The Eating Disorders
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Chapter 25

Eating Disorders in Infancy and Early Childhood

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INTRODUCTION

Eating disorders in infancy and early childhood have a number of unique characteristics that have inhibited our progress in understanding and diagnosing them. For example, their dyadic character has long been underemphasized [1]. To correct for this, pediatric textbooks refer to "feeding disoders" rather than "eating disorders of infancy." Another unique and confusing problem of the study of eating disorders of infancy is the linkage between eating disorders and growth disorders. At no other time in life will an aberration in eating have such a profound effect on growth and development. A third set of problems involves ethical and technical issues related to studying infants and young children. Informed consent, invasive procedures, prolonged hospitalizations, and blind research designs are all much more difficult with infants than with adolescents or adults. A fourth area of problems results from a persistent diagnostic confusion. The diagnostic blurring that has plagued investigation of eating disorders in infancy has greatly hindered meaningful research. For example, until recently no effort was made to distinguish nonorganic failure to thrive (NFTT) from psychosocial dwarfism (PSD), to separate rumination from gastroesophageal reflux (GER), or to delineate the relationship among NFTT, rumination, psychophysiological vomiting, and GER [1].

The *Diagnostic and Statistical Manual of Mental Disorders,* Third Edition (DSM-III) [2] exemplifies this confusion. It lists pica and rumination as the only eating disorders for this age-group. Obesity is considered a physical disorder. NFTT is described primarily as a problem of mother-infant attachment, and psychosocial dwarfism is not listed as a diagnosis.

A major contribution to this diagnostic confusion results from the multifactorial nature of eating disorders in this age-group. Virtually all of these disorders represent a final common pathway of diverse contributions at all levels of the human experience (figure 1). These contributions frequently include genetics, congenital vulnerabilies, intrapsychic disturbances between infant and parent, family dysfunction, and social stressors. In turn, the resultant eating disorder may well exacerbate some or all of the initiating problems, as well as cause new and even more maladaptive disturbances. Eating and growth disorders represent a closed system in which there is feedback between each "unit" of the system.

Although this conceptualization would seem to make any sort of diagnostic categorization hopeless, such nihilism is unwarranted. Rather, this systems approach to eating disorders should be kept in mind so that researchers and clinicians alike avoid becoming myopic in their search for "the cause" of eating disorders.

There are a number of different approaches to categorizing eating disorders of infancy and early childhood. Anna Freud [3] suggested one based on presumed theoretical etiology. She proposed that they could be divided into organic feeding disturbances, nonorganic disturbances of the instinctive process, and neurotic

