The Eating Disorders
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Chapter 35

Atypical Eating Disorders

Stanley L. Goodman, Barton J. Blinder, Barry F. Chaitin, and Jennifer Hagman

ATYPICAL EATING DISORDERS

Atypical eating disorders are classified in DSM III-R as disorders of eating that do not meet the criteria for a specific eating disorder due to absence of particular clinical criteria (frequency of symptom, clinical sign, and in special instances gender or age).

This chapter discusses the presence of anorexia nervosa and bulimia nervosa in children, males, and females beyond age 25 and in the elderly are also presented. In addition, cultural presentations of anorexia nervosa in Blacks and Hispanics, anorexia nervosa in neuropsychiatric conditions, and eating disorders associated with medical conditions, hyperphagic conditions, and eating disturbance in various endocrine disorders are also reviewed. Core psychological conflicts, etiologic considerations, precipitating events, prognosis, and treatment specific to these unusual and atypical presentations are also discussed.

ANOREXIA NERVOSA IN CHILDREN

Anorexia nervosa has been reported beginning at age four [1]. Childhood anorexia should fulfill criteria for adolescent- or adult-onset anorexia nervosa, except that in children, due to a diminished amount of body fat, a 25 percent weight loss is not necessary. In female childhood cases, primary amenorrhea occurs.

The incidence of prepubertal anorexia nervosa is three percent in a series of 600 consecutive patients of all ages evaluated for anorexia nervosa at the Mayo Clinic [2]. Females comprise 73 percent of all reported children with anorexia nervosa [1]. However, in one subgroup of anorectics (see below), 50 percent were males [3].

Developmental antecedents of childhood anorexia have not been systematically research [4]. Delaney and Silber [5] evaluated approximately 30 cases and noted lack of stage-specific negativism at age two, anxious clinging behavior upon commencement of school, and difficulty maintaining peer relations, leading to a degree of social isolation.

In infants ages nine to 26 months, Chatoor and Egan [6] described a developmental disturbance which they consider to be both a separation disorder and a form of infantile anorexia. These infants were noted to have a diminished growth rate and food refusal. Feeding became a battleground for maternal-infant autonomy struggles. The infants resisted feeding as a manifestation of their independence from an overwhelmingly strong maternal figure. The child's fight for independence through anorexic behavior is sharply contrated to the listless marasmic pattern that characterizes the withdrawn underfeeding in anaclitic depression [7].

Latency-age children, at the Piagetian stage of concrete thinking, conceptualize food and water together as one entity, resulting in global ingestive restriction. This may lead to rapid weight loss and serious dehydration. In addition, prepubertal children, especially girls, have less body fat than their adolescent counterparts and become more quickly emaciated [7]. In Irwin's series, over two-thirds of the children with anorexia were hospitalized within six months of the onset of the anorexia. Gislason [1] noted one death in 33 children with prepubertal anorexia nervosa.

Sargent [3] described three subgroups of prepubertal anorectics. The first group, similar to one described by Pugliese et al. [8] severely restricted their food intake, resulting in short stature. They had fears of becoming obese, and by their deficient weight gain they maintained both a physical and psychological immaturity. The second group consisted primarily of prepubertal females, ages ten to twelve, who were psychologically pseudo-precocious, engaging in overt behavior more characteristic of that of a pubescent 14-year-old. However, they were described as being "lost little girls" underneath this facade. Their parents discouraged age-appropriate behavior and strongly encouraged their pseudo-adolescent behavior. This

female subgroup is closet to the pubertal-onset anorexia nervosa. The third subgroup consisted of an equal number of male and female anorectics who were more psychologically impaired, having major ego deficits with the occasional prescence of psychotic episodes [9].

Gislason [1] noted premorbid personality characteristics of dependency, timidity, and schizoid traits, with features of depression. Significant disturbances of ego development, prepsychotic personality traits, and psychotic episodes have been reported [1]. However, these studies were completed prior to the more precise DSM III diagnostic criteria. Moreover, premorbid eating disturbances, including a history of being a finicky eater, have been noted [7].

Family patterns in childhood anorexia nervosa have not been systematically studied. Family structural characteristics found in adolescent anorexia nervosa, consisting of *rigidity, lack of conflict resolution, and triangulation*, appear to be present in the families of children with the disorder [7]. In childhood anorexia, Sargent [3] noted increased divorce among families, as contrasted to adolescent anorexia where the family divorce rate is approximately equal to the general population.

The clinical manifestation of childhood anorexia nervosa should fulfill most DSM III-R criteria. However, since prepubertal children, especially girls, have less body fat than their adolescent counterparts, a 15 percent reduction in body weight should be sufficient for diagnosis [7, 10]. These children, while recognizing that they are thin, deny feeling fat. It is unclear if there is a body image distortion equivalent to that of older anorectics. Furthermore, no systematic comparative studies in children have been undertaken. The child may be more concerned with separation-individuation issues than fears of sexuality [7, 11]. They frequently demonstrate alexithymia, the inability to translate one's feelings into words [12].

Overt or covert seduction or sexual abuse may be found in the histories of some patients, although Bruch [13], Blinder [14], and Piazza [15] question this hypothesis. Sloan and Leichner [16] reported childhood sexual abuse in a number of adult patients with bulimia nervosa. Oppenheimer et al. [17] reported that two-thirds of 78 eating disorder patients spoke of distressing sexual experiences as a child, including sexual abuse. However, the significance of a history of sexual abuse for the occurrence of anorexia nervosa remains unclear.

Many children with anorexia nervosa manifested signs of depression. These feelings may be the result of helplessness and ineffectiveness internally perceived and mirrored from family attitudinal reactions. Studies reported prior to DSM III did not utilize structured interviews such as Kiddie SADS. In child anorectics, no formalized studies specifying biochemical, diagnostic, or family criteria for major depressive disorders have been reported. While anorexia nervosa has been considered a variant of affective disorder [18,19], the relationship between childhood anorexia nervosa and affective disorder must still be clarified.

Anorexia nervosa has been associated with Tourette's Syndrome (stereotyped movement disorder) [20,21,22] with Turner's Syndrome, a chromosome disorder with XO genotype and gonadal dysgenesis [23], and with mental retardation [24].

