Failure To Thrive: An Old Nemesis in the New Millennium

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OBJECTIVES:

After completing this article, readers should be able to:

1. **Plot growth data accurately.**
2. **Delineate the percentage of normal infants who experience a downward shift of growth between 3 and 18 months of life.**
3. **Describe the common bases for failure to thrive and the approximate percentages of patients who have these etiologies.**
4. **Characterize the mainstay of intervention for failure to thrive.**
5. **Describe the effect of failure to thrive on future development, behavior, and cognition.**

Introduction

An early reference to an infant who "ceased to thrive" can be traced to more than a century ago in the initial edition of *The Diseases of Infancy and Childhood* by L. Emmett Holt in 1897. Holt equated infantile wasting conditions with malnutrition, although he clearly recognized that this could be associated with a variety of clinical circumstances. The phrase "fail to thrive" first seems to have appeared in print in 1933 in the 10th edition of that classic text. In the late 1960s, the psychosocial aspects of failure to thrive became synonymous with maternal deprivation syndrome (more appropriately renamed the "parental" deprivation syndrome) and earned an entry in the American Psychiatric Association's *Diagnostic and Statistical Manual* (DSM-III) as "reactive attachment disorder." Thus, the long litany of metabolic, infectious, and nutritionally derived conditions were made distinct from the environmental aspects when evaluating poor growth during infancy and childhood. This article focuses on childhood aspects of failure to thrive.

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Definitions

Considering the widespread use of the term failure to thrive, a consensus definition remains curiously elusive. By strict statistical definition, 3% of the pediatric population deviates from normal stature or weight for age and gender; indeed, some literature simply defines failure to thrive as a fall in weight below the 3rd percentile relative to age (weight-for-age). However, this definition would include children who experience transient weight decreases due to intercurrent illnesses. Other anthropometric criteria revolve around decreased weight-for-height or height-for-age. Still others have suggested that the definition encompass only children younger than 2 years of age whose growth (generally weight) decelerates and crosses two major percentiles on a standardized growth chart. Other literature qualifies further by suggesting that the poor growth must occur in the absence of biologic explanation, a definition that may not distinguish organic failure to thrive and nonorganic failure to thrive (vide infra). For many, the definition also connotes an inadequacy in social or developmental achievement. For the purposes of this review, the author suggests that the diagnosis of failure to thrive emphasizes the deceleration of growth (in stature or weight) without strict regard to cause, generally encompasses younger children and infants, and includes psychosocial and developmental concerns as integral parts of the diagnosis.

Creation

An understanding of abnormal growth requires a review of normal growth patterns. Just as postnatal growth is a good overall indicator of childhood health and development, prenatal growth is a fundamental tool to assess fetal health. It is beyond the scope of this text to review the complete intrauterine and extrauterine factors that influence fetal growth, and conditions leading to fetal macrosomia will not be reviewed. Although there are different definitions of normal fetal growth, an accurate determination of normal intrauterine growth is the ponderal index (PI): mass (g) divided three times by the crown-to-heel length (cm) multipled by 100.

$$PI = \frac{mass\ (g)}{length\ (cm)^3} \times 100$$

The normal range is 2.32 to 2.85 g/cm^3.

Fetal growth is characterized not only by cell enlargement, but also by cell replication. The initial phase of fetal growth, cellular hyperplasia, is characterized by active mitosis during the first 16 weeks of gestation. This is followed by a combination of slowing cellular hyperplasia and increasing cellular hypertrophy during weeks 16 to 32. From that time until term, there is further accretion of fat and protein. Irregularities during any phase may lead to subsequent growth abnormalities and may influence postnatal growth. Disruption of cellular growth during the first stage leads to so-called symmetric intrauterine growth retardation; disturbances during the last trimester result in asymmetric intrauterine growth retardation. Often the literature refers to these infants as being small for gestational age.