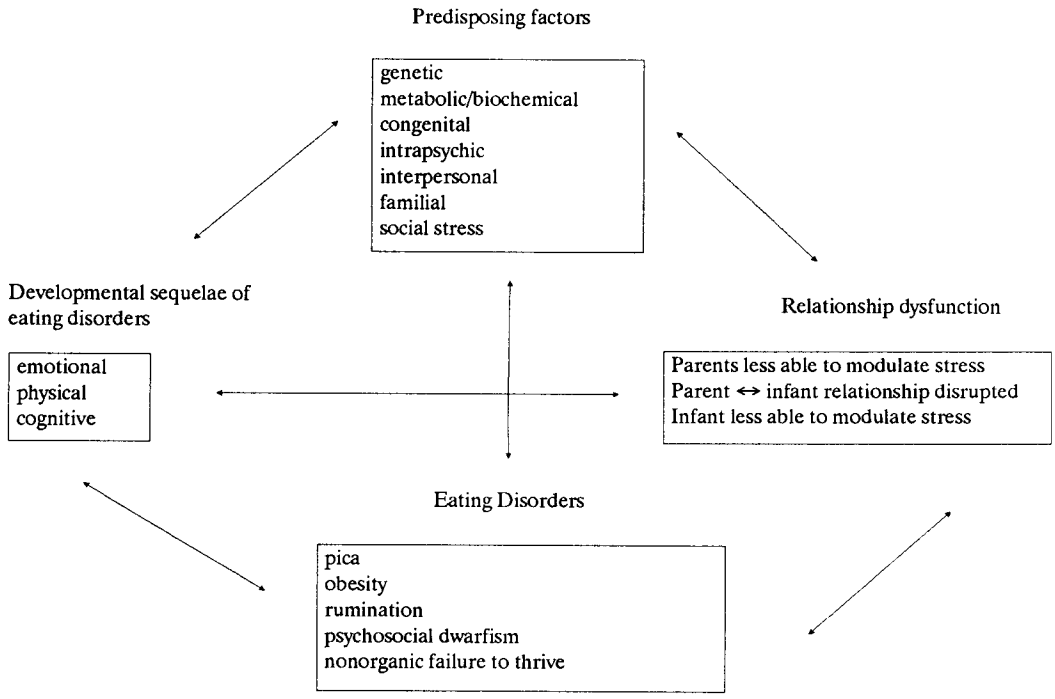

Figure 25.1 Virtually all of these disorders represent a final common pathway of diverse contributions at all levels of the human experience.

feeding disturbances. Unfortunately, so little is known about eating disorders of this age-group that a phenomenological, rather than etiological, classification seems a necessary first step. One such schema would be to differentiate those eating disorders that result in intake of nonnutritive substances, from those that result in excessive weight gain, from those that result in insufficient weight gain and growth [1]. Using this schema, pica would be in the first group, simple obesity in the second, and nonorganic failure to thrive, rumination, and psychosocial dwarfism in the third group.

SPECIFIC SYNDROMES

Disorder of Nonnutritive Substances: Pica

The eating disorder of inappropriate food choice is represented by the syndrome of pica or geophagia. The DSM-III [2] defines pica as the persistent eating of nonnutritive substances such as paint, plaster, clay, and leaves. Age of onset is between 12 and 24 months. Pica usually remits in early childhood, although it may persist into adolescence. Little is known about the cause or the natural history of pica, although the common complications of lead poisoning and intestinal obstruction are well publicized. Pica appears to be a syndrome that is the final common pathway of multiple interacting variables [1]. Wortis et al [4] reported that children manifesting pica tended to suffer more neonatal insults, to be slower in motor and mental development, to show more neurological defects, and to have more deviant behavior both before and at the time of the development of pica. Millican et al [5] examined pica from a psychoanalytic point of view. They proposed that pica might result from an identification with the mother's pica, the maternal fostering of oral activity as a defense against anxiety, or a regression of the oral stage in the face of frustration and deprivation. Although accidental poison ingestion is not pica, there seems to be a strong linkage between the two phenomena. Millican et al [5] reported that pica was found in 55% of children hospitalized for such accidental poisonings. Unfortunately, the existing studies are marred by methodological problems, so that understanding of the syndrome remains tentative. The differential diagnosis of pica includes vitamin and/or mineral deficiencies, infantile autism, mental retardation of a variety of etiologies, and certain physical disorders such as Klein-Levin syndrome (periodic somnolence, morbid hunger, and motor unrest) and Lesch-Nyhan syndrome (inherited abnormality or purine metabolism with associated mental retar-

dation movement disorders and compulsive automutilation).

Research into the causes, prevention, or treatment of pica is still in its most rudimentary phases. No new research articles have appeared in the literature in the last 15 years. Obviously, basic phenomenological and epidemiological work must occur before more definitive research can proceed [1].

Disorder of Excessive Weight Gain: Simple Obesity

Obesity is the syndrome characterized by an excessive deposition and storage of fat. There are a variety of research definitions that use weight for age, weight for height, and skinfold thickness as arbitrary inclusionary criteria. One commonly accepted criterion is for body weight to exceed ideal body weight for height by 20% or more [6].

The prevalence of simple obesity is between 3% and 20%, depending on the group examined and exact definitions, methods, and standards used. Best estimates are that 5% to 10% of preschool-age children, 10% of schoolage children, 15% of adolescents, and 30% of American adults are obese [6]. There is a strong correlation between parental and sibling obesity and obesity of index child. Girls are about twice as likely as boys to be obese.