Piazza [15] reported anorexia nervosa in association with childhood ulcerative colitis and ileitis. The patient manifested typical bowel symptoms with diminished appetite, and only upon careful questioning was the diagnosis of anorexia nervosa determined. One child had anorexia nervosa and abdominal complaints which initially masked colitis.

The etiology of childhood anorexia is uncertain. Irwin [7] feels psychodynamics in childhood anorexia nervosa are similar to the dynamics of adolescent onset and include identity disturbance, failure of separation/individuation with fears of growing up, maladaptive attempts to be in control, and failure of parents to resolve marital or family conflicts. An alternative pattern may include a child who is sensitive to family food preoccupation and identifies with a family member who has an eating disturbance.

Precipitating events associated with the onset of childhood anorexia nervosa include: 1) the birth of a sibling; 2) bereavement over the death of a parent or relative; 3) a disappointment in object relations; 4) family discord; 5) viral illness; 6) peer criticism about being fat; 7) the fear of becoming obese; 8) the onset of breast development; 9) sexual abuse; 10) sustained fear of choking while eating; 11) anticipated fear of parental loss related to an ill or depressed parent; and 12) the onset of a psychophysiologic disorder such as colitis [1] or ileitis [13].

In the treatment of the childhood anorectic, the therapist should work closely with a pediatrician to rule out medical and psychological conditions producing anorexia. A physical examination and laboratory studies are mandatory to monitor the child’s physical condition. Starvation effects include: 1) hypotension; 2) syncope; 3) bradycardia; 4) hypothermia; 5) dry skin; 6) languor hair; 7) diminished triceps skin-fold thickness; 8) hypoglycemia; 9) hair loss; 10) sensitivity to noise; 11) leukopenia; 12) fatigue; 13) cardiac arrhythmia; 14) electrolyte disturbance; 15) hypothalamic dysfunction; 16) diminished thermoregulation; 17) hypercortisolism; 18) vasopressor regulation; 19) gastric ulcer; and 20) initial motoric hyperactivity. Starvation can cause psychological and cognitive disturbances including food preoccupation, poor

concentration, social isolation, depression, and labile moods [25].

Children, especially those restricting both fluids and food, may need immediate hospitalization. A childrens inpatient psychiatric unit can be effective in resolving both physiologic and psychological problems [11,26]. The dietician must determine the appropriate caloric intake for the child. Calories and nutrients not ingested due to food refusal must be supplemented by liquid intensive formula per mouth, or by tube feeding if necessary. Parenteral hyperalimentation is rarely necessary for childhood anorexia.

Most children's psychiatric inpatient unit use flexible behavioral modification approaches to weight gain. Unlike adolescent or adult behavioral programs, children should be allowed to attend all ward activities regardless of specific weight gain [11]. Overactivity on the ward must be monitored and controlled to avoid weight loss. Panikar [27] used a wheelchair to restrain a severely overactive child. The schizoid, withdrawn child can be rewarded with points, tokens, etc., for engaging in verbally expressive social interaction. More intensive involvement of the family in the therapeutic pain may have to await medical stabilization and emerging data concerning developmental history, effects of separation (positive and negative), and family strengths and liabilities.

Play therapy using projective techniques can be useful. While children ages ten through twelve may feel too old for play therapy, they may not be cognitively prepared for verbal therapy. They may also be resistant in therapy; therefore, therapeutic creativity is needed. Panikar [12], noting alexithymia, draws smiling faces and asks the childre to identify various moods. Affective expression can be encouraged, such as in Gardner's "Thinking, Talking, and Feeling Game" [28]. Lucas [29] finds art therapy to be particularly effective when compared to play therapy which the anorectic child often resists and obscures.

Feinstein [30] alternating both male and female therapists, recognizes that the child can work through separate conflicts with different therapists.

Sargent [2] modifies treatment according to the three anorexic subgroups described above. In the first group of anorectics, with growth retardation described by Pugliese et al. [8] the social worker and nutritionist meet on a regular basis to improve caloric intake. Pugliese uses a cognitive approach, informing the children that they need to eat to be strong and grow.

In Sargent's [2] second group of precocious females, individual psychotherapy explores the child's underlying separation, identity and sexual conflicts and her need to recognize feelings of ineffectiveness and confusion. The parents must recognize their need to push their daughter into premature adolescence and must work with a dietician to promote normal caloric intake.

In the third group, the therapist must assist the child in recognizing his own sense of worth and must establish a strong therapeutic alliance encouraging normal ego development, interpreting, countering, and ameliorating what may be a chaotic enviornment in the family. The parents, often having severe psychopathology, need to resolve their own conflicts to stabilize the family. With such a family setting, a period of hospital separation may be a beneficial opportunity for clarification, intensive therapeutic focus on the child, and emergency of diagnostically significant elements of family psychopathology masked by the dramatic nature of the child's anorexia.

Chatoor [31], in her treatment of infantile anorexia, uses ten food rules, including restricting a specific mealtime to thirty minutes and refraining from emotionally laden comments. The goal is to allow the infant to reexperience hunger in a neutral, time-limited setting. In some cases, the mother may need to work through her own internal conflicts [5].

The prognosis in childhood anorexia is unclear. Sargent feels that the Group II females have less individual and family psychopathology, and have the most favorable outcome as contrasted to Group III, since both individual and parental psychopathology are severe. The Group I prognosis is intermediate between Groups II and III. Delaney and Silber [4] noted male anorectics with a poor prognosis and reported that many families have been resistant to follow-up. Gislason [1] summarized and reported cases of prepubertal anorexia and noted that 63 percent improved, 21 percent did not improve, and 3 percent died.

Russell [14] found prolonged delay of puberty (a late menarche) and possible permanent interference with growth in stature and breast development in children with prepubertal anorexia nervosa. In contrast, Pfeiffer, et al [15], noted relatively minimal growth retardation on a several-year follow-up of treatment. He stresses the importance of identifying childhood anorexia nervosa and returning the children to an optimum weight to safeguard their puberty. A long-term follow-up is necessary to accurately determine prognosis.

