pediatr Rev* 2000; 21:257.
21. Krugman SD, Dubowitz H. Failure to thrive. *Am Fam Physician* 2003; 68:879.
22. Bithoney WG. Elevated lead levels in children with nonorganic failure to thrive. *Pediatrics* 1986; 78:891.
23. Markowitz R, Duggan C. Failure to thrive: Malnutrition in the pediatric outpatient setting. In: *Nutrition in Pediatrics Basic Science and Clinical Applications*, 3rd, Walker WA, Watkins JB, Duggan C (Eds), BC Decker Inc, Hamilton, Ontario 2003. p.897.
24. Sills RH. Failure to thrive. The role of clinical and laboratory evaluation. *Am J Dis Child* 1978; 132:967.
25. Frank DA, Zeisel SH. Failure to thrive. *Pediatr Clin North Am* 1988; 35:1187.
26. Pugliese MT, Weyman-Daum M, Moses N, Lifshitz F. Parental health beliefs as a cause of nonorganic failure to thrive. *Pediatrics* 1987; 80:175.
27. Weston JA, Stage AF, Hathaway P, et al. Prolonged breast-feeding and nonorganic failure to thrive. *Am J Dis Child* 1987; 141:242.
28. Goday PS, Huh SY, Silverman A, et al. Pediatric Feeding Disorder: Consensus Definition and Conceptual Framework. *J Pediatr Gastroenterol Nutr* 2019; 68:124.
29. Bithoney WG, Newberger EH. Child and family attributes of failure-to-thrive. *J Dev Behav Pediatr* 1987; 8:32.
30. National Institute for Health and Care Excellence. Faltering growth: Recognition and management of faltering growth in children. NICE guideline (NG75). September 2017. Available at: <https://www.nice.org.uk/guidance/ng75> (Accessed on October 02, 2017).
31. Frank D, Silva M, Needlman R. Failure to thrive: Mystery, myth and method. *Contemp Pediatr* 1993; 10:114.
32. McDougall P, Drewett RF, Hungin AP, Wright CM. The detection of early weight faltering at the 6-8-week check and its association with family factors, feeding and behavioural development. *Arch Dis Child* 2009; 94:549.

33. Olsen EM, Skovgaard AM, Weile B, et al. Risk factors for weight faltering in infancy according to age at onset. *Paediatr Perinat Epidemiol* 2010; 24:370.
34. American Academy of Pediatrics Committee on Nutrition. Malnutrition/Undernutrition/Failure to thrive. In: *Pediatric Nutrition*, 8th ed, Kleinman RE, Greer FR (Eds), American Academy of Pediatrics, Itasca, IL 2019. p.781.
35. Mash C, Frazier T, Nowacki A, et al. Development of a risk-stratification tool for medical child abuse in failure to thrive. *Pediatrics* 2011; 128:e1467.
36. Blair PS, Drewett RF, Emmett PM, et al. Family, socioeconomic and prenatal factors associated with failure to thrive in the Avon Longitudinal Study of Parents and Children (ALSPAC). *Int J Epidemiol* 2004; 33:839.
37. Cardona Cano S, Hoek HW, Bryant-Waugh R. Picky eating: the current state of research. *Curr Opin Psychiatry* 2015; 28:448.
38. Ashenburg CA. Failure to thrive: Newer concepts in treatment (Twenty-eighth Ross Roundtable Report). In: *Pediatric Nutritional Challenges From Undernutrition to Overnutrition*, Ambulatory Pediatric Association (Ed), Abbott Laboratories, Columbus, OH 1997. p.14.
39. Parkinson KN, Wright CM, Drewett RF. Mealtime energy intake and feeding behaviour in children who fail to thrive: a population-based case-control study. *J Child Psychol Psychiatry* 2004; 45:1030.
40. Sanchez K, Spittle AJ, Allinson L, Morgan A. Parent questionnaires measuring feeding disorders in preschool children: a systematic review. *Dev Med Child Neurol* 2015; 57:798.
41. Jaafar NH, Othman A, Majid NA, et al. Parent-report instruments for assessing feeding difficulties in children with neurological impairments: a systematic review. *Dev Med Child Neurol* 2019; 61:135.
42. Levy Y, Levy A, Zangen T, et al. Diagnostic clues for identification of nonorganic vs organic causes of food refusal and poor feeding. *J Pediatr Gastroenterol Nutr* 2009; 48:355.
43. Alvares M, Kao L, Mittal V, et al. Misdiagnosed food allergy resulting in severe malnutrition in an infant. *Pediatrics* 2013; 132:e229.
44. Goldbloom RB. Growth failure in infancy. *Pediatr Rev* 1987; 9:57.
45. Robb AS. Eating disorders in children. Diagnosis and age-specific treatment. *Psychiatr Clin North Am* 2001; 24:259.
46. Chatoor I, Ganiban J, Hirsch R, et al. Maternal characteristics and toddler temperament in infantile anorexia. *J Am Acad Child Adolesc Psychiatry* 2000; 39:743.
47. Black MM, Cureton PL, Berenson-Howard J. Behavior problems in feeding: individual, family, and cultural influences. In: *Failure to thrive and pediatric undernutrition: a transdisciplinary*

- ry approach, Kessler DB, Dawson P (Eds), Paul H. Brooks, Baltimore 1999. p.151.
48. Wojcicki JM, Holbrook K, Lustig RH, et al. Chronic maternal depression is associated with reduced weight gain in latino infants from birth to 2 years of age. *PLoS One* 2011; 6:e16737.
 49. COUNCIL ON COMMUNITY PEDIATRICS, COMMITTEE ON NUTRITION. Promoting Food Security for All Children. *Pediatrics* 2015; 136:e1431.
 50. Kleinman RE, Murphy JM, Wieneke KM, et al. Use of a single-question screening tool to detect hunger in families attending a neighborhood health center. *Ambul Pediatr* 2007; 7:278.
 51. Hager ER, Quigg AM, Black MM, et al. Development and validity of a 2-item screen to identify families at risk for food insecurity. *Pediatrics* 2010; 126:e26.
 52. Makelarski JA, Abramssohn E, Benjamin JH, et al. Diagnostic Accuracy of Two Food Insecurity Screeners Recommended for Use in Health Care Settings. *Am J Public Health* 2017; 107:1812.
 53. Baer TE, Scherer EA, Fleegler EW, Hassan A. Food Insecurity and the Burden of Health-Related Social Problems in an Urban Youth Population. *J Adolesc Health* 2015; 57:601.
 54. Nelson CA, Scott RD, Bhutta ZA, et al. Adversity in childhood is linked to mental and physical health throughout life. *BMJ* 2020; 371:m3048.
 55. O'Brien LM, Heycock EG, Hanna M, et al. Postnatal depression and faltering growth: a community study. *Pediatrics* 2004; 113:1242.
 56. Weston JA, Colloton M, Halsey S, et al. A legacy of violence in nonorganic failure to thrive. *Child Abuse Negl* 1993; 17:709.
 57. O'Connor ME, Szekely LJ. Frequent breastfeeding and food refusal associated with failure to thrive. A manifestation of the vulnerable child syndrome. *Clin Pediatr (Phila)* 2001; 40:27.
 58. Wright C, Birks E. Risk factors for failure to thrive: a population-based survey. *Child Care Health Dev* 2000; 26:5.
 59. Bureau of Family and Community Health. The Massachusetts Growth and Nutrition Program Summary Report FY 2003. Available at: www.mass.gov/eohhs/docs/dph/com-health/nutrition/report-2003.pdf (Accessed on January 23, 2012).
 60. Skuse DH, Gill D, Reilly S, et al. Failure to thrive and the risk of child abuse: a prospective population survey. *J Med Screen* 1995; 2:145.
 61. Block RW, Krebs NF, American Academy of Pediatrics Committee on Child Abuse and Neglect, American Academy of Pediatrics Committee on Nutrition. Failure to thrive as a manifestation of child neglect. *Pediatrics* 2005; 116:1234.