Like pica, simple obesity of infancy and early childhood is a complex syndrome, which is the final common pathway of the interplay of multiple factors, including genetics, prenatal experience, familial and cultural practices, emotional factors, activity levels, etc [1]. Perhaps because of the multiplicity of factors that contribute to obesity, the literature about etiology, prognosis, prevention, and treatment is contradictory [7-9]. The relative contributions of such basic variables as caloric intake, physical activity levels, and metabolic efficiency remain obscure. The role of such specific factors as cellularity of adipose tissue also remains controversial [10-13]. One issue that is quite clear is that obese infants more commonly become obese adolescents and adults than do nonobese infants [7,14]. From a slightly different perspective, once an infant or child has become significantly overweight, he or she is very likely to stay overweight into adulthood.

The differential diagnosis of simple obesity in infancy and childhood includes a variety of endocrine, genetic, and congenital disorders: hypothyroidism, Cushing's syndrome, Frolich's syndrome, Prader-Willi syndrome, disorders of glycogenosis, Klinefelter's syndrome, Laurence-Moon-Bidel syndrome, pseudohypothyroidism, Alstrom's syndrome, multiple X chromosome, Mauriac syndrome, CNS disease, or brain damage secondary to tumor, trauma, or infection [15]. One common clinical finding that characterizes obese children for hormonal or genetic reasons is that they are short (usually less than the fifth percentile in height) and have delayed bone age, whereas children with simple obesity are almost always at the 50th percentile or greater and have a normal or advanced bone age [1].

Although there are numerous and well-defined etiologies for obesity secondary to other disorders, little research has been devoted to outlining specific subtypes of simple obesity. Such features as age of onset (eg, neonatal, infantile, toddler, preschool, school age), acuteness of onset, family history, presence of psychopathology in parents and/or child, and cultural practices or nutritional attitudes may all be quite important in clinically relevant categorization.

Forbes [16] is one of the few workers who has reported data that suggest that there are two groups of obese children. The first group is characterized by a definite increase in lean body mass as well as fat. In the second group, the excess body weight is due exclusively to the accumulation of fat. Subjects in the first group tend to be tall, to have advanced bone age, and to be obese since infancy. Subjects in the second group are, on the average, of normal height, are less apt to have accelerated bone age, and are more apt to have become obese during the mid- or late childhood years. Excess weight in this group is due entirely to fat accumulation. Forbes argued that these data suggest that "developmental" and "reactive" forms of obesity differ.

Several authors [17-19] have commented on the influence of dietary misinformation or changing cultural practices that have contributed to the development of infantile obesity. These authors reported that in England major changes have taken place in the feeding patterns of young infants, especially in the past 20 years. The incidence of breast feeding has declined, solid food is introduced at an increasingly early age, and artificial formulas are used in an excessively concentrated form. These practices have resulted in an increased incidence of obesity as a direct result of the excessive caloric intake of the infants.

An even more comprehensive diagnostic system for simple obesity would divide it into three categories: familial obesity, psychogenic obesity, and simple excessive caloric intake. In familial obesity, the onset of obesity starts in the neonatal or infantile period, and is gradual and progressive; there is a strong family history of obesity. Although there are no major areas of nutritional misinformation, there may be important cultural practices that favor a high-caloric diet. There is no major psychopathology in the family, parents, or child.

In psychogenic obesity, the onset may be either acute or more gradual and be at any age in childhood. There is no family history for obesity and no history of cultural or nutritional attitudes that support excessive caloric in-

take. The origin of the disorder is a significant disruption of the parent-infant relationship so that food is used to assuage psychological distress. There may or may not be evidence of formal psychopathology in the infant or parents.

Simple, excessive caloric intake obesity has an early age of onset and has no evidence of psychopathology in the parents, infant, or parent-infant relationship. The cause of the excessive caloric intake is secondary to relative well-defined and specific cultural practice or nutritional misinformation. There is a negative family history for obesity unless the same nutritional practices have been operating in previous generations.

Although this diagnostic classification system is supported by research and clinical findings, it can be viewed as helpful only if it is heuristically important. This system would lead researchers to readdress the basic assumption that there is a unity cause for a unitary syndrome of simple obesity. For example, we would expect metabolic and cellular contributions to familial-type obesity to be much more prominent than in psychogenic obesity or simple caloric intake. In addition, response to treatment would be predicted to be rapid in simple excessive caloric intake, variable in psychogenic obesity, and quite poor in familial obesity.