Anorexia nervosa has been reported in prepubertal children, many of whom appear to be more disturbed than adolescents with the same disorder. The precise etiology is uncertain, but life events centering on losses and separation may be onset factors. Due to less body fat and ingestive restriction of both food and water, this disorder may be more ominous in children, necessitating a rapid and vigorous therapy and frequently requiring inpatient treatment. Prognosis is guarded and uncertain. Furthermore, coexistent medical illnesses, colitis, or ileitis require careful physical examination

and diagnostic procedures and comprehensive integrated treatment and management.

ATYPICAL BULIMIA NERVOSA DISORDERS IN CHILDREN

There have been no extensive published reports of childhood bulimia in association with purging. The appearance of bulimia nervosa in latency may be rare. Prepubertal children were thought to be insufficiently sophisticated to purge; and latency-age girls were thought to be less vulnerable to the social pressure to be thin. However, Herzog, et al. [16] noted a ten-year old male, and Goodman [17] noted a fourteen-year old female, each hospitalized with an affective disturbance with both binging and purging initiated in latency. Moreover, Walsh [18] has identified adolescent female bulimics who admitted to purging while in latency.

Two cases of bulimia and/or purging (vomiting) beginning in latency are reported. One patient had a severe affective disorder with mood lability and frequent wrist cutting when seen as an adolescent. Two case reports of male bulimia occurred in association with object loss featuring global impulsiveness, learning disability, and in one case, possible Attention Deficit Disorder (ADD) [19].

There may be a subgroup of mildly obese, impulsive children (especially males) who have a variant of bulimia. A complete evaluation, including the way stressful events modify eating, the presence and frequency of binging, fluctuations in weight, depression, and delineation of individual and family psychodynamics, is warranted in obese children. Bulimia in latency, while probably rare, does occur in atypical forms. Primary physicians and school nurses need to recognize these symptoms and carefully evaluate eating patterns of latency-age obese and impulsive children.

ANOREXIA NERVOSA IN MALES

Anorexia was first described in males by both Morton [20] and Gull [21]. Anorexia in males accounted for approximately six percent of cases seen in an eating disorder clinic [16,22]. Anorexia may be underdiagnosed because many physicians, as well as the anorectics themselves, are unaware that this condition occurs in both sexes.

The mean age of onset of male anorexia has been reported ranging from as young as 17 years in a British series by Crisp and Burns [23] to 24 years [16,24]. Crisp found that the illness was present an average of three and one-half years and that most patients were mildly obese (127.3 percent of ideal body weight, IBW) prior to the onset of illness. Minimal weight dropped to 67.3 percent of IBW [24] during the acute phase of illness.

Apparently contrasting socioeconomic groups of origin for male anorectics may represent specific populations, seen in various programs. Andersen and Mickalide [25] found a high socioeconomic group at Johns Hopkins, while Herzog [16] in Boston and Verdereycken and Van den Broucke [22] in Belgium found an even socioeconomic distribution.

Clinical manifestations of male anorexia were reported in several series to be similar to female anorexia [22,23,26,27,28,29,30]. However, in a minority of reports [31,32] differences were noted; patients were from lower socioeconomic groups, feared competition, and were not successful either academically or in their vocation.

Clinical manifestations of two male anorectics reveal differences in presentation from female counterparts. Male patients are usually more active, have more sexual anxiety, have fewer bulimic episodes, with less vomiting or laxative abuse, and, on a personality inventory, indicated more preoccupation with food and weight, more achievement orientation, and more physical complaints [33].

Endocrine changes (diminished testosterone level) in male anorectics may have a role in diminishing conscious sexual concern or conflict, which may be congruent with inhibitory physiological defenses.

Yates et al. [33] compared male marathon runners to anorectics and found many similar sociocultural and personality characteristics. Runners were found to have a bizzare preoccupation with food, and even when they would achieve a lean body mass of 95 percent with only 5 percent body fat, they would aim for four percent body fat. Many have lost greater than 25 percent of their original weight and show a relentless pursuit of thinness or a disturbance of body image. Yates suggests male runners are searching for identity. She raises the question, "Do anorexia nervosa in females and obligatory running in males share a common psychopathology?"

Male anorectic characteristics include perfectionism and obsession [25]. Vandereycken and Van den Broucke [22] noted a high incidence of schizoid/introversion features (30 percent) as well as obsessional (29 percent), passive/dependent (15 percent) and anti-social features (18 percent). A comparison to female anorectics showed a higher percentage of undifferentiated-immature psychological structure (30 percent versus four percent), hysterical/histrionic features (25 percent versus four percent), and anti-social features (18 percent versus one percent), but an equal number of schizoid/introversion traits (28 percent) [22].

The etiology of male anorexia is unclear, but Crisp and Burns [23] hypothesize that it is related to

major gender identity problems in the premorbid personality, since the male desire is to be bigger and stronger as compared to the female preference for slimness. Herzog [16] found male anorexic patients experiencing sexual isolation, sexual inactivity, and conflicted homosexuality. He posited that the cultural pressure on the homosexual male to be thin and attractive places him at a greater risk for eating disorders. Undefined intrapsychic factors may promote an association between conflicted homosexuality and eating disorders.

Hall [26] in a series of nine male patients whose personal family history was reviewed, noted attention directed to bodily concerns caused by being overweight (2/9), having close contact with an eating-disordered patient (2/9), attempting to identify with a thin family member (2/9), attempting to treat acne through a stringent diet (2/9), and attempting to deal with the fear of having cancer (1/9).

Precipitating events of male anorexia nervosa include overwhelming stress such as divorce, children leaving for college, increased job responsibility, and a family member who becomes serious ill [25].

Endocrine disturbances present in pre-treatment male anorexia include decreased testosterone and gonadotrophins (luteinizing hormone-LH and follicle stimulating hormone-FSH) in proportion to weight loss. With weight gain, both testosterone and gonadotrophins increase to normal levels [25,34,35]. Andersen and Mickalide [25] noted that two of ten patients were infertile. The creased testosterone may also result in depression of the bone marrow.

Anorexia in males has associated medical disorders such as anemia and abnormal liver function [25]. There are also associated metabolic abnormalities such as self-induced vomiting, resulting in hypokalemic alkalosis [27].