62. Gahagan S, Holmes R. A stepwise approach to evaluation of undernutrition and failure to thrive. *Pediatr Clin North Am* 1998; 45:169.
63. Centers for Disease Control and Prevention (CDC). "Choking game" awareness and participation among 8th graders--Oregon, 2008. *MMWR Morb Mortal Wkly Rep* 2010; 59:1.
64. Mehta NM, Corkins MR, Lyman B, et al. Defining pediatric malnutrition: a paradigm shift toward etiology-related definitions. *JPEN J Parenter Enteral Nutr* 2013; 37:460.
65. Casey PH. Growth of low birth weight preterm children. *Semin Perinatol* 2008; 32:20.
66. Wright CM, Williams AF, Elliman D, et al. Using the new UK-WHO growth charts. *BMJ* 2010; 340:c1140.
67. Pollitt E, Eichler A. Behavioral disturbances among failure-to-thrive children. *Am J Dis Child* 1976; 130:24.
68. Homer C, Ludwig S. Categorization of etiology of failure to thrive. *Am J Dis Child* 1981; 135:848.
69. Berwick DM, Levy JC, Kleinerman R. Failure to thrive: diagnostic yield of hospitalisation. *Arch Dis Child* 1982; 57:347.

Topic 2864 Version 55.0

GRAPHICS

Causes of poor weight gain, according to pathophysiologic mechanism

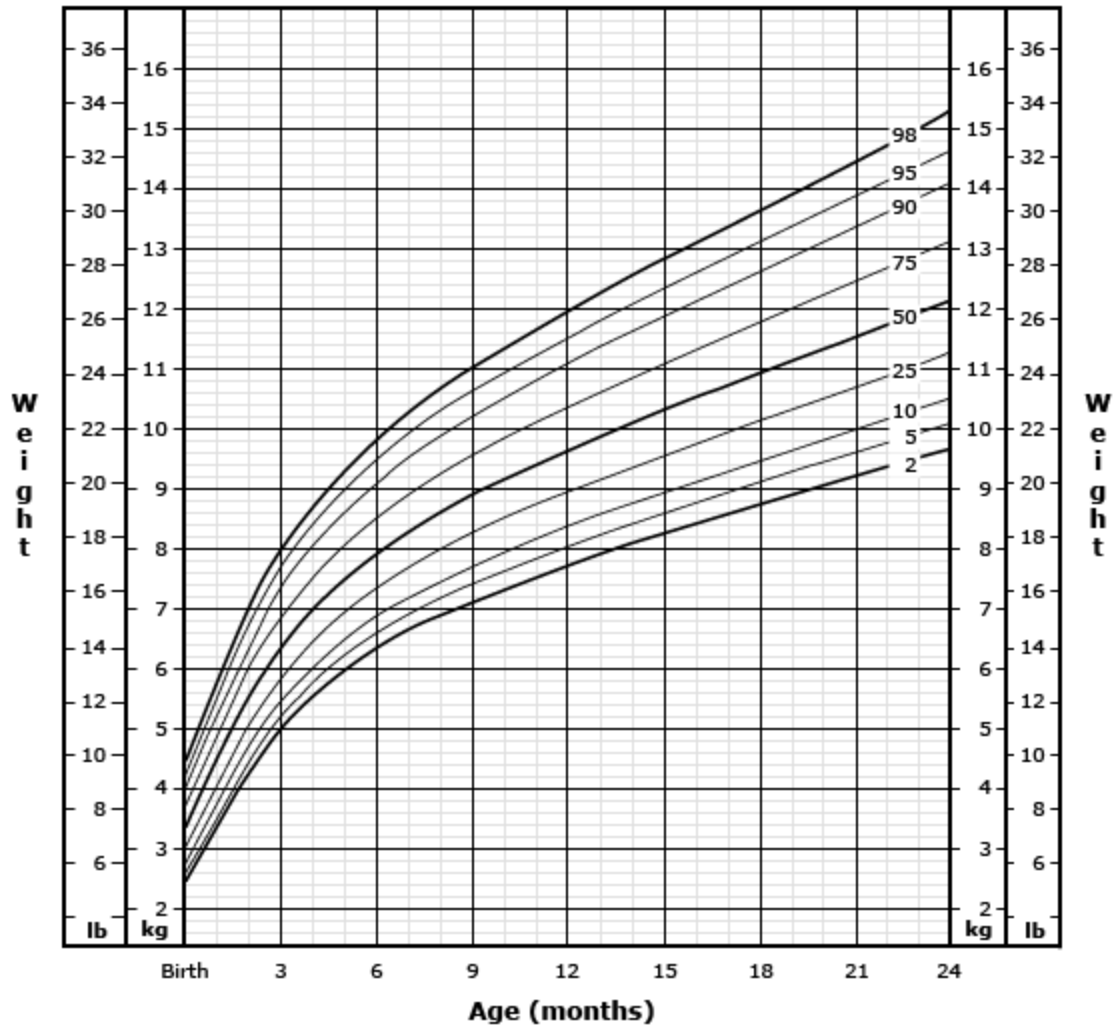
Inadequate nutrient intake	Inadequate nutrient absorption or increased losses
Inappropriate feeding technique	Malabsorption (lactose intolerance, cystic fibrosis, cardiac disease, malrotation, IBD, milk allergy, parasites, celiac disease)
Disturbed caregiver/child relationship	Biliary atresia, cirrhosis
Economic deprivation	Vomiting or "spitting up" (related to infectious gastroenteritis, increased intracranial pressure, adrenal insufficiency, or drugs [eg, purposeful administration of syrup of ipecac])
Inappropriate nutrient intake (eg, excess fruit juice consumption, factitious food allergy, inappropriate preparation of formula, inadequate quantity of food, inappropriate food for age, neglect, food fads)	Intestinal tract obstruction (pyloric stenosis, hernia, malrotation, intussusception)
Inappropriate parental knowledge of appropriate diet for infants and toddlers	Infectious diarrhea
Insufficient lactation in mother	Necrotizing enterocolitis or short bowel syndrome
Picky eater	Increased nutrient requirements or ineffective metabolic utilization
Gastroesophageal reflux	Hyperthyroidism
Psychosocial problems	Malignancy (including brain tumors such as diencephalic syndrome)
Maternal/infant dysfunction	Chronic IBD
Mechanical problems (cleft palate, nasal obstruction, adenoidal hypertrophy, dental lesions)	Chronic systemic disease (juvenile idiopathic arthritis)
Sucking or swallowing dysfunction (CNS, neuromuscular, esophageal motility problems) or chewing difficulty	Chronic or recurrent systemic infection (urinary tract infection, tuberculosis, toxoplasmosis)
Inadequate appetite or inability to eat large amounts	Chronic metabolic problems (hypercalcemia, storage diseases, and inborn errors of metabolism, such as galactosemia, methylmalonic acidemia, diabetes mellitus, adrenal insufficiency)
Oral aversion/problem with certain textures	Chronic respiratory insufficiency (bronchopulmonary dysplasia, cystic fibrosis)
Psychosocial problems – apathy or rumination	Congenital or acquired heart disease
Cardiopulmonary disease	
Hypotonia, muscle weakness, or hypertonia	
Anorexia of chronic infection or immune deficiency	
Cerebral palsy	

CNS pathology (eg, tumor, hydrocephalus)
Genetic syndromes
Anemia (eg, iron deficiency)
Chronic constipation
Gastrointestinal disorder (eg, pain from gastroesophageal reflux, intestinal tract obstruction)
Craniofacial anomalies (eg, cleft lip and palate, micrognathia)

CNS: central nervous system; IBD: inflammatory bowel disease.