DISORDERS OF INSUFFICIENT GROWTH AND WEIGHT GAIN

Psychosocial Dwarfism

Psychosocial dwarfism (PSD), also called deprivational dwarfism, is a syndrome of deceleration of linear growth combined with characteristic behavior disturbances (sleep disorder and bizarre eating habits), both of which are reversible by a change in the psychosocial environment [20-23]. The most characteristic abnormal behaviors include polyphagia, gorging and vomiting, stealing and hoarding food, and eating from garbage pails and animals' dishes. Polydipsia, including drinking stagnant water, toilet bowl water, and dishwater was seen. Some have insomnia and night wandering [20]. PSD has its onset between 18 and 48 months of life. The long-term prognosis and prevalence rate of PSD are unknown.

The differential diagnosis for the organic causes of growth delay or failure include hypopituitarism from a variety of organic etiologies, primodial dwarfism, intrauterine growth failure with persistent small size, congenital hypopituitarism, constitutional delayed growth, Turner's syndrome (XO chromosome pattern), osteochondrodystophies, various chronic diseases, including congenital heart disease, chronic renal failure, chronic gastrointestinal disease, and chronic pulmonary disease [15].

Research in PSD has been hampered by the persistent confusion in the literature of PSD with nonorganic failure to thrive (NFTT). Although these two syndromes have quite distinct ages of onset, psychosocial characteristics and neuroendocrine correlates [24,25], the fact that they both involve psychosocial disruption and growth delay has caused considerable confusion. An example of the persistent nature of this diagnostic confusion between NFTT and PSD is presented in the most recent and comprehensive review article about PSD [20]. Although the authors concisely defined PSD as not including malnutrition as directly contributory to the disorder, and although they quoted Blizzard's clear distinction between PSD and NFTT, they used the term "failure to thrive" as a synonym for PSD in several places of their literature review, discussion of etiology, and summary.

Much of the research on PSD has focused on neuroendocrine abnormalities found in PSD in an attempt to unravel the relationship between growth rate and neuroendocrine changes. Unfortunately, these investigations have led to the discovery of no pathognomonic or consistently abnormal findings, with the possible exception of depressed somatomedian levels [20]. The abnormalities in hormonal levels or responses to provocation that have been found tend to normalize partially or completely following the subject's removal from the inimical environment. This normalization occurs from as quickly as a few days to as long as two years, depending on the specific endocrine disturbance, and with no specific medical, hormonal, or psychiatric treatment [20]. It also should be emphasized that none of the abnormal hormonal findings correlate on a one-to-one basis with growth failure. Growth failure has occurred with normal endocrine values, and catch-up growth has occurred with subnormal values [20]. The mechanisms causing the growth failure in PSD are thus, as yet, unknown.

Rumination

Rumination or merycism is a syndrome in which previously ingested food is voluntarily regurgitated, rechewed, and partially reswallowed [26]. In the process of this regurgitation, the infant loses a considerable amount of food and so becomes malnourished [26]. According to Cameron [27], "the infant appears withdrawn and apathetic except when in the act of ruminating. At these times, the infant lies with an expression of supreme satisfaction upon its face, sensing the regurgitated milk and subjecting it to innumerable sucking and chewing movements...It is very evident that achievement of his/her purpose produces a sense of beatitude while failure results in nervous unrest and imitation." In

a review article, Flanagan [28] reported that predisposing factors, familial pattern, and prevalence are all unknown. The age of onset is usually between 3 weeks and 12 months of age in normal children, while in retarded children it may not occur until they are several years old [29]. The clinical course can range from a relatively benign, time-limited episode to a severe and chronic condition that causes severe malnutrition, growth failure, and even death [26,29].