BULIMIA NERVOSA IN MALES

Bulimia has been reported in male patients [16,36,37,38]. Herzog et al. [16] noted an incidence in males of approximately four to five percent of a total population of bulimic patients. Gwirtsman found that ten to 13 percent of male students met DSM-III criteria for bulimia. The mean age of onset ranged fro 21 [16] to 24 [36] years. Duration of illness prior to treatment ranged from six years [36] to 7.4 years [16]. This duration is significantly longer than the 4.2 years' duration of illness prior to treatment for bulimic females [16].

Approximately two-thirds of bulimic males had a history of being overweight as compared to one-third of bulimic females. Socioeconomic classes were equally distributed in one series [16]. Mitchell's [36] study noted that patients were employed, that they were functioning well, and that eleven of twelve were married.

Mitchell and Goff [36] noted that eleven of twelve bulimics were satisfied with their weight which ranged from 81 percent to 100 percent of ideal body weight (IBW). Marked fluctuations in weight (36 percent) were found and ranged from 83 percent to 109 percent of IBW. Twenty-five percent were obese during adolescence [36], in contrast to 64 percent of bulimic males in Herzog's group [16]. Only one patient used laxatives weekly for attempted weight control. None of the twelve patients admitted to the abuse of diuretics, to the use of enemas, or to sham eating. Eight of the twelve patients exercised excessively to control weight, and five of the twelve skipped meals following binges.

The clinical manifestations of male bulimia are comparable to female bulimia. Preoccupation with weight control and associations with the cultural pressures of professional life regarding personal performance (especially in sports, fashion, and music) have been related to the onset of bulimia in some male patients [37].

Psychiatric and drug histories in Mitchell and Goff's [36] series of twelve patients reveal that five patients admitted to alcohol or drug abuse problems in the past and that four had received chemical-dependency treatment. Two of the five developed problems with alcohol prior to the onset of bulimia, and another did so after the onset. One patient reported the simultaneous onset of alcohol abuse and bulimia during a stressful period in his life. Four of these five patients reported a history of chemical abuse problems in at least one first degree relative, and anxiety disorder. That patients periodically substituted alcohol abuse for his bulimic behavior. Gwirtsman and associates [37] noted that two of three patients engaged in drug and alcohol abuse, and that all demonstrated some degree of impulsive antisocial behavior.

Herzog [16] discussed sexual isolation, diminished sexual activity, and conflicted homosexuality in bulimic and anorexic males, but he did not specifically subgroup the sexual difficulties in bulimia. Gwirtsman and associates [37] mentioned anecdotally that bulimia may be more common in the male gay community than among heterosexuals. Furthermore, Mitchell and Goff [36] noted that three out of twelve patients had a history of depression, and that most patients had markedly disrupted social situations and were depressed when first seen, but their mood improved as their bulimia came under control.

Laboratory findings revealed electrolyte abnormalities, 9hypochloremic and hypokalemic alkalosis) resulting from self-induced vomiting and/or laxative abuse [36].

Abnormalities of liver enzymes and amylase were reported by Gwirtsman et al. [37]. Two of three

patients had dexamethasone nonsuppression, and one of three had a blunted TSH response to TRH. These neuroendocrine data are comparable in both male and female bulimic patients, and it is not known if these abnormalities are a consequence of bulimia or have primary neurobiologic significance.

Although Mitchell and Goff [36] noted a significant therapy dropout rate (four of twelve), treatment of eight of the twelve patients was successful in an intensive outpatient group treatment program meeting several times per week. Long-term follow-up data are not yet available.

ATYPICAL EATING DISORDERS IN MALES

Fichter et al. [69] defines atypical anorexia nervosa in males as that occurring in males who fulfill some, but not all, criteria of anorexia nervosa and who manifest atypical symptoms or characteristics.

Of four cases of male anorexia nervosa reported by Fichter et al. [69] two atypical male cases with obsessive-compulsive features were noted. One patient had anorexia, bulimic episodes, and obsessive-compulsive behaviors (hand washing rituals). The onset of illness occurred after a driving accident. The second patient, age 28, developed an obsessive-compulsive neurosis after his father's death, with obsessive thoughts of aggressiveness and with rituals to undo his evil deeds and thoughts. He starved himself and made several suicide attempts. However, body image disturbances and weight phobic symptoms were not present.

Another patient, with full criteria for anorexia nervosa, had a depressive-apathetic syndrome, secondary conflicts with his homosexual partner which produced labile personality changes, and depressive reactions [69].

In addition to weight loss, another patient developed disturbances in swallowing of psychogenic origin and displayed personality features seen in anorexia nervosa (rigidity, social anxiety, achievement orientations) [69].

Andersen and Mickalide [24] noted that 21 percent of male patients who were referred to Johns Hopkins Eating Disorders Clinic had an eating disturbance with weight loss or abnormal eating patterns in the absence of criteria of DSM-III anorexia nervosa.

One group had a swallowing phobia (fear of choking) with significant weight loss, previously misdiagnosed as anorexia nervosa. An earlier choking episode (often vaguely recalled) and a second, more recent choking episode resulted in a sustained fear of choking associated with severe dietary restrictions of solid food. Blinder [39] noted that this syndrome may be a variant of anorexia occurring in a post traumatic context; he found patients who exhibited similar fears after mouth injury or dental surgery. Choking and aspiration, associated with a rare, chronic ruminatory disorder, may also lead to food restriction [40].

Andersen and Mickalide [24] also noted patients who had a classic panic disorder with an associated preoccupation with fears of public vomiting, leading to food restriction and diminished weight. In contrast, a patient with general anxiety had specific overeating episodes unassociated with the fear of obesity [24].

These atypical eating disorders are differentiated from anorexia nervosa, since full DSM-III criteria for anorexia nervosa re not present, and may be defined as a mild form of anorexia nervosa.

ANOREXIA NERVOSA IN FEMALES OVER AGE 25

Anorexia nervosa may occur after age 25 in females [41,42,43]. The oldest reported patient was a 68-year-old woman with no prior history of eating disturbance. While the incidence of anorexia in the general population is 0.37 per 100,000 [44], the incidence of anorexia nervosa in old age is unknown. Less than 100 older patients, both male and female, have been reported in the world's literature [25,41,43,45,46,47]. Adult onset cases usually come from upper middle class families [41].