Graphic 69846 Version 9.0

Weight-for-age percentiles, males 0 to 24 months, WHO growth standards

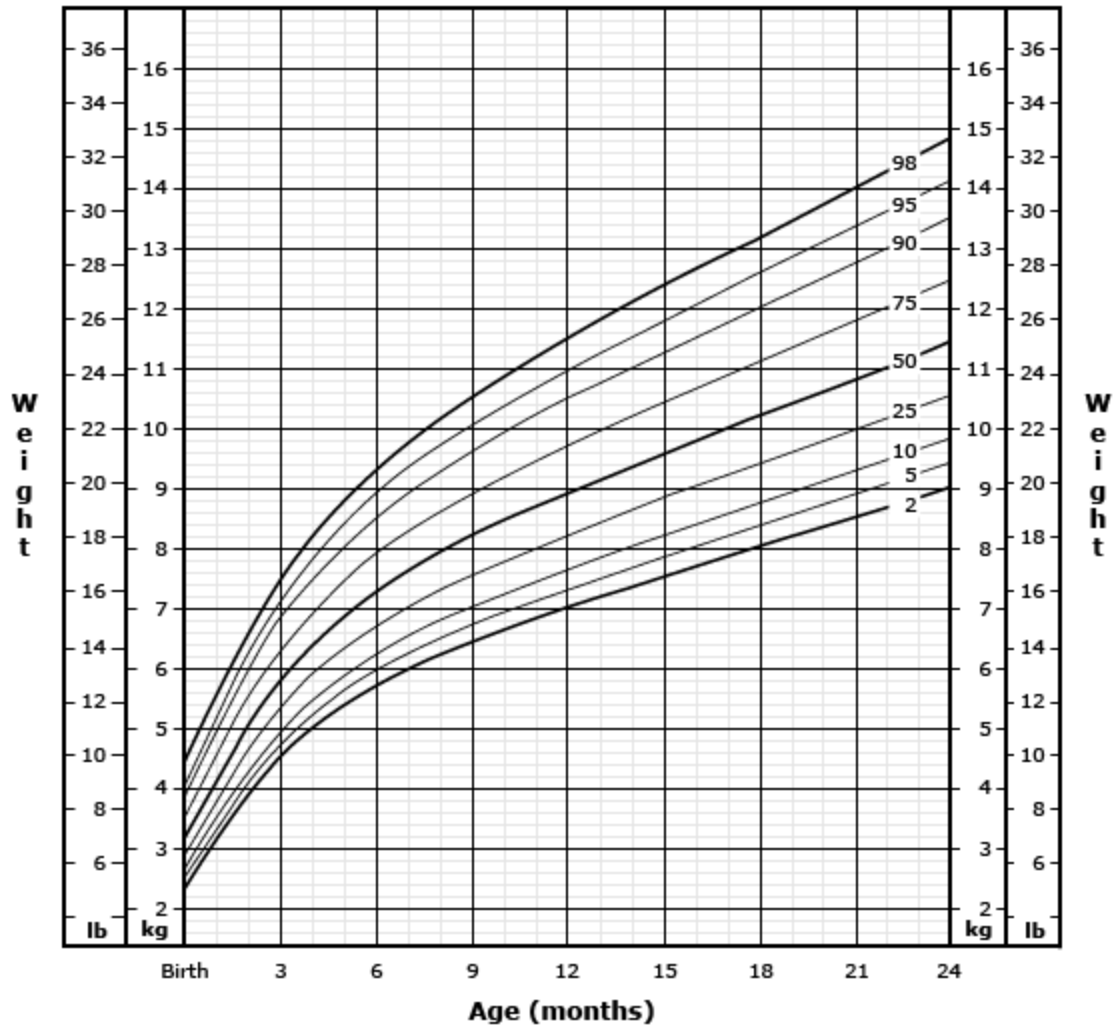


WHO: World Health Organization.

Reproduced from: Centers for Disease Control and Prevention based on data from the WHO Child Growth Standards.

Graphic 50006 Version 4.0

Weight-for-age percentiles, females 0 to 24 months, WHO growth standards

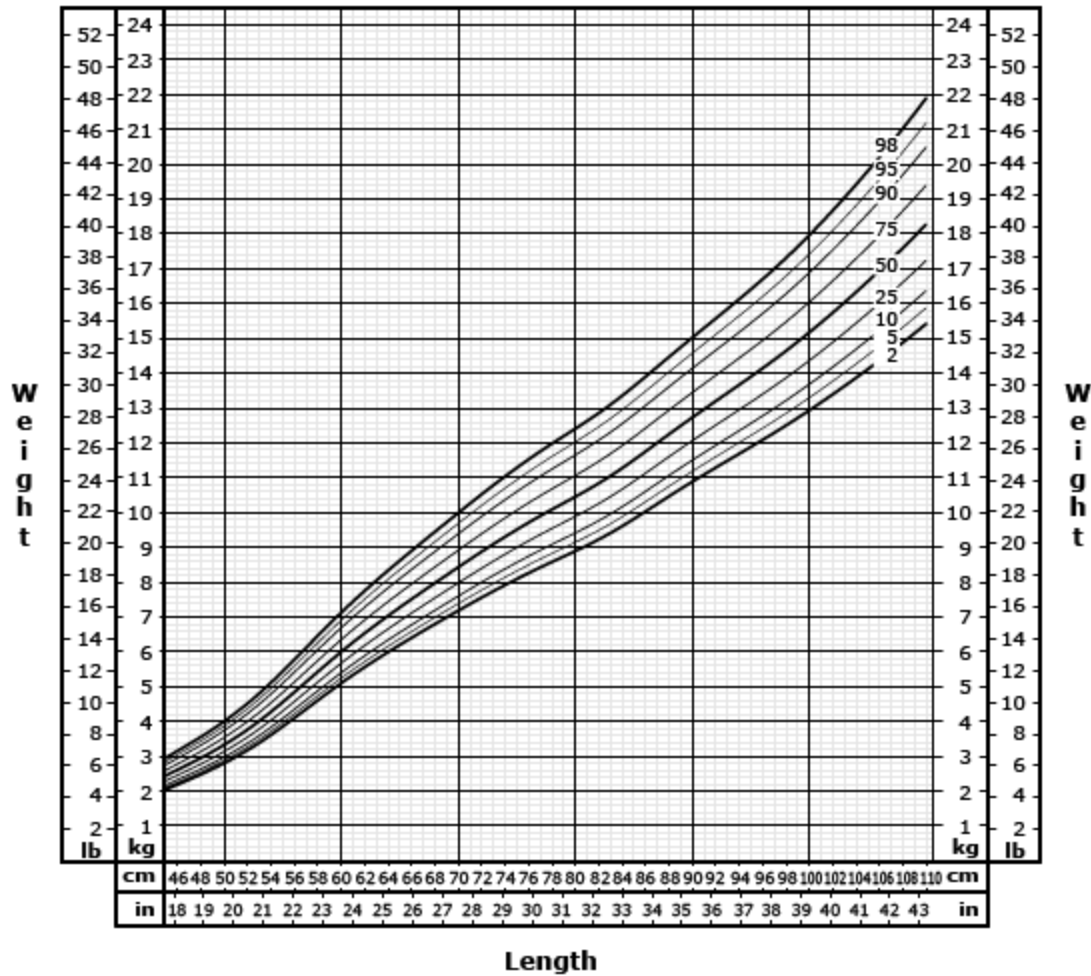


WHO: World Health Organization.

Reproduced from: Centers for Disease Control and Prevention based on data from the WHO Child Growth Standards.

