PEDIATRIC DENTISTRY/Copyright © 1990 by
The American Academy of Pediatric Dentistry
Volume 12, Number 3

Failure to thrive: review of the literature, case reports, and implications for dental treatment

Craig E. Elice, DDS Henry W. Fields, DDS, MS, MSD

Abstract

The symptom failure to thrive (FTT) is increasing in the general population, and has a prevalence of 9.6% among infants in the United States. Other factors occur in association with growth failure such as medical and dental anomalies and developmental delays that influence the management of these children. Four cases are presented to demonstrate the spectrum of problems and interdisciplinary management required to provide successful dental treatment.

Introduction

Failure to thrive (FTT) is an increasing symptom in our population because more infants with serious medical problems are surviving the pre- and postnatal period and because more well-intentioned parents are feeding their infants an inappropriate, restrictive diet of questionable nutritional value. Mitchell et al. (1980) reported a 9.6% prevalence of growth failure among infants. Up to 5% of all pediatric hospital admissions may be related directly to failure to thrive (Levine 1978). The purpose of this paper is to provide a literature review and present a series of case reports to illustrate problems these patients can present to the pediatric dentist.

Review of Literature

The symptom FTT was described in the textbook *Diseases of Infancy and Childhood* (Holt 1899) and traditionally has been reserved for infants and toddlers younger than 3 years of age. Older children with similar symptoms are labelled as "failure to grow" or "short stature" (Reinhardt 1979). By definition, FTT consists of:

1. Weight or height below the third percentile for age
2. Weight less than 80% of ideal weight for age
3. Failure to maintain a previously established growth pattern; and/or
4. Growth failure of unknown origin (Accardo 1982).

This definition has shortcomings because racial, environmental, and hereditary differences are not addressed in comparative growth charts. No single measure accurately characterizes the degree of growth failure.

For these reasons, a complete medical examination, social and family history, and midparental height (average for both parents) are required for an accurate evaluation (Smith et al. 1976; Levine 1978; Accardo 1982). Three growth descriptors — height, weight, and head circumference — help estimate the timing of the etiologic insult and the effects of treatment (Lubchenco 1976). Many times, these data are available, but have not been plotted prior to referral to a tertiary care facility.

The disease processes associated with FTT occur pre- or postnatally and can involve most pediatric diseases (Riley et al. 1968; Beck and van den Berg 1975; Lubchenco 1976). Several authors (Hannaway et al. 1970; Sills 1978; Homer and Ludwig 1981) described FTT as organic (18–50% of cases), nonorganic (35–60% of cases), or mixed (0–25% of cases).

Organic FTT requires identification of an associated organic disease or other condition, including: neurologic (7–18%), gastroenterologic (8–14%), or endocrine (2–5%) disorders; malignancy (1%); congenital heart (2–4%), renal (2–5%), or hepatic (2–4%) diseases; chromosomal abnormalities (2%); HIV (1%) or intrauterine (2%) infections; or dental caries (Sills 1978; Hannaway et al. 1980; Accardo 1982).

Nonorganic FTT refers to the exclusion of an organic etiology and findings consistent with an adverse environment, such as: inadequate maternal information in caring for the infant, disturbance in attachment (4%), and problems in separation/individuation which reflect abnormal infant behavior (25%) (Egan et al. 1978; Cupoli 1980).

Homer and Ludwig (1981) defined mixed FTT as patients with both organic and nonorganic causes. Gordon and Vasquez (1984) subdivided mixed FTT into three different types. The first involved an adverse environment that precipitated organic symptoms. The

second type required a diseased infant and an adverse environment that magnified growth failure. In the third type, the affected infant caused an adverse environment (e.g.: negative parental reactions).

Although description of the etiologies of FTT appears straightforward, clinicians often confuse causative agents with environments. Case histories that involve multiple medical problems amplify this point.

Case Reports

The following case reports illustrate some of the medical and environmental problems associated with FTT and the problems that occur when managing these patients' dental needs. In all cases, the chief complaint, past medical history, etiology of FTT, and dental treatment are described.

Case 1

This 7-year-old black male was referred from a pediatric endocrinology clinic for treatment of extensive dental caries believed to be contributing to his FTT. Hypopituitarism and hypothyroidism were diagnosed at 4 years of age. The child had frequent upper respiratory infections and was experiencing separation difficulties, behavior problems at school, and crowded living conditions at home. This combination was classified as mixed etiology FTT.