Anorexia is a symptom seen in other disorders such as severe melancholia [48], schizophrenia [49], and somatization disorder, and in various medical conditions such as carcinoma, orthopedic injuries [50], central nervous system disorders as such Wernicke's encephalopathy [51], and infiltrating hypothalamic tumor [52]. Anorexia (food refusal) not meeting the full DSM-III criteria may occur secondary to purely psychological causes such as hysterical food refusal and food aversion due to somatic pain [43].

Precipitating factors antecedent to the occurrence of anorexia nervosa in susceptible patients include multiple surgical procedures or illnesses [45], stress secondary to childbirth or marriage [46], or death of a spouse [42].

Sexual abuse may be involved in the development of anorexia nervosa in vulnerable women. Sloan and Leichner [48] recently described six anorectic women, first hospitalized as adults, who were sexually abused in childhood or adolescence. In married anorectics whose dependency needs have been shifted to their children, the child's absence resulting from moving or marriage has been associated with an acute onset of anorexia [41]. The adult patients reported feeling ashamed and embarrassed, and they defended the abuse because of

intense super ego pressures, only later, in therapy, revealing the sexual harassment.

Numerous onset patterns have been described. The most common pattern is one in which the patient has a chronic eating disturbance or peculiar eating habits and a stress produces a full-blown clinical expression of anorexia nervosa. In other patients, an anorexic episode may have occurred as an adolescent, followed by a long remission, with stressful events serving to precipitate anorexia at a later time in young adulthood. The most uncommon pattern is an adult patient who develops anorexia *de novo* [43]. The therapist must obtain a very detailed history of the patient's early eating patterns to determine if a prior episode occurred.

Some patients who exhibit pure restrictive anorexia develop bulimia during or after treatment. Failure of symptomatic restraint may first be manifested in bulimic episodes. Vandereycken [43] suggests that some anorectics who fail treatment develop vomiting, purging, or frank bulimia. Kellett and Associates [46] described a 52-year-old woman who purged and vomited in addition to the anorexia.

In a study of 50 married patients, Dally [41] divided anorectics into four groups (see Table 1 [41]). In Group I, onset of anorexia started during the engagement period prior to marriage. In Group II, onset occurred while subjects were married and prior to a pregnancy. Onset in Group III occurred within three years of becoming pregnant. The period after menopause marked the onset of anorexia in Group IV. Dally felt that the anorexia that developed in Groups I and II was a maladaptive solution to an emerging marital crisis.

In Group III, women developed anorexia after childbirth. The preexisting lack of warmth and understanding in their marriages was complicated by a feeling of being trapped by the responsibility of raising children. Unable to express their turbulent emotions, they retreated into anorexia.

In Group IV, women developed anorexia after menopause. These women were withdrawn and depressed, and their anorexia was related to their wish to die, an actual or threatened loss, the death or serious illness of the spouse (36%), or the marriage or dispersal of their children (45%). These women, who had problems with their own separation-individuation and dependency, were overwhelmed by the threatened loss of a spouse or their children, which stirred up their own unmet dependency needs.

Dally [41] reported a varied picture of premorbid problems. In his study, 44 percent of Groups I and II had an earlier episode of anorexia nervosa. Patients who developed anorexia nervosa at or shortly after marriage were more likely to have had a previous anorexic episode. The post-menopausal group had no prior episode but displayed transitory eating disorders with weight loss and food fads in earlier years. Most patients desiring a child hoped for a close mother-child relationship but were fearful of both childbirth and the responsibility of being a good mother. The older group (IV) had more conflicts concerning motherhood, were sick and depressed in pregnancy, and were depressed and anxious during the child's assertive stage. The child's developmental issues may have stirred up their own unresolved conflicts centering on identity and separation-individuation.

Marital conflicts were manifest in all groups, centered around closeness and intimacy. The patients felt themselves to be failures, unlovable, and greedy. Husbands, who were classified in three categories (I-III), were considered immature and over-idealized by their wives. They felt anger at the anorexic behavior and either colluded in the wife's illness, which created a strong dependency bond (50%), or emotionally detached themselves, putting their energy into another relationship.

No consistent family interactional patterns were recognized from Groups I and II. However, patients from Group I through Group III were strongly dependent on their parents, even if they had married, raised children, and lived apart from their family of origin [41].

The course of anorexia nervosa in later life is variable. Crips [49] notes that some chronic anorexic patients who have the illness throughout their reproductive life (puberty to menopause) shed the illness at menopause, while others remain ill, surviving as "isolated, eccentric, and wizened old ladies."

Vandereycken [43] conceptualizes anorexia as an incurable illness in some patients with spontaneously occurring remissions and exacerbations. This chronic course seen in older patients is a form of "process" anorexia nervosa, as differentiated from a more "reactive," self-limited disorder seen in younger, mainly adolescent patients. Furthermore, Vandereycken likens anorexia, with or without bulimia, to an addiction, including the development of malignant autonomy, apparent physical dependency, social descent, and physical deterioration.

Dally [41] notes that Group IV post-menopausal-onset anorectics are markedly depressed and suicidal and may have a more ominous course than their younger counterparts. Treatment of the late-onset patient is complex, and risks such as a psychotic reaction or suicidal depression may occur with refeeding.

Treatment modalities should include nutritional counseling and individual psychotherapy. Family therapy with the spouse and children is indicated to work through family conflicts and disruptions associated with illness. The use of a psychotrpic medication in older anorexic patients to increase weight or mood has not been evaluated. In Dally's Group IV menopausal anorectics with major

depressive disorder, a clinical trial of a tricyclic antidepressant may be useful.

Though some patients with late onset or chronic anorexia nervosa may recover after intensive treatment, patients failing to maintain their weight at four- to eight-year follow-ups may have to inevitably recognize their decision to remain anorectics. In these cases, the goal of treatment is to minimize the physical and emotional handicaps of the disease.