Several variables influence fetal growth, ranging from maternal health factors to fetal components (Table 1). The major determinants

TABLE 1. Factors That Affect Fetal Growth Adversely

Maternal
- Congenital
  - —Genetic
    - Ethnic
    - Age
  - —Malformation (eg, bicornuate uterus)
- Acquired
  - —Inflammation
    - Autoimmune
    - Infectious
  - —Toxins
    - Alcohol
    - Tobacco
    - Medications and illicit drugs
    - Irradiation
  - —Tumor (eg, uterine leiomyoma)
  - —General health
    - Nutrition
    - Cardiac
    - Pulmonary
    - Renal
    - Endocrine
      - —Hypertension
      - —Diabetes
    - Hematologic
    - Previous parity
  - —Socioeconomic
  - —Environmental factors (eg, high altitude)

Fetal
- Congenital
  - —Genetic
    - Gender
    - Multiple gestation
    - Chromosomal
    - Inborn metabolic errors
    - Genetic syndromes
  - —Malformation
    - Placental
    - Fetal
  - —Nongenetic syndromes
- Acquired
  - —Infectious
    - Viral
      - —Rubella
      - —Human immunodeficiency virus
      - —Cytomegalovirus
    - Bacterial (eg, syphilis)
    - Parasitic
      - —Toxoplasmosis
      - —Malaria
  - —Trauma

are parental genetics and maternal nutritional status. The fetus is relatively resistant to the effects of maternal malnutrition, although chronically undernourished women may give birth to smaller infants primarily because malnutrition has led to decreased maternal prepregnancy height and weight. Placentas of malnourished women have decreased DNA content, consistent with diminished growth during the hyperplasia phase of fetal growth. Fetal effects from acute maternal malnutrition are determined by the point in gestation at which the insult occurs.

The principal cause of asymmetric intrauterine growth retardation is uteroplacental insufficiency, usually due to maternal hypertension, diabetes mellitus, or renal and collagen vascular disease. Fetal factors negatively affecting growth include a variety of known (eg, trisomy) and yet-to-be defined (eg, Russell-Silver syndrome) genetic conditions. As many as 40% of infants who fail to thrive have birthweights of less than 2,500 g. It is unclear why a significant number of small-for-gestational age or intrauterine growth-retarded infants do not experience "catch-up" growth.

The fetal endocrine system is believed to play a relatively minor role in fetal growth. Insulin is likely the major anabolic hormone and works in conjunction with insulin-like growth factors I and II (IGF-I and IGF-II) in promoting fetal growth. In contrast, fetal growth hormone plays a much less significant role in prenatal growth. Indeed, the fetus and infant are relatively resistant to growth hormone, with low concentrations of growth hormone receptors present until the second trimester. In general, infants who have pituitary aplasia or congenital hypopituitarism, as well as those subsequently found to have growth hormone insensitivity syndrome, have normal weights at birth.

Postnatal growth also is influenced by a variety of factors. Normal changes in the rate of linear growth occur frequently during infancy, allowing some infants to "cross percentiles" (up or down) during the first months of life. More than 50% of babies experience an upward shift of growth during the first 3 months of postnatal life. Families (and clinicians) usually are not concerned when the baby experiences a pattern of positive growth, but does a downward shift always represent failure to thrive? Smith et al demonstrated that nearly 30% of normal babies experienced a downward shift between the ages of

3 and 18 months of life; the average age of achieving the new channel of growth was 13 months (Figure).