The growth charts (Figs 1 and 2) illustrated that between ages 4 and 5 years growth was below the third percentile. After growth hormone was implemented, both height and weight velocity increased and approached the third percentile.

A number of factors were significant for this child's dental treatment. He had nursing bottle caries, defiant behavior, and poor motivation. The patient's inappropriate behavior was managed using conscious sedation and restraint. His inadequate oral hygiene practices and cariogenic dietary habits were addressed with his parents during restorative visits. The restorative care included placement of stainless steel crowns and alloys on the posterior teeth and extraction of the primary anterior teeth. Diet counseling measures and oral hygiene instruction were coordinated with the referring pediatricians. The patient was lost to follow up because of poor compliance with dental appointments.

Case 2

This 3 year, 11 month black female was referred by a pediatrics clinic because of enamel hypoplasia. She was a 3 kg product of a 41-week gestation period and was delivered by Cesarean section because of fetal distress. The patient had seizures at 8 days of age. Subsequently, the child was diagnosed with hepatomegaly, pulmonic stenosis requiring SBE precautions, sleep apnea, and developmental delays. A karyotype was performed because of her dysmorphic features, which revealed an extra autosomal segment on the short arm of her X chromosome. This confirmed the diagnosis of a chromosomal abnormality (46XXP+) and FTT of organic etiology.

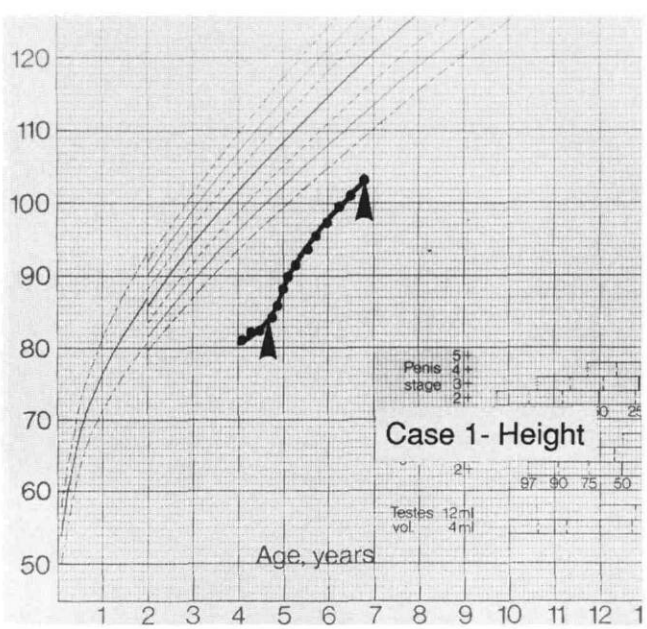

Fig 1. This patient with growth hormone deficiency was below the third percentile (lowest curve plotted) and deviated to lower percentiles an increasing amount between 4 and 5 years of age. Arrows indicate the time span of growth hormone therapy during which the curve progressed toward the third percentile.

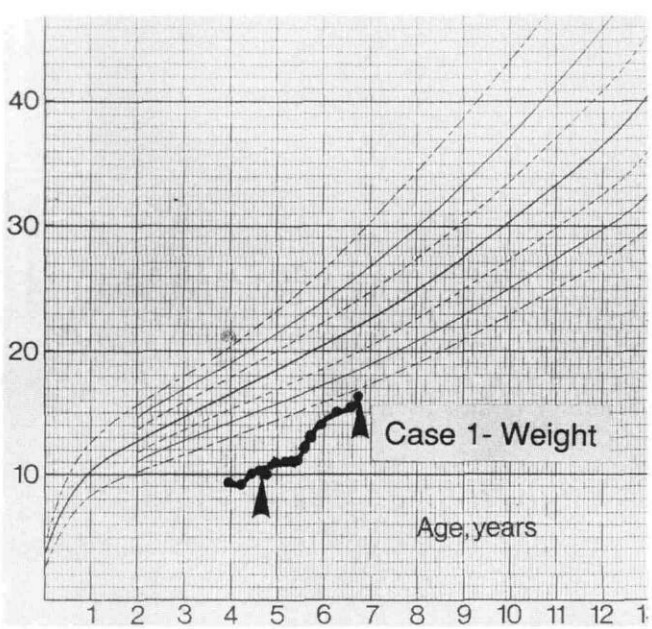

Fig 2. This patient with growth hormone deficiency was below the third percentile (lowest curve plotted). Following growth hormone therapy (indicated by arrows), the child's weight approximated the third percentile.