Vandereycken [43] raises ethical questions concerning treatment of chronic anorectics and bulimics. Although the patients may feel life is barren with anorexia, life may become even more barren and painful without it. Furthermore, chronic bulimics can organize their life around the bulimia, with bulimic episodes becoming "institutionalized."

Anorexia nervosa after adolescence may occur in women who have not worked through developmental issues of identity or separation-individuation. They remain dependent on their parents and, to a lesser extent, on their husbands. They feel ineffective, dependent, and conflicted due to their ideal wish to be a "good" mother and wife. Unable to control their lives, they control their bodies, returning to a state of "goodness" by developing anorexia nervosa.

Women beyond age 25 who develop an anorexia syndrome and whose medical examinations fail to demonstrate another etiology should be carefully evaluated for anorexia nervosa as a positive diagnosis.

BULIMIA IN FEMALES BEYOND AGE THIRTY

Bulimia may be underreported in women over age 30. Population surveys have uncovered few older bulimic patients. Jonas [50] reported a 56-year old woman with rapid-cycling bipolar disorder and unexplained vomiting. She had no prior history of an eating disorder, and during hospitalization, the staff discovered surrepitious vomiting. Bulimia disappeared with individual medication trials, first with iipramine, and then phenelzine. Older patients with affective disorder and enexplained vomiting not secondary to psychotropic drug toxicity should be screened for bulimia. Lithium use may be hazardous, therapeutically unpredictable, or lethal in patients with self-induced, surrepitious vomiting, due to electrolyte disturbance. Patients taking lithium for bipolar disorder, emotionally unstable character disorder, or recurrent unipolar depression should be screened for bulimia. Bipolar patients with bulimia may respond to carbamazepine [51] as an alternative to lithium carbonate.

CULTURAL PRESENTATIONS OF ANOREXIA NERVOSA

Anorexia Nervosa in Blacks

Anorexia nervosa has been reported in American Blacks [52, 53, 54, 55] and in Blacks of Afro-Caribbean extraction from the West Indies [56]. The incidence of black anorexic patients in a total population of patients with anorexia nervosa is less than five percent [53]. Case reports from lower socioeconomic groups of West Indian patients living in England [56] and middle-to-upper-class patients [52] are noted.

Anorexia Nervosa in Hispanics

Hispanic females, described by Silber [52], experienced significant disruption moving from South America to Washington, D.C. with resultant object losses of friends and family, including grandparents. The onset of anorexia nervosa in these cases was associated with family disturbance and sexual abuse. Family dynamics in the Hispanic cases included severe disruption, infidelity by the father, alcoholism, depression, suicide attempts, and parental separation, creating a chaotic environment for the patient.

Silber [52] noted that the Hispanic females had high personal ideals. Being raised in a more traditional Latin culture, they may have had difficulty when expected to assimilate into the American culture, where thinness and academic achievement were highly valued. In addition, they had to contend with contrasting sexual attitudes, which may have exacerbated their own conflicts. Development of anorexia nervosa, with its regression to a prepubertal psychological structure, served as a maladaptive attempt to cope with issues of identity and cultural and sexual conflict.

ANOREXIA NERVOSA IN NEUROPSYCHIATRIC DISORDERS

Anorexia Nervosa in Tourette's Syndrome

Anorexia has been seen in association with Tourette's Syndrome (TS) [57, 58, 59]. Blinder et al. [57] described a 14-year-old anorectic with Tourette's Syndrome diagnosed at age nine. The development of anorexia was associated with a family move, a change of schools, and a demanding social environment. The use of haloperidol, with consequent weight gain, may have been an additional provocative factor in initiating a restrictive eating pattern. Larocca [59] reported a 12-year-old male with obsessive-compulsive symptomatology who developed TS near the time of weight gain one year previously. For unexplained reasons, the patient exercised excessively and severely restricted his dietary intake. In Tourette's Syndrome, inadequate

impulse inhibition places an overwhelming stress on the ego which is weakened by this neurophysiologic disorder. In adolescence, these patients may need to cope with both heightened sexual and aggressive conflicts, separation-individuation and identity issues. Anorexia nervosa may be a maladaptive attempt at homeostasis. In the 12-year-old male, and in the case of a 22-year-old female with both Tourette's Syndrome and anorexia nervosa, described by Yayura-Tobias [58] severe depression with overdose or self-mutilation occurred. The coexistence of anorexia nervosa and Tourette's created an overwhelming sense of ineffectiveness resulting in helplessness and depression.

A common central nervous system mechanism may underlie both Tourette's and anorexia nervosa. In addition, Yayura-Tobias [58] hypothesizes that both entities share a common CNS (hypothalmic, caudate) locus, since TS and anorexia nervosa present with a high incidence of associated obsessive-compulsive symptoms. Although neurotransmitter levels have not been studied in patients with both Tourette's Syndrome and anorexia nervosa, Cohen et al. [60] found increased 5-hydroxyindole acetic acid (5-HIAA) in the cerebral spinal fluid of TS patients, suggesting increased serotonin turnover. Serotonin has been implicated in eating inhibition and a shift away from carbohydrate consumption [61].

Neurotransmitter labeled positron emission tomography may be helpful in determining shared neurotransmitter dysfunction, and CNS localization in these coexisting disorders. Further research into common psychodynamic, cognitive, and neurotransmitter determinants, including cerebral mechanisms, are indicated.

Anorexia Nervosa in Schizophrenia

Anorexia nervosa has been reported in patients with schizophrenia [24, 62, 63, 64]. Hsu [62] described six patients who had paranoid delusions and auditory hallucinations in which several heard people stating, "You're so fat and ugly." Prior to the onset of overt psychosis, depressive and suicidal symptoms were present. In addition, major depression but not schizophrenia, was found in the families. Hsu [62] concluded that these patients would be better diagnosed as schizoaffective disorder than schizophrenia. Treatment with phenothiazines was effective in diminishing psychosis, and one patient became psychotic again with refeeding. Another patient with schizoaffective disorder and borderline mental retardation (IQ) was reported [65]. Similar developmental conflicts concerning separation, individuation, autonomy, and control issues may occur in both disorders [66, 44].