History Repeats

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of processes that lead to failure to thrive is very broad (Table 2). Historically, the etiologies of failure to thrive were grouped as organic and nonorganic. Organic failure to thrive refers to a major disease process or a single or multiple organ dysfunction. Nonorganic failure to thrive suggests insufficient emotional or physical nurturing without distinct pathophysiologic abnormality; this process has been described as a reactive attachment disorder. Some have suggested that there is mixed failure to thrive, with the dichotomous classification considered obsolete by authors who favor a "transactional model." In this model, the quality of care provided by the caregiver depends on a full spectrum of medical and psychosocial interactions, including economic considerations, social networks, health beliefs, parental/familial health, and family psychosocial dynamics (Table 3). Emphasis is given to the psychologic aspects of the caregivers and the child. Frank and Zeisel observed that "not all children effectively elicit parental care . . . children who become malnourished may be difficult to nurture for many reasons." For example, the child's primary diagnosis (eg, prematurity or a dysmorphism syndrome) may make physical and emotional nourishment difficult. Furthermore, a cycle may develop whereby the irritability or lassitude of the malnourished child leads to further emotional distance between the child and the caregiver. It has been observed that infant temperament and parental perception of the temperament influence interactions between infants and parents. Interestingly, studies have shown that only 35% of couples agree about the clinical interpretation of their infant's temperament (ranging from "easy" to "difficult"). Infants rated as "difficult" by mothers were less responsive, as judged by clinical observers, during feedings. A similar study noted that infants who were judged by parents as "less predictable" in temperament also were rated by observers as being less responsive during feedings.

Children who fail to thrive often experience recurrent infections, which reflects impaired immune status associated with malnutrition. Upper respiratory tract and gastrointestinal illnesses often are associated with anorexia, vomiting, or diarrhea. A cycle may develop that leads to cumulative nutritional deficits, which in turn may predispose to recurrent, and perhaps progressively more severe, infections. Failure to thrive may be a sign of human immunodeficiency virus (HIV) infection. No significant differences have been found in birthweights and lengths of HIV-infected versus noninfected babies, but in the first 6 months of life, HIV-infected infants experience poorer linear growth compared with that of noninfected babies. The degree of viremia correlates with the poor growth.

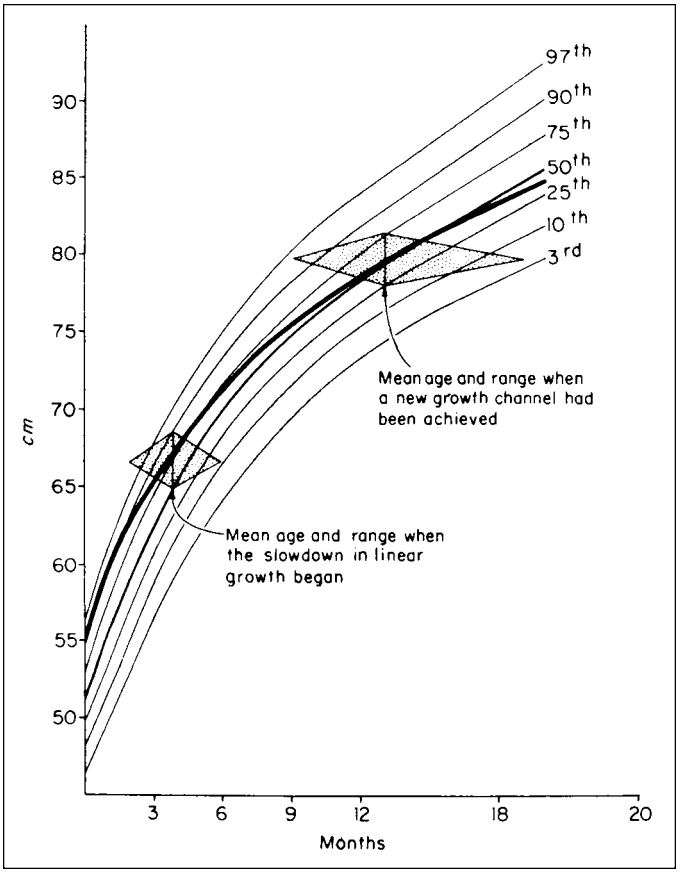

FIGURE. Mean growth curve for 11 male and 5 female infants who were close to the 90th percentile in linear size at birth and near the 40th percentile by 2 years. They are plotted on the growth chart for male infants because the majority were boys. Reprinted with permission from Smith, et al.

DIETARY HISTORY

The dietary history should assess methods of feeding, breastfeeding patterns (including maternal diet and use of medications that can affect milk production and let-down such as alcohol or diuretics), formula preparation, volume consumed, and feeding techniques. Quantitated calorie counts may need to be employed. A detailed history of formula preparation may reveal a dilute formula that contains insufficient calories and excess water.