Growth data indicated that the child did not grow in height between 1 year, 11 months and 2 years, 4 months, which may have been related to either chronic illness or the chromosomal abnormality. Her weight gain during the same period was very low.

Because of the multiple dental lesions, inability to communicate, and multiple medical problems, the dental treatment was performed under general anesthesia. Her hypoplastic carious teeth were susceptible to recurrent decay; therefore, all of her posterior teeth were restored with stainless steel crowns, and the anterior teeth were extracted. Preventive oral health measures were instituted and maintained successfully by the parents. In the two years since the patient's initial exam, she has continued to grow at a velocity below the third percentile.

Case 3

This patient was an 11-year, 10-month-old white male referred by a pediatric endocrinology clinic for an orthodontic evaluation. At 3 years of age the patient was admitted to the hospital because of right knee pain that was diagnosed subsequently as atypical nonHodgkins lymphoma. As a result, the patient received CNS radiation and chemotherapy. At 10 years, 2 months a testicular biopsy indicated malignancy, and an orchiectomy was performed. At that time, an indwelling catheter was placed, and chemotherapy and local radiation were reinstituted.

During radiation therapy, growth curves deviated across percentiles, and the height curve shifted from the seventeenth percentile at 3 years of age to below the third percentile at 7 years of age. Pediatric endocrinology subsequently made a diagnosis of organic failure to grow secondary to cranial radiation. At 9 years of age, growth hormone therapy was instituted for 3 months with no effect. Chemotherapy following diagnosis of the testicular lesion probably was responsible for a second shift to below the third percentile in stature. Growth hormone was reimplemented at 11 years, 10 months, and his growth rate improved. Predictably, the growth hormone therapy had an even greater effect on weight.

Because of the patient's severe malocclusion, he was referred for an orthodontic evaluation at 11 years, 10 months, and was monitored for facial growth changes during the next 18 months. The lateral cephalometric radiographs obtained at 11 years, 10 months and 12 years, 8 months were superimposed and demonstrated downward and forward growth with reasonable velocity, but not a velocity usually expected during the adolescent growth spurt. Orthodontic treatment for growth modification was begun based on the sustained positive growth velocity.

Case 4

This 3-year, 4-month-old white male was referred by pediatric endocrinology because of extensive dental caries suspected of contributing to his FTT. His past medical history indicated only normal childhood diseases. No family history for short stature was apparent, and the patient's older brother was of normal stature.

Initially, the patient was referred to the hospital because of loss of appetite at 1 year, 10 months. A physical examination, blood test, and endocrine screen were negative for organic disease, and subsequently the child was placed on a frequent follow-up schedule. Later, bone age was determined radiographically to be at 2 years of age, despite his 3 year, 6 month chronological age. Nonorganic FTT was the etiologic classification based on exclusion of organic disease.

Up to 4 months of age, this patient maintained growth at the fiftieth percentile. Beginning at 6 months of age, growth curves deviated to the twenty-fifth percentile. The pediatrician suspected juvenile diabetes, but a glucose test ruled out diabetes as a cause of deceleration in growth. Between 1 year and 2 years, 6 months, the patient demonstrated almost no weight gain. At 2 years of age, he began growth hormone therapy, and his stature was maintained at the third percentile while his weight showed a more dramatic improvement with the growth hormone therapy.

The child was referred to the pediatric dental clinic because the physicians suspected a relationship between the dental caries and the child's FTT. A diet analysis indicated that the child was breast fed for 22 months with limited solid food supplementation. The child ate snack foods throughout the day, but refused to eat at meal time when balanced meals were served. In this case, the nonorganic etiology may relate to the patient's refusal to eat and lack of parental understanding of proper dietary management. The pediatric endocrinologists counselled the parents on diet modification and behavior management to improve the child's eating habits. A dental examination revealed moderate nursing bottle caries with incipient decay on the posterior teeth. Although normal motor and mental development were achieved, he required chloral hydrate and hydroxyzine pamoate sedation for his dental treatment because of his inability to cooperate. The child received fluoride supplementation and was placed on a three-month recall. Despite instituting preventive measures, the conservative alloys required replacement with stainless steel crowns due to recurrent decay. In the 18 months after his first visit, the patient's growth velocity accelerated to approach the third percentile. Whether this growth acceleration was due to growth hormone therapy, improved diet, or dental treatment is not clear.