Anorexia Nervosa in Post-traumatic Stress Disorder

Anorexia nervosa has been reported in patients with post-traumatic stress disorder. In three patients, an accident caused physical injury, disfigurement, and preoccupation with their bodies. Damlounji and Ferguson [67] posit that physical injury and placement in a stressful hospital environment resulted in body image distortion, which may have been etiologic in the development of anorexia nervosa. Similarly a patient developed anorexia after prolonged use of the Milwaukee Brace [68] which restricted physical activity and may have promoted undesired weight gain.

Anorexia Nervosa in Depression

Fichter et al [69] reported a 15-year-old male presenting with depression, hyperactivity and fasting who lost 35 percent of body weight, but did not have other criteria of anorexia nervosa.

Anorexia Nervosa in Obsessive-Compulsive Disorders

In some patients with severe obsessive-compulsive disorders, not fulfilling DSM-III criteria for anorexia nervosa, obsessive-compulsive traits such as spending hours cutting and eating small amounts of food in a ritualized manner are present [32].

Anorexia Nervosa in Mental Retardation

Anorexia nervosa has been described in patients with mental retardation [70, 71]. A 15-year-old patient with agitated, withdrawn behavior and an IQ of 62 had a distorted body image and anorexia. This patient was treated with behavior therapy. Anorexia nervosa in the retarded may go undiagnosed because of the misconception that mentally retarded individuals do not develop this disorder [70]. Anorexia nervosa has been reported in a 35-year-old female with Down's Syndrome [71]. Due to the developmental and cognitive delays of retardation the patient only recently experienced adolescent issues (e.g., separation individuation) associated with the onset of anorexia nervosa. Treatment approach involved modification of environment combined with family therapy.

ANOREXIA NERVOSA IN ASSOCIATION WITH MEDICAL DISORDERS

Crohn's disease has been reported coexisting with anorexia nervosa [72,73, 74]. Hershman and Hershman [72] reported a 27-year-old anorectic female with diarrhea and increasing lower abdominal pain who developed a palpable cecal mass with inflamed appendix. At surgery a diagnosis of Crohn's disease was made. Diagnostic confusion can arise between patients with Crohn's disease, atypical anorexia and anorexia nervosa because of the similar symptoms of nausea, anorexia and abdominal pain. In addition these two disorders can coexist.

Blinder et al. [40] noted that a 34-year-old patient following right temporal lobectomy for post-traumatic intractable motor seizures developed anorexia nervosa. Central nervous system disorders have been reported in anorexia nervosa. A 25-year-old female presenting with complaints of poor memory, nausea, ataxia, diplopia and dysarthria was later diagnosed to have Wernicke's encephalopathy. The anorexia, producing a thiamine deficiency, may have caused this disorder. However, thiamine levels were not performed because she presented six months after resuming a normal diet. Anorexia patients developing mental status changes with ataxia and nystagmus should be screened for Wernicke's encephalopathy [75]. A 19-year-old female who presented with both acute, severe depression and anorexia nervosa syndrome subsequently developed petechial skin hemorrhages, suddenly collapsed and died. At postmortem disseminated herpes simplex infection with massive intra-cerebral hemorrhage was note. The sudden onset of depression was due to the herpes simplex infection. The patient's malnutrition contributed to a lowered immunological defense and other susceptibility to herpes simplex [76]. Symptoms of anorexia nervosa have been reported in the initial stage of multiple sclerosis [77].

Anorexia Nervosa in Genetic Disorders

Anorexia nervosa has been reported in genetic disease such as Turner's Syndrome [59] and Gaucher's disease [78]. There are thirteen case reports of patients with the coexistence of anorexia nervosa with Turner's Syndrome, a disorder manifesting a 45-chromosome XO genotype, webbed neck, and gonad hypodevelopment. Endocrine treatment with estrogen at pubertal age may induce sufficient body and weight changes in the Turner Syndrome patient to provoke attempts at dieting or restrictive eating.

Anorexia Nervosa with Autophonia

An interesting report associates anorexia nervosa with autophonia, the perception of one's own voice and breathing. Rapid weight loss seen in a variety of wasting disorders including anorexia nervosa has been associated with autophonia. The therapist should not confuse a patient's complaint of hearing her own voice with transitory psychotic phenomena occasionally seen in anorexic patients [79].

EATING DISORDERS IN ENDOCRINE DISTURBANCE

There have been no systematic studies of abnormal eating attitudes or behavior in classical endocrine diseases such as excess or insufficient thyroid or adrenal states. Excess cortisol levels can be associated with depression, mania and organic mental syndromes and may result in a moderate weight gain. Patients with restrictive anorexia nervosa may have high blood cortisol levels related to increased corticotropic releasing factor (CRF) [80, 81]. Recent findings demonstrating GABA neuronal receptor activation by cortisol suggest CNS inhibitory and stimulatory potential for cortisol with implications for neurobiologic consequences in Cushing's Syndrome, depression, and anorexia nervosa (hypercortisol states) [82]. Cushing's patients may not manifest overtly abnormal eating behavior, however their eating habits may be similar to patients with mild obesity. A slight increase in appetite may be present. In Cushing's disease, there may also be increased urinary-free cortisol which may not be present in either anorexia nervosa or depression. Cortisol, which has a catabolic effect and destroys tissues may possibly have a role in increased appetite to augment protein intake, restoring lost tissue and muscle mass.

A woman age 27-years-old, with a prior diagnosis of anorexia nervosa and a 54 percent loss of body weight, subsequently developed a pituitary corticotrophic cell adenoma with Cushing's Syndrome alleviated by transsphenoidal surgery. Within two years of surgery in the absence of hypercortisolism, anorexia features reappeared [83] suggesting a common CRF-inducing mechanism.

In contrast to Cushing's disease, cortisol insufficiency is found in Addison's disease. These patients may have a seemingly normal appetite, but satiety occurs with minimal food ingestion. Exogenous steroids, when abruptly withdrawn, can produce a similar effect. Delayed gastric emptying seen in eating disorder patients may produce early satiety and feelings of fullness [84, 85]. These effects may persist after renutrition and may be related to gut neuroendocrine dysfunction.