Like many poorly understood syndromes, the spectrum of etiological explanations for rumination ranges from purely physiological [30] to purely psychological [26,27,29,30]. The literature on the psychological understanding of rumination has been sharply divided between psychodynamic and behavioral approaches, even though these two ways of viewing rumination are not necessarily mutually exclusive [29]. The psychodynamic explanation focus on an unsatisfactory mother-infant relationship [26,31-33], which causes the infant to seek an internal source of gratification. This turning inward by the infant has been proposed to occur either because the environment is more stimulating than the infant can tolerate [28], because the environment is not gratifying enough [26,31], or because the environment is too stimulating with negative effects [32,33].

Learning theory explanations focus on the reinforcing feedback responses that the rumination elicits. These proposed feedback mechanisms include positive reinforcement (a desired "event" such as pleasure or attention follows the rumination) and negative reinforcement (an undesired "event" such as anxiety is reduced or removed). A more sophisticated theory involves combining the concept of positive and negative reinforcement by proposing a change in the valence of the behavioral consequnces. Consequences that are normally negative, behavior suppressing, may acquire positive, behavior-reinforcing characteristics if other, usually more positive, consequences are lacking [29].

From a very different perspective, some authors have argued that rumination is totally the result of physical disorders, including hiatal hernia and other esophageal abnormalities [30,34,35]. The relationship among the syndromes of rumination, gastroesophageal reflux (GER), psychophysiological vomiting [32] and NFTT is unknown. GER or chalasia is the syndrome of regurgitation of the stomach contents into the mouth and esophagus. New studies [36] indicate that this disorder may be caused by hypomobility of the gastric fundus and associated delayed gastric emptying rather than a weakened esophageal sphincter. Severe GER is associated with failure to thrive as well as specific behavioral abnormalities such as abnormal postures and torsion spasm of the neck [30,37]. A possible but untested hypothesis is that GER is the physiological substrate upon which various psychosocial disruptions act so that rumination syndrome develops.

The vociferousness with which each group of proponents argues in favor of their theory as the sole explanation for rumination is a testament to how much needs to be learned about this syndrome. A more realistic etiological approach would attempt to synthesize the various psychological and physical theories into a more coherent whole that recognizes that rumination probably has many "causes," some of which will be more prominent in different individuals. Three factors seem to be required for the development of rumination syndrome: an impaired ability by the infant to regulate his/her internal state of satisfaction, a physical propensity to regurgitate food, and a learned association that regurgitation helps relieve the internal state of dissatisfaction. Both research and clinical practice need to evaluate the relative importance of each one of these three factors in each patient if a better diagnostic understanding and treatment is to occur.

Nonorganic Failure to Thrive Syndrome (NFTT)

NFTT is a disorder of the first 24 months of life characterized by marked deceleration of weight gain and slowing of acquisition of developmental milestones. A deceleration of linear and head circumference growth is an associated but not a primary phenomenon. Failure to thrive is a common disorder, occurring at a rate of 1% to 5% of pediatric hospital admissions [38,39]. From 15% to 58% of these admissions have no demonstrable organic cause for their poor growth and are called NFTT [39,40].

The relative importance of hyponutrition versus psychosocial deprivation in the development of NFTT is a controversial issue. The DSM-III apparently has decided that the psychosocial deprivation is primary, since it calls the syndrome "Reactive Attachment Disorder of Infancy" rather than failure to thrive [2]. However, Whitten et al [41] have shown that even grossly understimulated infants with NFTT will gain weight rapidly if given enough food. Conceivably, both sides of the controversy may be correct. Psychosocial deprivation can cause infantile depression and developmental delay without deceleration of weight gain [42], and lack of nutrition may cause weight gain deceleration without developmental delay. Obviously these two separate processes are intimately interrelated, since starvation produces characteristic behavioral and developmental changes [43,44] and since behavioral and affective disturbances caused by psychosocial deprivation create problems for adequate feeding. In addition, the same type of psychosocial factors that lead to insufficient parental psychosocial stimulation of the infant may also contribute to parental inability to provide sufficient

feeding of the infants [1].