Graphic 63877 Version 6.0

Weight-for-length percentiles, males 0 to 24 months, WHO growth standards

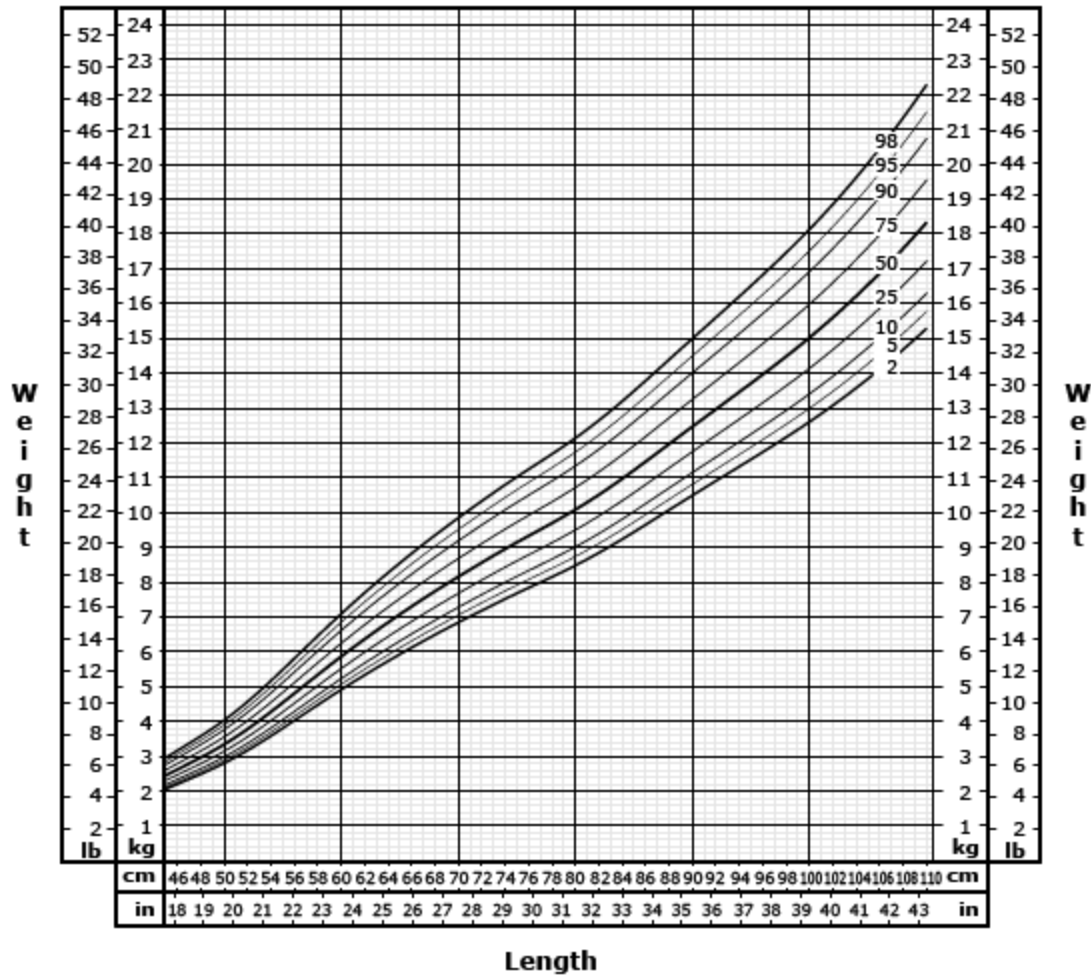


WHO: World Health Organization.

Reproduced from: Centers for Disease Control and Prevention based on data from the WHO Child Growth Standards.

Graphic 53474 Version 6.0

Weight-for-length percentiles, females 0 to 24 months, WHO growth standards

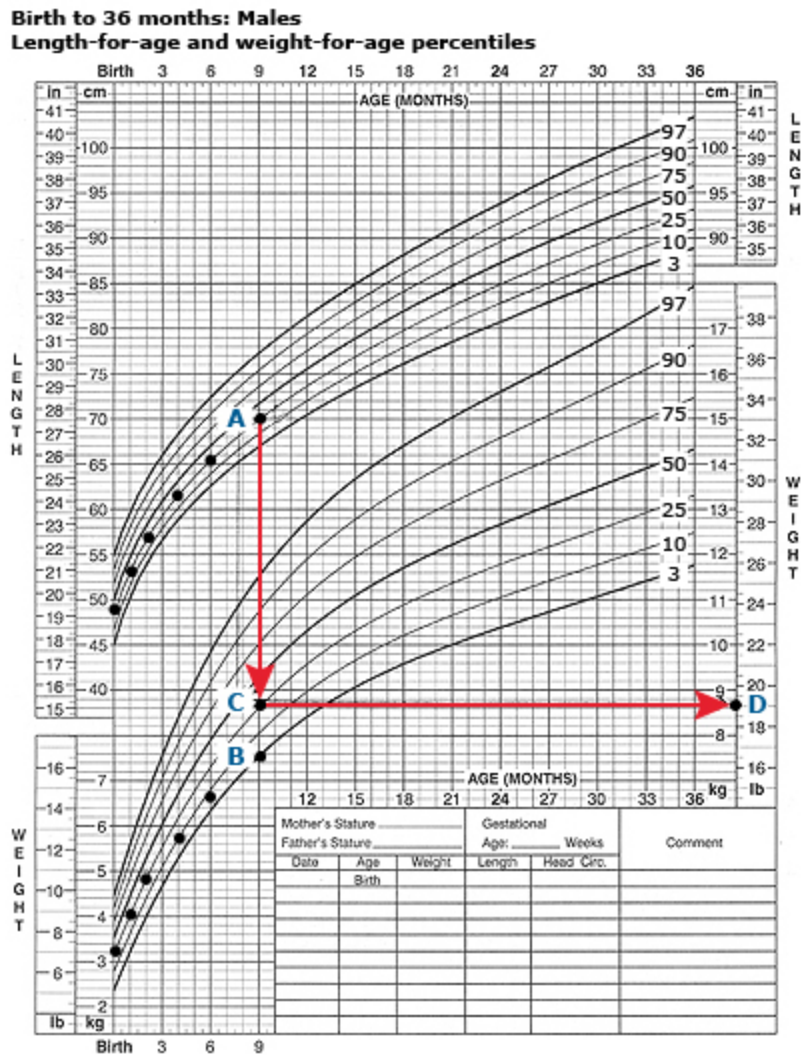


WHO: World Health Organization.

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Graphic 73036 Version 7.0

Determination of percent ideal body weight for a 9-month-old male who weighs 7.6 kg and is 70 cm long



To determine ideal body weight (IBW), plot the child's length on the length curve (point A) and the weight on the weight curve (point B). Determine the child's length percentile (in this example, 25th). Draw a vertical line from the length point to the same percentile curve for weight (point C). Draw a horizontal line to the weight scale to determine ideal body weight (point D). Percent IBW = actual weight/IBW × 100. In this example, IBW = 8.6 kg, and percent IBW = 88%.

Data from: The National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000). Available at www.cdc.gov/growthcharts.

Graphic 57059 Version 3.0

Average daily weight gain for age

Age	Median daily weight gain (g)
0 to 3 months	26 to 31
3 to 6 months	17 to 18
6 to 9 months	12 to 13
9 to 12 months	9
1 to 3 years	7 to 9
4 to 6 years	6

From: Kistin CJ, Frank DA. Failure to thrive. In: Zuckerman Parker Handbook of Developmental and Behavioral Pediatrics for Primary Care, 4th ed, Augustyn M, Zuckerman B (Eds), Wolters Kluwer, Philadelphia 2018. Copyright © 2018. Reproduced with permission from Wolters Kluwer Health. Unauthorized reproduction of this material is prohibited.

Graphic 73565 Version 7.0

Alternative methods of classifying severity of malnutrition in children younger than 2 years of age with poor weight gain in resource-abundant settings^[1]

Growth parameters	Mild malnutrition	Moderate malnutrition	Severe malnutrition
Weight-for-length Z-score*	-1 to -1.9	-2 to -2.9	≤ -3
Mid-upper arm circumference Z-score	-1 to -1.9	-2 to -2.9	≤ -3
Decline in weight-for-length Z-score	≥ 1	≥ 2	≥ 3

WHO: World Health Organization.

* The WHO weight-for-length percentiles for infants <24 months can be determined using the related UpToDate calculator.

¶ As an alternative, the WHO also defines wasting in children 6 to 59 months as mid-upper arm circumference 115 to 125 mm (moderate wasting) and <115 mm (severe wasting). These flat cutoffs can be used as field measures in place of Z-scores, particularly in low-resource settings^[2].

References:

1. Becker P, Carney LN, Corkins MR, et al. Consensus statement of the Academy of Nutrition and Dietetics/American Society for Parenteral and Enteral Nutrition: Indicators recommended for the identification and documentation of pediatric malnutrition (undernutrition). *Nutr Clin Pract* 2015; 30:147.
2. Note on terminology of "wasting" and "acute malnutrition." World Health Organization 2023. https://fscluster.org/sites/default/files/documents/note_on_terminology_acute_malnutrition_and_wasting_who_feb_2023.pdf (Accessed on July 19, 2023).