Discussion

The prevalence of children who fail to thrive is remarkably high. Because several diseases in children, especially HIV and related infections, are rising at an extraordinary rate, FTT will contribute more to medical, psychological, and developmental problems than it has in the past and will influence the appropriate management of these children's dental needs. Although the symptom FTT does not cause disease, it contributes to the severity.

Pediatric dentists treat many severely ill and debilitated patients; therefore, these patients will become more prominent in specialty practice. Our responsibility is to recognize the many types of etiologic factors that can be active, including dental caries, and be prepared to treat these children in an interdisciplinary manner.

Treatment between medical and dental specialties needs to be coordinated to meet a patient's complex needs. Cases 3 and 4 illustrate this fact in several ways. The third patient needed an evaluation of the present malocclusion and a determination of whether dentofacial growth modification was feasible. Records obtained by the endocrinologist were not useful in determining the effects of growth hormone on the craniofacial complex. Facial growth can be gauged accurately only by evaluating facial structures. When growth hormone therapy is judged successful for the patient's somatic and dentofacial problems, it can be continued and orthodontic treatment instituted. One health care discipline alone cannot evaluate adequately the state of the patient's complete health status.

Multidisciplinary care also is required for patients with inappropriate dietary habits. Dietary counselling and intervention by the pediatric dentist alone can lead to problems with behavior modification regimes and nutritional criteria set by the physician and dietitian. For example, high-calorie snack foods can be appropriate and even part of an initial reinforcement schedule for some children. Conflicting, or more often confusing, information can undermine or stop progress. Adjustment can be made in medical and dental treatment regimens to work toward mutually satisfactory goals as treatment progresses.

Treatment can alter the child's status dramatically. Appropriate cardiac surgery, a transplant, counselling, medication, or better nutrition can alter the child's physical and psychological status dramatically. In these cases, the practitioner must be able to evaluate these changes and alter treatment methods as needed. This will require additional interdisciplinary communication.

Because of their low developmental level, children whose chronological age would ordinarily dictate treatment by conventional behavior management means (e.g.: tell-show-do and positive reinforcement) often require more extraordinary means (i.e. conscious sedation, restraint, or general anesthesia). Some of these children may be older than patients who normally might require restraint or sedation and/or require modification of drug regimens and restraining devices and techniques. Sometimes the older child simply is too strong or large to handle safely in an outpatient setting.

The first case demonstrated (as did the fourth) that severe dental decay can be a contributing etiologic agent in FTT. Both children were referred for treatment of severe dental caries that can inhibit adequate nutrition. Often it is overlooked that teeth, although not necessary for adequate nutrition, make efficient, pleasurable nutrition possible. Oral pain and discomfort, on the other hand, can reduce nutritional and fluid intake to dangerous levels. Data from a recent survey revealed that children with chronic malnutrition and stunted growth have a higher caries prevalence, suggesting a relationship to FTT (Alverez 1988).

Reliable growth records can help identify children who are suffering from FTT. Most often these data need to be plotted to be meaningful. Growth charts are available and are illustrated in contemporary pediatric dentistry texts (Casamassimo et al. 1988). Because the pediatric dentist usually maintains an aggressive recall schedule, this type of monitoring can be accomplished more readily in the dental setting at the semiannual (or more frequent) recalls than by the physician.

Conclusions

The symptom of FTT is recognized in 9.6% of the pediatric population, and given, the dramatic rise in some of the diseases contributing to FTT, can be expected to rise in prevalence.

1. Pediatric dentists should expect to encounter these medically or developmentally impaired patients.
2. Developmental delays resulting from FTT may require special behavior management techniques including restraint, sedation, or general anesthesia.
3. Medical and dental treatment must be coordinated for routine restorative care or orthodontic growth modification because of the complex problems these patients have and their changing treatment situations.
4. Dental caries can be a symptom or etiology of FTT.
5. Height and weight should be recorded periodically during dental exams as an adjunct to identifying or confirming the symptom FTT.

The authors acknowledge the review of this manuscript by Drs. F.T. McIver and W.F. Vann, Jr. and the assistance in manuscript preparation of Ms. Carolyn Gravely.

Dr. Elice is a resident in the department of pediatric dentistry at the School of Dentistry, University of North Carolina, Chapel Hill. Dr. Fields is assistant dean for academic affairs and professor in the departments of pediatric dentistry and orthodontics, also at the University. Reprint requests should be sent to: Dr. Craig E. Elice, Dept. of Pediatric Dentistry, School of Dentistry, University of North Carolina, CB 7450, 205 Brauer Hall, Chapel Hill, NC 27599-7450.