A major weakness in research in NFTT has been the failure to investigate or even classify the degree of malnutrition that the infant is suffering [1]. Merely describing the infant as having fallen below a certain weight gain percentile curve does not describe the infant's nutritional status [45]. Partly because of the confusion with psychosocial dwarfism, numerous authors [46-48] have argued that the lack of weight gain in NFTT is not related to caloric intake but rather occurs as a result of metabolic/hormonal changes secondary to the psychosocial stress factors. Newer studies [1,41,49,50] strongly support the alternative hypothesis that weight gain recovery in NFTT is directly proportional to caloric intake. However, Bell and Woolston[49] described a group of infants with NFTT whose percentage of ideal body weight for length, age, and sex (% ideal body weight) was 81% or greater. In this group of infants there was no such linear correlation between caloric intake and weight gain, apparently because other factors such as activity level and metabolic state become more heterogeneous as an infant approaches the ideal body weight.

Since there are specific behavioral and hormonal changes that occur with certain levels of hyponutrition, the degree of protein-calorie malnutrition must be established if meaningful research is to be done. Such areas as special developmental characteristics of the infants and levels of psychosocial distress of the parents may be dependent variables to malnutrition.

One of the major issues that faces clinicians who treat infants with failure to thrive is how to differentiate organic failure to thrive from NFTT [1]. Although the literature on the special characteristics of infants with NFTT and their caretakers is voluminous (eg, review articles Cupoli et al [51] and Goldbloom [45]), it is not particularly helpful at providing nonexclusionary criteria to separate NFTT from other forms of FTT. Most of the articles are flawed by methodological weaknesses, and ways to easily differentiate NFTT from organic FTT are not yet available.

Infants with NFTT frequently have been found to display a variety of unusual behaviors such as "radar gaze" [52], unusual watchfulness [53] or unusual postures [54]. However, since only one study [55] that found special behavioral characteristics for NFTT used a control group of infants with organic FTT, it is unclear whether these special behaviors are really characteristic of NFTT in particular or rather are associated with all types of FTT. In this study of infants between 6 and 16 months of age, Rosenn et al [55] controlled for both the effects of weight loss and hospitalization. They used both hospitalized children with normal weight and children with organic FTT as controls. They found that they could reliably distinguish among these three groups by observing their response to a structured social interaction. Infants with NFTT predictably preferred distant social encounters and inanimate objects, while organic FTT infants and medically ill control patients consistently responded most positively to close personal interactions such as touching and holding.

Mother-infant interaction in NFTT has not been well studied [1]. In one of the few reports that examined mother-infant behavior, Vietze et al [56] used a prospective, multiple-parameter behavior frequency design. Their study was controlled for race, marital status, maternal age, level of education, parity, and gravidity, but not for infantile poor growth. They found no dyadic or infant behavior that differentiated infants who later became NFTT. The only difference between the two groups was that mothers of infants who were later diagnosed as NFTT spent less time in visually attending to their infants. The paucity of behavioral differences reported in this study is presumably due to studying newborns who were not yet failing to thrive.

The caretakers and families of NFTT infants have been found to have a variety of psychiatric, psychological, and psychosocial problems [1]. Many authors have found the mothers to be either acutely or chronically depressed and overwhelmed [53,57-59]. None of the studies was controlled for socioeconomic status, impact of hospitalization, rater bias, or nutritional status of the infant. Fishcoff et al [58] found mothers of children with NFTT to have histories of early childhood disturbance; poor performance in current day-to-day activities; desire for an anaclitic relationship; the use of denial, isolation, and projection as major mechanisms of defense; and a predisposition toward action as opposed to thought. However, they also found that 2 of the 16 mothers they studied had none of these characteristics.

Several studies have indicated that families of NFTT children are marked by many stigmata of psychosocial disruption [39,52,53,57]. However, Glaser et al [60] reported that most of the children in their study were members of intact, relatively stable families with steady incomes. Kotelchuck and Newberger [61] reported that three family/ecological factors differentiate NFTT families from families of control infants matched for race, age, and similar socioeconomic status. The parents perceived their child as sickly and themselves as more isolated from their neighborhood and had a larger discrepancy in their education. Demographic data, pregnancy, and contemporaneous stress factors were not significant. The authors used these findings to question whether previous reports of parental psychopathology and family dysfunction might be the effect of having an infant with NFTT, in addition to the cause of it.