Graphic 134408 Version 4.0

Potential factors contributing to poor weight gain by age of onset

Age of onset	Potential contributing factors
Prenatal	<ul style="list-style-type: none"> ▪ Fetal (intrauterine) growth restriction ▪ Prematurity ▪ Prenatal infection ▪ Congenital syndrome (metabolic or chromosomal) ▪ Teratogenic exposure (eg, alcohol, anticonvulsant, tobacco smoke, caffeine)
Birth to 6 months	<ul style="list-style-type: none"> ▪ Poor quality of suck (whether breast- or bottle-fed) and/or oral motor dysfunction ▪ Improper formula preparation ▪ Breastfeeding problems, including insufficient milk supply ▪ Inadequate number of feedings ▪ Poor feeding interactions (eg, infant gags or vomits during feeding, caretaker misreads signals of hunger or satiety) ▪ Child neglect ▪ Parental mental illness ▪ Metabolic, chromosomal, or anatomic abnormalities ▪ Underfeeding (possibly related to poverty) ▪ Milk protein intolerance ▪ Cystic fibrosis ▪ Congenital heart disease ▪ Gastroesophageal reflux disease
7 to 12 months	<ul style="list-style-type: none"> ▪ Feeding problems, for example: <ul style="list-style-type: none"> • Autonomy struggles, particularly if caretaker is unduly anxious about intake or cleanliness • Oral motor dysfunction that interferes with adaptation to more textured foods • Delayed introduction of solid foods • Refusal to eat new foods when first offered so caretaker no longer offer: • Caretaker does not offer adequate quantity or variety of solid foods ▪ Intestinal parasites ▪ Food allergies
>12 months	<ul style="list-style-type: none"> ▪ Coercive feeding ▪ Picky eater ▪ Highly distractible child ▪ Distracting environment ▪ Acquired illness

- Social factors (eg, underfeeding related to fear of overfeeding, poverty)
- New psychosocial stressor (eg, divorce, job loss, new sibling, etc)
- Sensory-based feeding disorders in children with developmental disorders (eg, autism spectrum disorder)
- Chewing or swallowing dysfunction
- Excessive milk or juice intake
- Caretaker does not offer enough quantity or combination of healthy foods
- Celiac disease

References:

1. Cardona Cano S, Hoek HW, Bryant-Waugh R. Picky eating: The current state of research. *Curr Opin Psychiatry* 2015; 28:448.
2. Emond A, Drewett R, Blair P, Emmett P. Postnatal factors associated with failure to thrive in term infants in the Avon Longitudinal Study of Parents and Children. *Arch Dis Child* 2007; 92:115.
3. Frank D, Silva M, Needlman R. Failure to thrive: Mystery, myth and method. *Contemp Pediatr* 1993; 10:114.
4. McDougall P, Drewett RF, Hungin AP, Wright CM. The detection of early weight faltering at the 6-8-week check and its association with family factors, feeding and behavioural development. *Arch Dis Child* 2009; 94:549.
5. Olsen EM, Skovgaard AM, Weile B, et al. Risk factors for weight faltering in infancy according to age at onset. *Paediatr Perinat Epidemiol* 2010; 24:370.

Graphic 104765 Version 6.0

Historical clues to potential organic causes of poor weight gain in children

Historical clues	Potential significance
Diarrhea	Malabsorption (celiac disease, inflammatory bowel disease, cystic fibrosis secondary lactase deficiency, food allergy, short bowel syndrome), excessive use of fructose-or sorbitol-based juices, diets high in sugar or other carbohydrates
Chronic constipation	May cause decreased appetite
Abdominal pain	Gastroesophageal reflux, esophagitis, chronic constipation
Vomiting or spitting up	Gastroesophageal reflux, delayed gastric emptying, intestinal obstruction food allergy/intolerance
Gagging, tactile hypersensitivity, prolonged feeding time	Oral motor dysfunction
Decreased appetite	Excessive juice (or other nonnutritious liquid) intake, chronic disease, medications, stressful psychosocial conditions
Recent travel to developing country, camping, housing in shelter, day care	Infectious diarrhea (eg, giardiasis, nematodes, enteric pathogens)
Chronic otitis media	Immune deficiency, structural abnormality that impairs intake
Snoring or mouth breathing	Adenoidal hypertrophy
History of wheezing	Mechanical obstruction (eg, vascular ring), chronic pulmonary disease
Polyuria, polydipsia, polyphagia	Diabetes
Frequent infections	Immune deficiency

Data from:

1. Frank D, Silva M, Needlman R. Failure to thrive: Mystery, myth and method. *Contemp Pediatr* 1993; 10:114.
2. American Academy of Pediatrics Committee on Nutrition. Failure to thrive. In: *Pediatric Nutrition*, 7th ed, Kleinman RE, Greer FR (Eds), American Academy of Pediatrics, Elk Grove Village, IL 2014. p.663.

Graphic 57170 Version 9.0

Important aspects of the feeding history in the evaluation of the child (<2 years of age) with poor weight gain

General aspects
When does the child eat? (Are there regular and appropriately spaced feeding intervals?)
How much juice (or other sweetened beverages such as soda) does the child drink per day?
Does the family follow a special diet (eg, vegetarian, "heart healthy")? What advice has been followed thus far?
When did the child begin solid foods? How were they introduced? How were they tolerated?
Does the child spit up or vomit in relation to feeding?
Are there any stooling patterns associated with feeding?
Does the infant tire with feeding? (May indicate cardiac or pulmonary disorder.)
Breastfed infants
How often does the infant nurse? (Should be at least 8 times per day between 0 to 4 months; 5 times per day between 4 and 8 months; and on demand thereafter.)
How long does the infant nurse?
Is feeding providing adequate stimulation of milk production?
Can the mother hear the infant swallow?
Does the baby have a strong suck/strong latch? (A weak suck may indicate neurologic disorder.)
Does the mother feel the sensation of let-down (a warm, tingly feeling as the milk begins to flow from the breast when the infant cries)?
Is the mother stressed or fatigued? (These may result in low milk production.)
Does the infant tolerate vitamin D supplement?
Formula-fed infants
How much formula does the child ingest? How often? (Daily intake should be approximately 16 to 32 oz at 0 to 4 months, 24 to 40 oz at 4 to 6 months, 24 to 32 oz at 6 to 8 months, 16 to 32 oz at 8 to 10 months, and 16 to 24 oz at 10 to 12 months.)
How is the formula mixed?
Does the caregiver add anything (eg, infant cereal, baby foods) to the bottle?
Does the caregiver hold the bottle during feedings or prop the bottle in the infant's mouth?
Feeding environment
Who feeds the child? If the child is fed by multiple caregivers, do they have consistent feeding styles?
Where does the child eat (eg, in a high chair, on a caregiver's lap)?

Does the child usually eat alone or with others?

Are there mealtime distractions (eg, television)?

Are there any limitations (eg, food insecurity or self-imposed dietary restrictions)?

Feeding behavior/interactions

How does the caregiver know when the child is hungry?

How does the caregiver know when the child is not hungry?

Does the caregiver say or do anything when the child eats well?

Does the caregiver say or do anything when the child eats poorly?

Does the child refuse food?

Do the caregiver and the child have struggles over feeding?

Does the child have strong likes and dislikes (ie, is the child "picky")? If so, can/does the caregiver feed the child accordingly?

Does the child feed differently with different people?

Does the child make a mess when they eat? If so, is this difficult for the caregiver?

Data from:

1. Zenil JA Jr. Failure to thrive: A general pediatrician's perspective. *Pediatr Rev* 1997; 18:371.
2. Bithoney WG, Dubowitz H, Egan H. Failure to thrive/growth deficiency. *Pediatr Rev* 1992; 13:453.
3. Gahagan S, Holmes R. A stepwise approach to evaluation of undernutrition and failure to thrive. *Pediatr Clin North Am* 1998; 45:169.
4. Frank D, Silva M, Needleman R. Failure to thrive: Mystery, myth and method. *Contemp Pediatr* 1993; 10:114.
5. Frank DA, Zeisel SH. Failure to thrive. *Pediatr Clin North Am* 1988; 35:1187.