There may be parental misconceptions about children's caloric needs. Parental restriction of "unhealthy," "sweet," or "fattening" foods or erroneous concerns about "food allergy" have led to failure to thrive. Therefore, parental attitudes and eating habits should be assessed. Provision of fruit juice, rather than

milk, is a common dietary alteration that needs to be addressed. A history of diarrhea, malabsorption, or both can suggest cystic fibrosis, celiac disease, or lactose intolerance. When recurrent pneumonia and chronic diarrhea are associated with failure to thrive, cystic fibrosis is always an important consideration.

Because milk is the only dairy product in the United States that is required to be fortified with vitamin D, a diet history that includes primarily cheese and yogurt may be misleading in terms of calcium and vitamin D intake. Nutritional deficiencies of iron and calcium may enhance absorption of lead and other heavy metals. In the second National Health and Nutrition Examination Survey (NHANES-II), blood lead concentrations in the range of 0.24 to 1.7 mcmol/L (5 to 35 mcg/dL) were correlated with impaired growth. NHANES-III documented a similar inverse correlation between blood lead levels and linear growth and head circumference in children ages 1 to 7 years, although weight and body mass index changes did not correlate.

TABLE 2. Common Causes of Childhood Growth Retardation

- Intrinsic short stature
  - —Genetic
    - Familial
    - Chromosomal abnormalities
      - —Trisomy 13, 18, 21
      - —Chromosome 22 deletions
      - —Gonadal dysgenesis (45,X and variants)
    - Skeletal dysplasia
  - —Intrauterine growth retardation associated with other somatic anomalies
    - Russell-Silver
    - Prader-Willi
    - Cornelia de Lange
- Constitutional delay in growth and development
- Systemic disorders
  - —Failure to thrive
    - Psychosocial
    - Nutritional
      - —Kwashiorkor
      - —Marasmus
      - —Zinc/iron deficiency
  - —Gastrointestinal
    - Feeding disorders
      - —Oral-motor apraxia
      - —Cleft palate
      - —Dentition
    - Vomiting
      - —Gastroesophageal reflux
      - —Structural anomalies
      - —Central nervous system lesion
    - Diarrhea
      - —Chronic toddler diarrhea
      - —Infectious
      - —Malabsorption
        - Cystic fibrosis
        - Celiac disease
        - Inflammatory bowel disease
    - Hepatic
      - —Chronic hepatitis
      - —Glycogen storage disease
  - —Infectious
    - Tuberculosis
    - Human immunodeficiency virus
  - —Cardiac
  - —Pulmonary
    - Tonsillar hypertrophy
    - Cystic fibrosis
  - —Renal
    - Chronic pyelonephritis
    - Fanconi syndrome (and variants)
    - Chronic renal insufficiency
  - —Endocrine
    - In younger children
      - —Hypothyroidism
      - —Rickets
        - Vitamin D deficiency
        - Vitamin D resistance
        - Hypophosphatemic
      - —Growth hormone deficiency/resistance
    - In older children
      - —Hypothyroidism
      - —Growth hormone deficiency/resistance
      - —Hypercortisolism
      - —Pseudohypoparathyroidism
      - —Type I diabetes mellitus (poorly controlled, Mauriac syndrome)
  - —Central nervous system
    - Pituitary insufficiency
    - Diencephalic syndrome
  - —Other chronic diseases and treatment thereof
    - Oncologic

PSYCHOSOCIAL ISSUES

Identifying psychosocial issues is critical in the diagnostic process. Recent data estimate that 20% of all children in the United States live below the poverty level. In 1998, the poverty level was $13,133 for a family consisting of a single parent and two children. It should be emphasized that this is a relatively poor measure of children's well-being because it is limited to assessment of cash income and excludes support programs such as food stamps and housing assistance. However, it is simplistic merely to focus on economic issues when inquiring about psychosocial status. Other considerations include, but are not limited to, marital stress, possible homelessness, domestic violence, parental employment, and parental substance abuse. Children born to mothers younger than 18 years of age have been shown to have poorer growth, especially during the first year of life, than children born to women older than age 18 years. Children of younger mothers were two times as likely to experience maltreatment (mostly neglect) than those born to older mothers.