Even the concept of the homogeneity of the syndrome of NFTT has been questioned [1]. Egan et al [62] have proposed that there is a second syndrome of weight gain failure of infancy that is quite distinct from "typical" NFTT. They suggested that some infants between 6 and 18 months of age develop such a struggle for autonomy with their caretakers that a vicious cycle is initiated that results in weight gain failure. They described this syndrome as characterized by overly intense mother-infant interaction and active food refusal by the infant.

Woolston [1] has proposed that various syndromes of NFTT should be defined by three axes: psychosocial characteristics of the mothers, behavioral/developmental disturbances of the infants, and specific abnormalities in the mother-infant interaction. Using this schema, NFTT might be divided into three subgroups types (table 1).

In Type I the mother both undernourishes and understimulates her infant. One would expect the mother to be emotionally unavailable, the infant to show developmental delays and an abnormal response to proximal interactions with others, and the mother-infant interactions to be characterized by a paucity of warmth and nurturance.

In Type II, the mother provides adequate stimulation for her infant but, as a result of misinformation or lack of resources, does not provide adequate nutrition. One would expect the mothers to appear within normal limits on psychological testing, the babies to show no abnormalities except in growth, and the mother-infant interaction to be within normal limits.

In Type III, the infant is struggling to create autonomy from the mother. One would expect the mothers to be angry or depressed, the babies to show specific behavioral disturbances focused on food refusal but without developmental abnormalities, and the mother-infant interaction to be characterized by negative and angry interchanges.

Although this subgrouping schema of the NFTT syndrome is compatible with the current available data, it must be regarded as tentative [1]. Empirical studies using controlled and blind methodologies will confirm, modify, or disprove the utility and accuracy of this schema.

The ultimate aim of refining our diagnostic work is to allow reliable classification of NFTT into homogeneous subgroups [1]. Once this subgrouping has been achieved, a variety of research questions about etiology, prognosis, and treatment can be vigorously pursued. The linkage between NFTT and major depressive disorder could be investigated on genetic, epidemiologic, endocrine, and neuroendocrine parameters. The relationship between protein-calorie malnutrition and be-

Table 25.1 Three Subgroups of NFTT

NFTT Type 1 (Reactive attachment disorder of infancy)
Infants
1. Significant developmental delays in motor, language, and adaptive areas
2. Lack of developmentally appropriate signs of social responsivity as defined by DSM-III in reactive attachment disorder of infancy
3. Onset of failure to thrive (FTT) before 8 months of age
Mothers
1. Perceive their infants as sick
2. Psychopathology characterized by depression and social isolation
Mother-Infant Interaction
1. Few interactions indicative of pleasure and mutual social responsivity
2. Infants prefer distal to proximal interaction
3. Infants show apathy and/or active withdrawal in proximal and feeding interactions
NFTT Type II (Simple calorie-protein malnutrition)
Infants
1. No or minimal developmental delays
2. Developmentally appropriate signs of social responsivity
3. Onset of FTT before 12 months
Mothers
1. Perceive their infants as sick
2. No characteristic psychopathology or psychosocial disruptions
Mother-Infant Interaction
1. Frequent interactions indicative of pleasure and mutual social responsivity
2. Infants prefer proximal to distal interactions
3. Infants cooperative and vigorous in proximal and feeding interactions
NFTT Type III (Pathological food refusal)
Infants
1. No or minimal developmental delays
2. Developmentally appropriate signs of social responsivity
3. Onset of FTT between 6 months and 16 months
Mothers
1. Do not perceive their infants as sick
2. Psychopathology characterized by depression and hostility
Mother-Infant Interaction
1. Few interactions indicative of mutual social responsivity and pleasure
2. Infants prefer distal to proximal interactions
3. Infants show angry withdrawal and active avoidance in proximal and feeding interactions

havioral/developmental and mother-infant interactional abnormalities could be explored. The effects of specific treatment interventions could be rigorously and scientifically evaluated.

As exemplified by NFTT syndrome, eating disorders of infancy and early childhood are complex and poorly understood phenomena, which are only just beginning to be described in detail. Research in this area is frought with many obstacles that continue to impede its progress. However, the application of more sophisticated research techniques should help to resolve some of the current confusion.

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