Graphic 55626 Version 9.0

Important aspects of the physical examination in the evaluation of poor weight gain in children

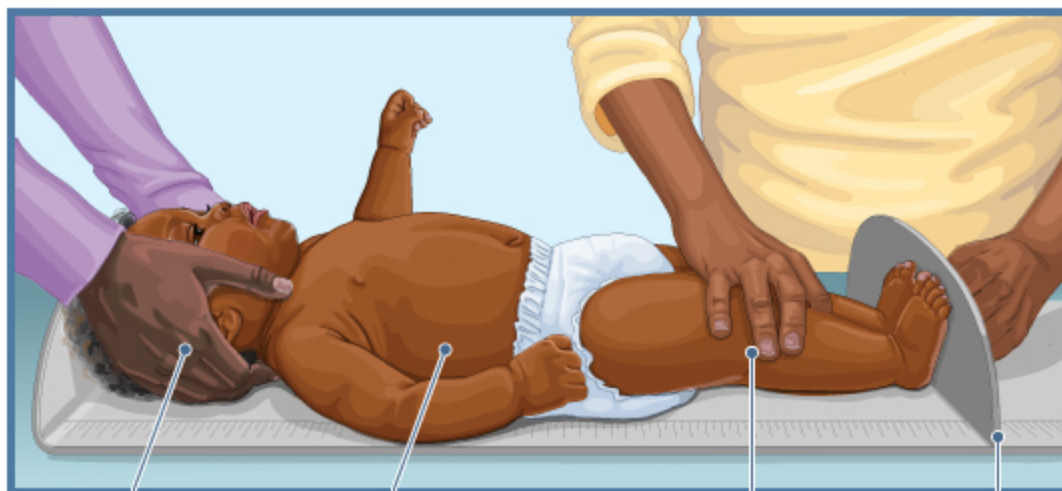
Examination clues	Potential significance
Vital signs	
Hypotension	Adrenal or thyroid insufficiency
Hypertension	Kidney disease
Tachypnea/tachycardia	Increased metabolic demands
General appearance	
Pallor	Anemia
Drooling	Oral motor dysfunction
Cachexia, temporal wasting, sparse hair or alopecia	Significant malnutrition
Inadequate adiposity	Indicator of nutritional inadequacy
Dysmorphic features	Clinical or genetic syndrome associated with poor weight gain
Head and neck	
Microcephaly	Neurologic disorder, fetal alcohol syndrome
Delayed closure of fontanelle	Vitamin D deficiency, hypothyroidism
Short palpebral fissures	Fetal alcohol syndrome
Cataracts	Congenital infection, galactosemia
Papilledema	Increased intracranial pressure
Smooth philtrum	Fetal alcohol syndrome
Aphthous stomatitis	Crohn disease
Thin vermilion border	Fetal alcohol syndrome
Oropharyngeal lesions (eg, caries, tongue enlargement, mandibular hypoplasia, tonsillar hypertrophy, defects in soft or hard palate)	May interfere with eating
Delayed tooth eruption	Delayed bone age
Thyroid enlargement	Thyroid disease
Low hairline	Genetic syndrome (eg, Klippel-Feil)
Chest	

Wheezing, crackles, prolonged expiratory phase, hyperexpansion	Cystic fibrosis, asthma
Cardiac murmur	Congenital or acquired heart disease
Abdomen	
Abdominal distension, hyperactive bowel sounds	Malabsorption
Hepatosplenomegaly	Liver disease, glycogen storage disease, malignancy
Genitourinary	
Genitourinary abnormalities	Endocrinopathy
Rectal fistulae, large perianal skin tags	Crohn disease
Musculoskeletal	
Clubbing	Chronic hypoxia due to cardiac or pulmonary disorders
Bony deformities (craniotabes, beading of the ribs, scoliosis, bowing of the legs or distal radius and ulna, enlargement of the wrist)	Rickets
Edema	Protein deficiency
Neurologic	
Abnormal deep tendon reflexes	Cerebral palsy
Hypotonia, weakness, spasticity	May be associated with oral motor dysfunction
Neuropathy	Vitamin deficiencies – B12, B3 (niacin), B6 (pyridoxine), E (tocopherol)
Skin and mucous membranes	
Scaling skin	Zinc deficiency
Candidiasis	Immune deficiency
Spoon-shaped nails	Iron deficiency
Cheilosis	Vitamin deficiency – B2 (riboflavin), B3 (niacin), or B6 (pyridoxine)
Chronic diaper rash	Possible neglect
Bruises in characteristic patterns	Possible abuse

Data from:

1. Frank D, Silva M, Needlman R. Failure to thrive: Mystery, myth and method. *Contemp Pediatr* 1993; 10:114.
2. American Academy of Pediatrics Committee on Nutrition. Failure to thrive. In: *Pediatric Nutrition*, 7th ed, Kleinman RE, Greer FR (Eds), American Academy of Pediatrics, Elk Grove Village, IL 2014. p.663.

Technique for measuring recumbent length

A


Head should be aligned with body and gently held in position

Child should be fully supine (flat)

Knees should be pressed down gently

Moveable baseboard

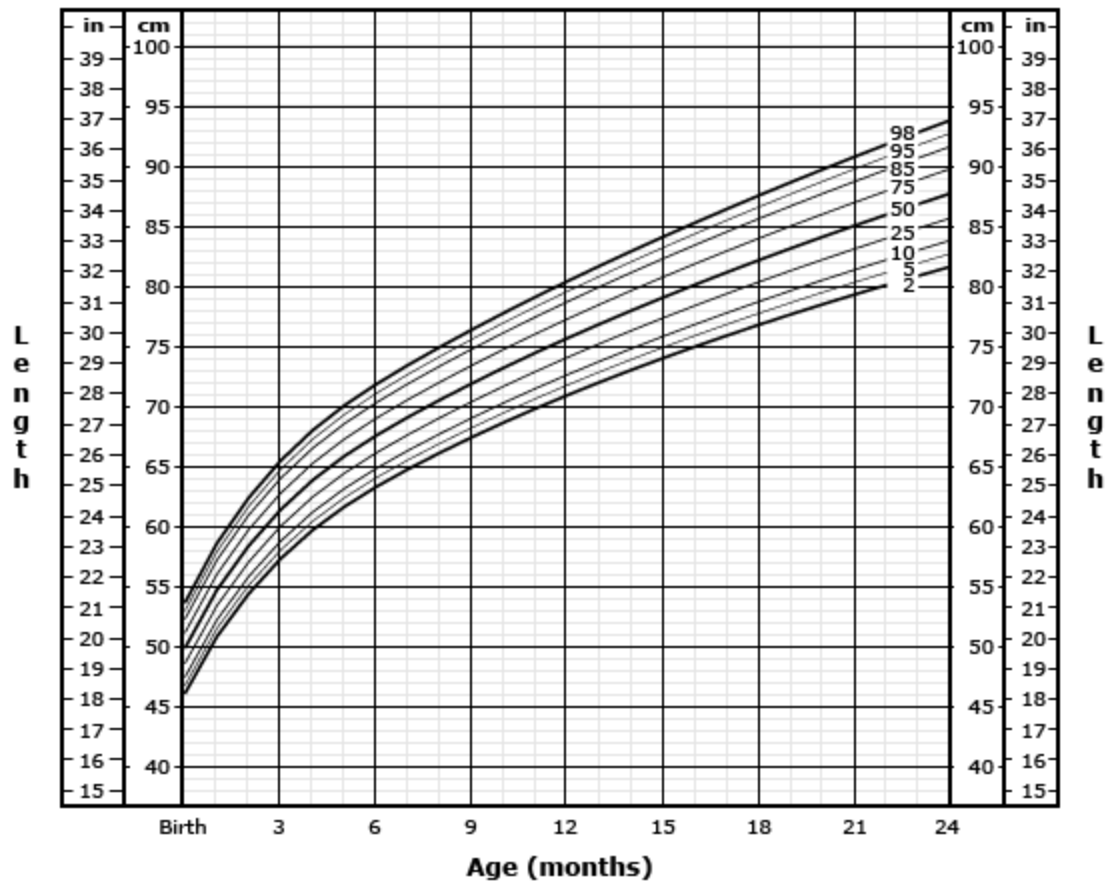
B


(A) Measurement of length using a length stadiometer. This is the preferred method for measuring stature in a child less than 2 years old. For an accurate measurement, the child must be held fully supine, with knees fully extended, as shown.