FAMILY HISTORY

The family history, including stature and growth patterns of siblings, parents, and grandparents, may provide insight about genetic target growth. Information may be available regarding chromosomal or metabolic disorders or other inherited diseases.

PHYSICAL EXAMINATION

The complete physical examination should include accurate measure-

TABLE 3. Psychosocial Considerations for the Child Who Has Failure to Thrive*

- Poverty
  - —Family employment
  - —Nutrition assistance
  - —Lodging
    - Homelessness
    - Shelter
    - Transportation
  - —Health insurance
- Family Dysfunction/ Parent-Child Interaction
  - —Marital stress
  - —Mental illness
  - —Substance abuse
  - —Family history of child abuse
  - —Assessment of feeding
    - Feeding technique
    - Parental dietary attitudes
    - Infant comorbidity
    - Parent-infant temperament
- Child maltreatment
  - —Physical abuse
  - —Intentional/nonintentional neglect

** Data from Frank and Zeisel.*

ments of head circumference, weight, and length (or preferably height after age 3 years) sequentially plotted on the appropriate growth charts to allow comparisons to previous measurements. It is vital to plot these data on the appropriate growth charts. Because we are all "longer than tall," a height measurement graphed erroneously on a length chart may give the false impression that a child is experiencing growth deceleration. Another frequent problem is misplotting of measurements for age.

Several graphs and tables are available for referencing normal growth and height velocity ranges for age and gender. In the United States, growth charts derived from the National Center for Health Statistics are readily accessible and often are considered the "standardized growth charts." However, these charts derive from data published in 1979. Given the epidemic of obesity in the United States, updated charts, which were projected to be distributed in 1998 to 1999, will be welcome. (Note: The Centers for Diseases Control and Prevention announced the release of the revised growth charts on May 30, 2000. They can be downloaded from the Internet at www.cdc.gov/growthcharts.) These revised charts will not be ethnic-specific and will exclude preterm infants weighing less than 1,500 g in the birth to 3-year charts. In addition, the revised charts will incorporate the 3rd to 97th percentiles and extend to age 19 years. For ages 2 to 19 years, charts will be available for stature, weight, and body mass index-for-age. Current charts provide a helpful tool that too frequently is overlooked: weight-for-stature graphs. Plotting these data can help the clinician assess nutritional status.

Interestingly, the revised charts will exclude data for weight and body mass index-for-age collected on children older than 6 years in NHANES-III. The rationale was that NHANES-II and -III have documented a secular trend in weight gain that reflects "environmental influences not believed to be biologically or medically desirable." Including the weight and body mass index data from the NHANES-III children in the revised charts elevated the curves from the 75th percentile and above, thus potentially underidentifying overweight status.

Despite limitations in growth charts, plotting of the data is critical. It is reported commonly that small head circumference implies severe, long-standing malnutrition or primary failure of brain growth/skull development. Similarly, the presence of short stature is said to suggest failure to thrive of long duration. Frank and Zeisel advocate that growth parameters be corrected for gestational age until postnatal age 18 months for head circumference, age 24 months for weight, and age 40 months for stature!

IMPEDIMENTS TO FEEDING

Structural and physical impediments to feeding must be sought. Children who have cleft lip and palate have a higher risk for poor growth. Children who have isolated cleft palates have significantly more problems with feeding (generally nasal regurgitation and vomiting) than do those who have combined lip and cleft defects or those who have isolated lip defects. After early surgical repair, weight approaches average by age 25.5 months. It is important to note, however, that such children are more likely to be short at follow-up evaluations. Children who have cleft palate spectra have been estimated to have 40 times the risk of evolving hypothalamic-pituitary dysfunction.