Diabetes Mellitus coexisting with anorexia nervosa and bulimia has been frequently reported [86, 87, 88, 89, 90]. The prevalence of anorexia nervosa with Diabetes Mellitus ranged from zero percent [88] to 6.5 percent [91]. The presence of bulimia ranged from 6.5 percent [111] to 35 percent [143]. Rodin et al. [91] noted a six-fold increase for bulimia over the expected prevalence for nondiabetic individuals. Patients who failed to take their insulin developed glucosoria and thereby effected an indirect chemical method of "purging" [86]. The treatment of Diabetes Mellitus offers patients numerous opportunities to pursue their morbid goal of weight loss by dangerous maneuvers including surreptitious vomiting after bulimic episodes, adjustment of the insulin dose, failure to inject insulin and failure to provide urine samples [86, 92, 93]. Fairburn and Steel [94] noted that girls with anorexia nervosa could skillfully adjust their insulin dosage to match their reduced carbohydrate consumption.

Patients with growth hormone deficiency, which may occur in panhypopituitarism, may have diminished appetite [95]. This syndrome has been identified with non-organic failure to thrive and with maternal deprivation and may simulate idiopathic hypopituitarism. These children may show pica, eat from unusual places such as garbage cans and drink from toilets. They may steal food and polyphagia and polydipsia may alternate with vomiting and self-starvation. Patients may be overweight or underweight for their dwarfed height, but not emaciated. In contrast increased growth hormone levels may occur in malnutrition syndromes including kwashiorkor, marasmus and anorexia nervosa. When the patient is placed in a more normal environment, eating and drinking patterns normalize [96].

EATING DISORDERS IN THE ELDERLY

Morley and Castle [97] have reported atypical anorexia syndromes in the elderly. Anorexia in the elderly was first described in 1890 in Guy's Hospital when it was termed "senile marasmus." Patients were anorexic and died with no apparent cause of death [97].

A spectrum of anorexia occurs in the elderly. In bereavement appetite can be markedly diminished and overt depression may not be apparent. A second anorexic pattern occurs in the elderly where patients decide to stop eating. Denying hunger and refraining from eating, they may become emaciated and die. A distortion of body image is present as they do not consider themselves thin. They deny suicidal ideation and, if asked, wish to be resuscitated in the event of cardiac arrest [97]. One atypical patient engaged in sham eating in that he would chew and then spit out most ingested food. In spite of weight loss, he felt his body size was "just right" [97].

Morley [98] has not seen bulimia in the elderly manifested by binging or purging. However, he considers the almost universal laxative use in the elderly a possible iatrogenic form of purging. Diminished olfactory sensitivity, appetite disorders and impaired taste sensation may contribute to eating disorders in the elderly. Zinc deficiency, sometimes present in the elderly, produces dysguesia and may also have a role in decreasing enjoyment of food [97].

HYPERPHAGIA

Hyperphagia is defined as excessive ingestion of food beyond that needed for basic energy requirements. Ingestion may occupy unusual amounts of time. Eating may be obligatory and disrupt normal activity. In contrast, bulimia usually occurs surreptitiously in defined episodes and is terminated by abdominal pain, guilt or sleepiness.

Hyperphagic conditions may occur in association with central nervous system (CNS) disorders including gangliocytoma of the third ventricle [99], hypothalmic astrocytoma [100], Kleine-Levin Syndrome [101, 102, 103], Froehlich's Syndrome [104], Parkinson's Disease [105], genetic disorders including Praeder-Willi Syndrome (deletion of the long arm of chromosome 15) [105, 106, 107, 108], major psychiatric disorders including anxiety, major depressive disorder [44], depressive phase of bipolar disorder [109], seasonal affective disorder [110, 111, 112], and schizophrenia [113, 114], psychotropic medication, including delta-9 tetrahydrocannabinol [109], antidepressants and neuroleptics [115, 116] and sleep disorders including sleep apnea [117]. Recent evidence evaluating episodic hormone secretion during sleep in Kleine-Levin Syndrome reveals an abnormality in the hypothalmic regulation of pituitary hormones [114].

Hyperphagia Associated with Sleep Disorders

Sasson [117] has noted that inpatients with sleep apnea who are somnolent during the day, there is obligatory eating to induce alertness, thus reducing daytime drowsiness. This hyperphagia has produced markedly increased body weights in such patients. Binge eating behavior and morning anorexia have been described by Stunkard [118] in the context of a "night eating" syndrome, suggesting a component of sleep disturbance. In the Kleine-Levin Syndrome [101] hyperphagia is associated with hypersomia. Recent evidence evaluating episode hormone secretion dorms sleep in Kleine-Levin Syndrome reveals an abnormality in the hypothalamic regulation of pituitary hormones [119].

Hyperphagia Associated with Psychiatric Disorder

Hyperphagia may occur in psychiatric disorders such as depression, anxiety [44] and schizophrenia [113]. A subgroup of patients with anxiety overeat and gain weight [44] as do some patients with unipolar depression [44] and the depressive phase of a bipolar disorder [119].

Rosenthal [110, 112] reported patients with seasonal affective disorder who appeared to have an atypical depression with hypersomnia, compulsive hyperphagia, carbohydrate craving, and weight gain, a syndrome which recurred beginning in the fall of the year and lasting through the winter months, with resolution during the increasing daylight hours in spring and summer.

Lyketsos et al. [113] noted that schizophrenic women were found to give too much time and thought to food and to be preoccupied with food or they were perceived by nursing staffs as becoming anxious and greedy at mealtimes. In addition, it was noted that 60 percent of schizophrenic women were

overweight, in contrast to 33 percent of schizophrenic men. The hyperphagic effects of phenothiazines appear to have only a minor role in increasing appetite.

Arieti [114] noted unusual eating patterns and described a terminal stage of schizophrenia wherein food selectivity was lost and indiscriminate eating, including pica (non-nutritive eating) occurred. A number of medications, including psychotropics and antidepressants, specifically amytriptiline [115, 116], neuroleptics [115] and many other medications [115] increase appetite. Furthermore, Vaupel and Morton [109] noted that a number of abused substances, such as marijuana (Delta-9 tetrahydrocannabinol) increased appetite. Eating disorder syndromes may be found in increasing association with substance abuse with more extensive clinical and diagnostic delineation.

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