Accardo PJ: Failure to Thrive in Infancy and Early Childhood: A Multidisciplinary Team Approach. Baltimore: University Park Press, 1982, p 119.

Alverez JO, Lewis CA, Seman C et al: Chronic malnutrition, dental caries, and tooth exfoliation in Peruvian children aged 3–9 years. Am J Clin Nutr 48:368-72, 1988.

Beck GJ, van den Berg BJ: The relationship of the rate of intrauterine growth of low birth weight infants to later growth. J Pediatr 86:504-11, 1975.

Casamassimo P, Christensen J, Fields H: Examination, diagnosis, and treatment planning for general and orthodontic problems, in Pediatric Dentistry: Infancy Through Adolescence. Pinkham JR, Casamassimo PS, Fields HW, McTigue DJ, Nowak AJ, eds. Philadelphia: WB Saunders, 1988, pp 463-66.

Cupoli JM, Hillock JA, Barness LA: Failure to thrive. Curr Probl Pediatr 10:1-41, 1980.

Egan J, Chatoor I, Rosen G: Nonorganic failure to thrive: pathogenesis and classification. Clin Proc Child Hosp NMC 36:173-81, 1980.

Gordon EF, Vasquez DM: Failure to thrive: an expanded conceptual model, in New Directions in Failure to Thrive: Implications for Research and Practice. Drotar D, ed. New York: Plenum Press, 1984, pp 69-77.

Hannaway PJ: Failure to thrive: a study of 100 infants and children. Clin Pediatr 9:96-99, 1970.

Holt LE: The Diseases of Infancy and Childhood. (1899) Reprinted 1980. New York: D Appleton and Co., 1980, p 661.

Homer C, Ludwig S: Categorization of etiology of failure to thrive. Am J Dis Child 135:848-51, 1981.

Levine MI: Failure to thrive: an introduction. Pediatr Ann 7:737-42, 1978.

Lubchenco LO: The High Risk Infant. Philadelphia: WB Saunders Co, 1976, pp 125-51, 233-81.

Mitchell WG, Gorrell BW, Greenberg BA: Failure to thrive: a study in a primary care setting. Pediatrics 65:971-77, 1980.

Reinhardt JB: Disturbances of development, in Basic Handbook of Child Psychiatry, Vol. II. Joseph D. Noshpitz, ed. New York: Basic Books, 1979, pp 593-99.

Riley RL, Landwirth J, Kaplan SA: Failure to thrive: an analysis of 83 cases. Calif Med 108:32-38, 1968.

Sills RH: Failure to thrive: the role of clinical and laboratory evaluation. Am J Dis Child 132:967-69, 1978.

Smith DW et al: Shifting linear growth during infancy. Illustration of genetic factors from fetal life through infancy. J Pediatr 89:225-30, 1976.

Older female teens more likely than male teens to be HIV-infected

Although U.S. teenagers rarely have AIDS, their rate of HIV infection may be higher than previously thought, warns a study that appeared in the April 18, 1990 issue of the *Journal of the American Medical Association*.

About one of every 3,000 teenaged applicants for U.S. military service tests positive for the human immunodeficiency virus (HIV), according to the authors, Col. Donald S. Burke, MC, USA, of the Division of Retrovirology, Walter Reed Army Medical Center, Washington, DC, and colleagues. Older female teens (aged 17 and 18) are more likely to have been infected with HIV than their male counterparts, whereas adolescent male AIDS cases outnumber female cases by a four to one margin, they said.

"Adolescents typically have little understanding regarding risks of infection with HIV," the authors wrote. "The data presented in this study suggest that HIV is a real and immediate threat to teenagers throughout the United States."

Since October 1985, all applicants for U.S. military service have been tested for HIV. Between October 15, 1985 and March 31, 1989, more than 1.1 million teenagers applied for military service. Of these, 393 (345 males, 48 females) were found to be seropositive. The prevalence of HIV among black teenagers (1.06 per 1,000 applicants) was greater than among whites (0.18 per 1,000) or Hispanics (0.31 per 1,000), the researchers found.

The rate of HIV seropositivity varied markedly in different geographic locales. The most populous states had the greatest number of infected teenagers (California, 52; New York, 45; and Texas, 38). Throughout the north-central states, there was less than 0.1 per 1,000 applicants with HIV, compared with 2.0 per 1,000 in urban counties of Maryland, Texas, New York, and the District of Columbia.

The overall prevalence of HIV infection was similar among teenaged males and females, although

continued on page 203