More subtle anatomic barriers, such as dental caries and abscesses, may interfere with food intake. Tonsillar and adenoidal enlargement often manifest with poor growth. The mechanism is unclear and may relate to mechanical difficulties and to secondary hypoxia. Increased weight gain following tonsillectomy or adenoidectomy has been documented well.

FURTHER EVALUATIONS

In 1988, Frank and Zeisel reported that 3% to 5% of admissions to academic pediatric medical hospitals were for failure to thrive. At this author's regional pediatric referral center, 3.9% of inpatient admissions from November 1998 to October 1999 carried failure to thrive as a discharge diagnosis. Although it may seem pragmatic to begin the evaluation of failure to thrive with a litany of investigations to find the "occult illness" that is inferred in Table 2, Berwick et al demonstrated that the diagnostic yield of hospitalization was not cost-effective. They found that only 0.8% of all tests demonstrated an abnormality that contributed to an underlying diagnosis. On their review of the inpatient records of 122 infants who had unexplained failure to thrive by ages 1 to 35 months, approximately 33% had no diagnosis following the evaluation, 32% had a social or environmental etiology, and 31% were given a specific physiologic or organic diagnosis. Of this latter group, 66% had gastroesophageal reflux or nonspecific diarrhea. The presence of vomiting often was associated with structural or other organic disease. Thus, only approxi-

mately 10% of patients were found to have some "other" underlying, explanatory disease process.

Such findings underscore that results of a careful history and physical examination should be used to guide the diagnostic evaluation, such as stool analysis for malabsorption or sweat testing for cystic fibrosis. Children at risk should be considered for HIV and tuberculin skin testing. A simple determination of serum thyroxine and thyroid-stimulating hormone (if even those) may suffice for the endocrinologic evaluation of failure to thrive, at least in the younger child. The evaluation for growth hormone deficiency is expensive and usually is postponed until other avenues have proven unfruitful or results of the history and physical examination suggest otherwise, such as a history of hypoglycemia or central nervous system infection or trauma or the finding of midline defects, including microphallus in boys. The special testing required to assess pituitary growth hormone secretory capacity is best performed following referral to an endocrinology clinic. For the malnourished child, random determinations of IGF-I, as a reflection of the growth hormone axis, usually are not helpful because the values almost invariably are low.

Nostradamus—Predicting Psychosocial/Developmental Outcome

In a 1988 study from Case Western Reserve, cognitive development was assessed by Stanford-Binet intelligence quotient (IQ) scores at 36 months of age in children who had a prior history of nonorganic failure to thrive. The mean IQ score was 85.4, with a standard deviation of 15 and a range of 57 to 132. In an Israeli study, intellectual and educational achievement was found to differ by age 5 years among children who had failure to thrive compared with controls. Approximately 11.5% of children previously identified as failing to thrive had some degree of developmental delay compared with none of the control children, and 18% had poor scholastic performance compared with 3.3% of controls. School performance was correlated positively with higher anthropometric scores. Other positive correlations with cognitive development included maternal socioeconomic level and number of persons per room in the home.

Stratifying by social class, the Wessex Growth Study showed that teachers' comparisons of short-normal children and control children indicated decreased language, number, gross motor, and fine motor skills at the time of school entry among those who had a history of failure to thrive. A follow-up study to the Case Western investigation demonstrated that even children who had prior nonorganic failure to thrive and participated in early intervention programs demonstrated deficiencies in personality development and problem solving and had more behavioral problems compared with controls. Behavioral problems included less ability to control impulses, delay gratification, or change behavior in response to new situations.

A University of Maryland study showed a cumulative detrimental effect of failure to thrive (growth failure) and social neglect on mean Mental Developmental Index scores in infants and toddlers compared with those suffering only failure to thrive or neglect or neither (85.5 versus 97.4, 97.7, 110.8, respectively). These types of studies suggest that the Denver Developmental Screening Test may be inadequate for proper assessment of development in infants who fail to thrive.