(B) Measurement of length using a tape measure. This is less accurate and should only be used if a length stadiometer is not available. The measurement should be done on a firm, flat surface, such as the examining table. With the child held gently in the proper position, make marks on the surface to indicate the position of the head and heels, then remove the child from the table and measure the distance between the marks.

A similar technique may be used for older individuals who are unable to stand, recognizing that recumbent length (when optimally measured) is generally slightly more than standing height.

Length-for-age percentiles, males 0 to 24 months, WHO growth standards

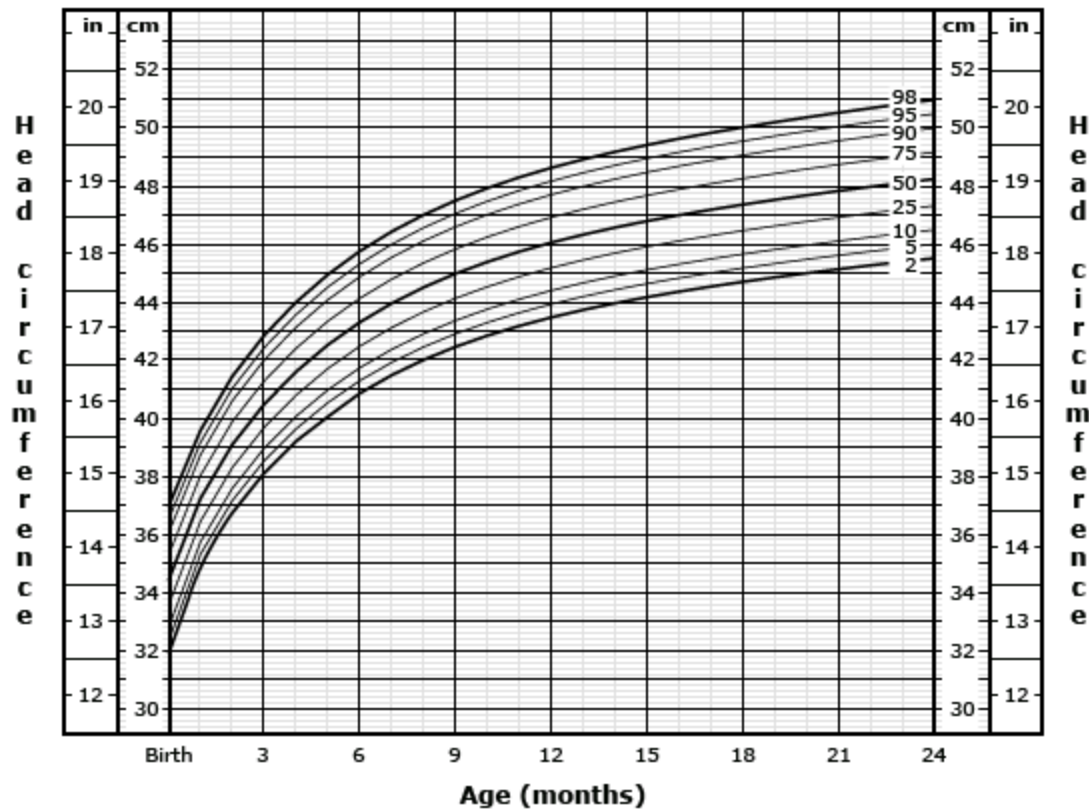


WHO: World Health Organization.

Reproduced from: Centers for Disease Control and Prevention based on data from the WHO Child Growth Standards.

Graphic 67950 Version 6.0

Head circumference-for-age percentiles, males 0 to 24 months, WHO growth standards

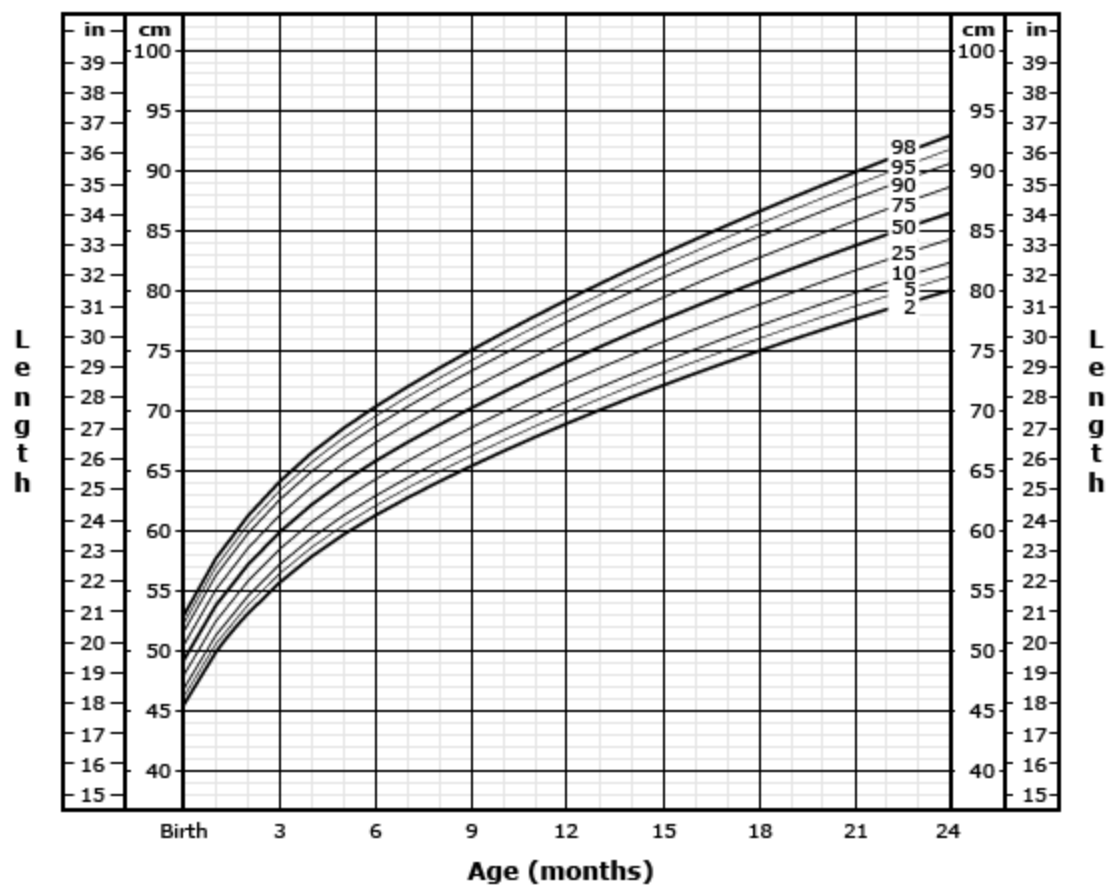


WHO: World Health Organization.

Reproduced from: Centers for Disease Control and Prevention based on data from the WHO Child Growth Standards.