Becoming Y2K Compliant—Intervention and Management

FEEDING

Whenever possible, the underlying cause of failure to thrive should be addressed and treated. However, the major contributor to failure to thrive is caloric inadequacy. Daily caloric requirements (kcal/kg) for catch-up growth have been estimated as:

$$120 \text{ kcal/kg} \times \frac{\text{median weight (kg)}}{\text{current weight (kg)}}$$

The median ("average") weight relative to the patient's height is determined from reference tables or graphs and divided by the patient's current weight, with the resulting value multiplied by 120. This product is the number of calories per kilogram that are estimated to be required to achieve catch-up growth. By this estimation, most children will require 1.5 to 2 times the expected intake for age to achieve optimal catch-up growth. Protein intake must be enriched proportionally, and micronutrient needs, including iron and zinc, addressed. The latter is gaining wider acceptance, especially globally, because supplements of 25 mg of zinc sulfate have been shown to enhance growth. A meta-analysis has demonstrated the positive effects of zinc in promoting linear growth and weight gain in growth-retarded children. As a practical matter, it is difficult to measure total body zinc stores and losses, and plasma levels may be misleading, so the author often supplements infants who have failure to thrive empirically with up to 25 mg of zinc daily, especially if there is a history of recurrent diarrhea or pneumonia. Because zinc, per se, has no direct pharmacologic effect on growth, a positive effect on growth following zinc supplementation suggests a pre-existing, growth-limiting zinc deficiency. Vitamin D may be required if there is biochemical or radiographic evidence of rickets.

Certainly it is not reasonable to expect a child to eat twice the usual amount of food. Appetite-enhancing medications, such as cyproheptadine, have been advocated by some, but results have been variable. Therefore, the child's diet must be fortified for caloric density by concentrating formula or by adding glucose polymers or extra lipids. All of these maneuvers should be performed in the outpatient setting. Enteral tube feedings or gastrostomy should be considered for patients in whom oral enteral feedings do not achieve adequate caloric intake, although these interventions, unfortunately, often are considered only for preambulatory patients or those who have significant developmental diagnoses (eg, cerebral palsy, chromosomal

abnormality, perinatal infection). Studies in children up to age 6.5 years show significant catch-up growth in weight and height within 12 to 18 months following gastrostomy virtually regardless of age, although the most rapid catch-up growth occurs in children older than age 2 years. Depending on the child's diagnosis, parents may be resistant to the use of these procedures.

HOSPITALIZATION

Hospitalization for failure to thrive remains the mainstay of management if the child is refractory to the previously mentioned outpatient procedures. As suggested, hospitalization has little impact (other than increasing costs) on the diagnostic categorization of failure to thrive. However, management may be enhanced during a hospitalization by providing a more controlled environment to assess caloric intake, feeding techniques, and parent-child interactions. Hospitalization of an infant who has failure to thrive allows timely input from important ancillary health-care staff, including nutritionists, social workers, occupational and physical therapists, therapeutic recreation workers ("child life specialists"), behavioral and developmental specialists and psychologists, and bedside nurses. Indeed, a formal team approach that includes these professionals and orchestrates home visits has been shown to be successful in promoting growth and in achieving higher scores on motor and cognitive development. Greater benefit has been shown for children and families receiving in-home intervention modalities compared with solely clinic interventions. Disappointingly, not all studies reporting on intervention have shown beneficial effects on childhood behavioral problems, problem-solving skills, or cognitive development scores. Outpatient team approaches have been shown to decrease the frequency of hospitalization.

A hospitalization of 10 to 14 days or greater with adequate caloric intake commonly is believed to be sufficient to demonstrate appropriate weight gain. Rarely do affected children do worse in the hospital. Those who have organic failure to thrive and nonorganic failure to thrive have been shown to gain similarly during controlled hospitalizations, which reiterates the dubious distinction between these diagnostic categories and underscores the fallacy that weight gain in the hospitalized setting denotes the presence of nonorganic failure to thrive. During the hospitalization, it is prudent to limit solid foods that have less caloric density than milk or formula in the appropriate-age infant.