Graphic 58632 Version 4.0

Length-for-age percentiles, females 0 to 24 months, WHO growth standards

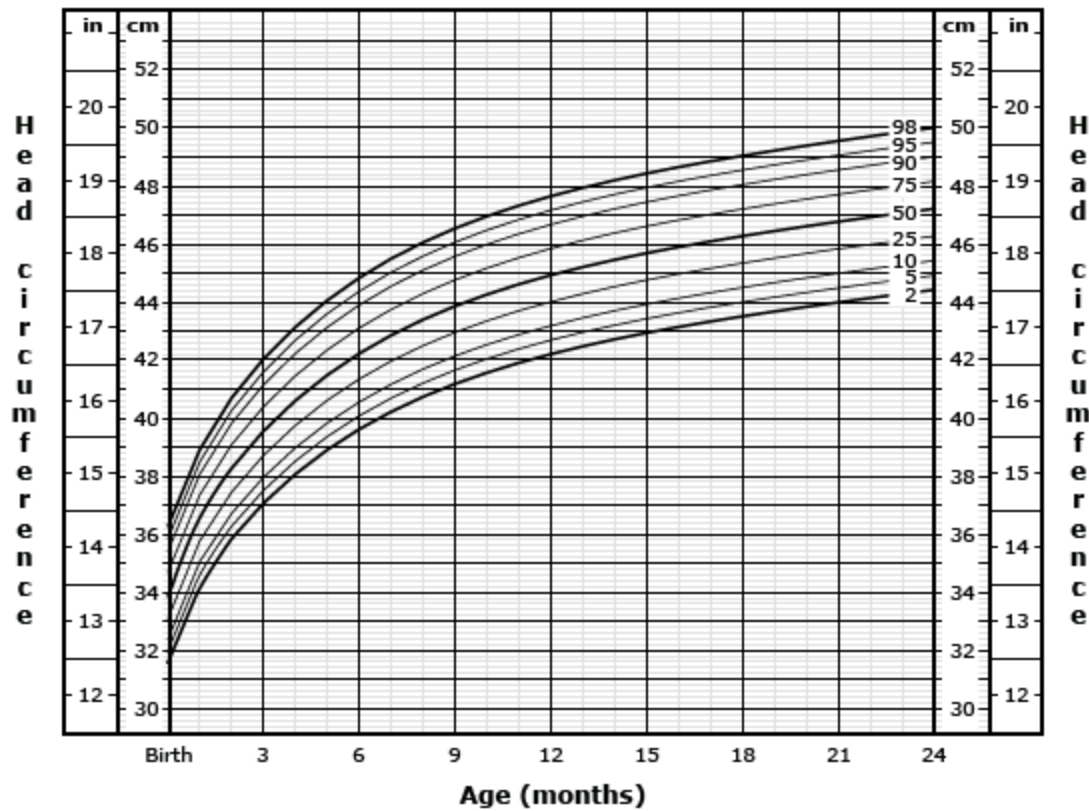


WHO: World Health Organization.

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Graphic 80511 Version 4.0

Head circumference-for-age percentiles, females 0 to 24 months, WHO growth standards



WHO: World Health Organization.

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Graphic 74503 Version 4.0

Initial laboratory and imaging evaluation for poor weight gain in children younger than 2 years in resource-abundant countries

Test(s)	Rationale
Basic tests (performed in most children)	
CBC, CRP, ESR	<ul style="list-style-type: none"> Screen for anemia, chronic infection, inflammation, malignancy
Urinalysis, urine culture	<ul style="list-style-type: none"> Screen for protein or carbohydrate loss (eg, glucosuria) and indolent kidney disease (eg, renal tubular acidosis, UTI)
Additional tests for initial evaluation if clinically indicated by the history or examination	
<ul style="list-style-type: none"> Electrolytes, BUN, creatinine, glucose Calcium, phosphorus, magnesium Albumin, total protein, liver enzymes Amylase, lipase Prealbumin 	<ul style="list-style-type: none"> Evaluation of kidney, liver, and pancreatic disease Assessment of nutritional status
Stool studies: <ul style="list-style-type: none"> Guaiac, leukocytes Routine culture, ova and parasite smears <i>Giardia</i> antigen 	<ul style="list-style-type: none"> Evaluate gastrointestinal infection
Chest radiograph	<ul style="list-style-type: none"> Evaluate cardiopulmonary disease

CBC: complete blood count; CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; UTI: urinary tract infection; BUN: blood urea nitrogen.

Graphic 134406 Version 1.0

Advanced laboratory and imaging tests in the evaluation of poor weight gain in children younger than 2 years in resource-abundant countries

Suspected condition(s)	Test(s)
Food allergy	<ul style="list-style-type: none"> Serum IgE, RAST, and skin tests to selected food antigens
Gastrointestinal disease	
<ul style="list-style-type: none"> Celiac disease 	<ul style="list-style-type: none"> Tissue transglutaminase and serum IgA
<ul style="list-style-type: none"> <i>Helicobacter pylori</i> 	<ul style="list-style-type: none"> Stool antigen assay or 13C-urea breath test
<ul style="list-style-type: none"> Malabsorption 	<ul style="list-style-type: none"> Carbohydrates: Stool reducing substances Protein: Stool alpha-1-antitrypsin Fat: Stool elastase
<ul style="list-style-type: none"> Inflammatory bowel disease 	<ul style="list-style-type: none"> Stool guaiac, leucocytes, and calprotectin Upper GI series with small bowel follow-through Advanced endoscopic studies
<ul style="list-style-type: none"> Gastroesophageal reflux disease 	<ul style="list-style-type: none"> Esophageal pH and impedance monitoring Advanced endoscopic studies
<ul style="list-style-type: none"> Swallowing dysfunction or aspiration 	<ul style="list-style-type: none"> Swallowing function study
<ul style="list-style-type: none"> Vomiting or anorexia related to an intra-abdominal or intracranial process 	<ul style="list-style-type: none"> Possible studies include: <ul style="list-style-type: none"> Abdominal ultrasonography Radionuclide scans for gastric and biliary tract emptying Abdominal and head computed tomography scans
Cystic fibrosis	<ul style="list-style-type: none"> Sweat chloride test
Inborn errors of metabolism or genetic syndromes	<ul style="list-style-type: none"> Possible studies include: <ul style="list-style-type: none"> Serum amino acids Urine organic acids Urine reducing substances Serum carnitine Chromosome studies
Endocrine disorders	
<ul style="list-style-type: none"> Hyperthyroidism 	<ul style="list-style-type: none"> Serum TSH, free T4, and T3

▪ Growth hormone deficiency	▪ IGF-1 and IGFBP-3
Infectious diseases	
▪ HIV infection	▪ Refer to UpToDate topics on HIV testing and diagnosis
▪ Tuberculosis	▪ TST
▪ CMV	▪ CMV IgM and IgG
▪ EBV	▪ EBV IgM and IgG
▪ Hepatitis A, B, or C	▪ Hepatitis panel

Select advanced laboratory and imaging tests that may be performed in the evaluation of poor weight gain in children <2 years of age in resource-abundant countries. Advanced testing is warranted if specific diagnoses are suspected or if the diagnosis remains uncertain and the child has not responded to dietary and/or behavioral interventions. Testing should be targeted to clinical findings.

IgE: immunoglobulin E; RAST: radioallergosorbent test; IgA: immunoglobulin A; GI: gastrointestinal; TSH: thyroid-stimulating hormone; T4: thyroxine; T3: triiodothyronine; IGF-1: insulin-like growth factor; IGFBP-3: insulin-like growth factor binding protein-3; TST: tuberculin skin test; CMV: cytomegalovirus; IgM: immunoglobulin M; IgG: immunoglobulin G; EBV: Epstein-Barr virus.

Graphic 134405 Version 1.0

Contributor Disclosures

Teresa K Duryea, MD No relevant financial relationship(s) with ineligible companies to disclose. **Jan E Drutz, MD** No relevant financial relationship(s) with ineligible companies to disclose. **Craig Jensen, MD** Consultant/Advisory Boards: Mead Johnson Nutrition [Pediatric nutrition]. Speaker's Bureau: Mead Johnson Nutrition [Pediatric nutrition]. All of the relevant financial relationships listed have been mitigated. **Marilyn Augustyn, MD** Grant/Research/Clinical Trial Support: Health Resources and Services Administration [Fellowship training]; Irving Harris Network [Fellowship training]. All of the relevant financial relationships listed have been mitigated. **Alison G Hoppin, MD** No relevant financial relationship(s) with ineligible companies to disclose.

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