Meta-analysis has highlighted the efficacy of hospitalization on physical growth in those who have nonorganic failure to thrive, with an approximate doubling of the probability of catch-up growth, but suggests only modest psychosocial strides. Unfortunately, it has become clear in today's medical environment that third-party payers are reluctant, if not sometimes refusing, to authorize hospitalization for the evaluation and treatment of failure to thrive. Perhaps if practitioners scrutinized their diagnostic evaluation of such children or performed key diagnostic studies on an outpatient basis, the hospitalization policies would be liberalized. Pediatric clinicians must be children's advocates and hospitalize for failure to thrive as warranted.

Summary

The most correct definition of failure to thrive is difficult to determine. Although the nomenclature of organic versus nonorganic failure to thrive is helpful, the practical therapeutic distinctions may be blurred, especially when considering the complexities of the broad psychosocial applications.

Generally, children who fail to thrive have nutritional inadequacies. The history and physical examination initially should focus on these problems, with special attention to feeding disorders and vomiting. Review of specific dietary practices, formula preparation, and feeding techniques, including caregiver/child interaction, is imperative. It is cost-effective and appropriate to limit the laboratory evaluation, but it is reasonable to consider diagnostic studies that have further implications for health, such as sweat chloride determination, blood lead level, or parameters of immune function, or to employ other special testing (eg, tuberculin skin testing or HIV status) in children at risk. Results of the history may guide the selection of further studies such as stool analysis for malabsorption and ova/parasites, metabolic profiles, and radiographic procedures. Psychological and developmental assessment should be undertaken once any acute medical condition has been identified and addressed.

Aggressive nutritional intervention may include caloric and micronutrient (including zinc) supplementation as well as tube feedings or gastrostomies. The value of the team approach, employing the assistance of nutritionists, psychologists, and home health workers, cannot be overemphasized.

The new millennium imagines continued scientific breakthroughs in diagnostic testing, insights into the human genome, and treatments for rare diseases. Failure to thrive has been present since the dawn of humans, and it is a disorder that deserves extinction.

SUGGESTED READING

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PIR QUIZ

Quiz also available online at www.pedsinreview.org.

1. You are evaluating a 4-month-old infant whose weight is below the 3rd percentile. The presence of which of the following *most* suggests an organic cause for his malnutrition?
   A. Birthweight at the 25th percentile.
   B. History of persistently loose stools.
   C. Low socioeconomic status.
   D. Maternal age of 17 years.
   E. Maternal perception of a difficult temperament.

2. A young mother expresses concern that her 5-month-old daughter does not seem to be gaining weight appropriately. The mother seems to be apprehensive when she gives the baby her bottle. You begin to take a detailed history to determine the cause for the infant's poor weight gain. Of the following, you are *most* likely to focus on:
   A. History of chronic diseases in other family members.
   B. Level of maternal education.
   C. Method of formula preparation and feeding techniques.
   D. Prenatal complications.
   E. Presence of fever in the infant.

3. You are seeing a 2-month-old infant for the first time. The mother explains that she has four other children and that she has little family support. You suspect that the mother is caring but overwhelmed by her responsibilities, and you note that she is diluting her formula too much in an effort to "stretch" her dollar. The interaction between the infant and mother seems appropriate. Physical examination reveals a thin child who has little subcutaneous fat, but no other remarkable findings. Her weight is below the 3rd percentile, and her height and head circumference are at the 10th percentile. Your *next* step is to:
   A. Admit the child to the hospital for a comprehensive evaluation.
   B. Advise the mother to add fruit juices to the baby's diet.
   C. Educate the mother about proper formula preparation and begin a trial of outpatient observation.
   D. Recommend placement of a gastrostomy tube.
   E. Report the mother to child protection services.

4. A *true* statement about infants who fail to thrive is that:
   A. An underlying physiologic diagnosis can be identified in the majority of cases.
   B. Appropriate weight gain during hospitalization is proof of a nonorganic etiology.
   C. Developmental delay can be prevented with early intervention.
   D. Most infants who have organic failure to thrive are found to have a metabolic disorder.
   E. Recurrent infections are common.

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