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Chapter 30

Pica: A Critical Review of Diagnosis and Treatment

Barton J. Blinder, Stanley L. Goodman, and Phyllis Henderson

INTRODUCTION: OVERVIEW AND DEFINITION

Pica is defined as a pathological craving for either a food item or its constituents or for substances not commonly regarded as food [1]. DSM-III emphasizes repeated nonnutritive ingestion for a period of time as a habitual mode of response [2].

Pica must be viewed in a developmental context (age level, physiologic state, level of cognitive and intellectual development) and also related to sociocultural and historical patterns that may determine the food selection of a people or a region [1,3,4]. Animal studies suggest that pica may result from specific deficiencies or be part of a nutrient-specific appetite [1]. A similar pattern has been inferred in humans [5-12]. Nutrient deficiencies and medical consequences such as iron deficiency, lead intoxication, growth and cognitive impairment, and intestinal obstruction are freqently associated with the idiosyncratic dietary habits [13,14].

Pica has been reported in certain schizophrenic patients [15-18] and is frequently observed in the mentally retarded [19-21]. It has been attributed to delusional beliefs [18], behavioral lag [19,22], and the developmental chaos of autistic children [23].

Historical Perspectives

Cooper [24] extensively reviewed the existing historical literature concerning pica. Table 1 summarizes the varied ideas relating to presumed target patient populations, biological determinants and mechanisms, ecologic variables, and psychosocial correlations.

Etiology

Much of the early work is based on superstition and folklore. Early case studies are conspicuously absent. Most writers deal primarily with pica in pregnant women, although references to its occurrence in both sexes appear. Recommendations for dietary alteration and the empirical use of iron preparations as treatment appeared before iron deficiency was proposed as a factor in the development of pica [1]. Indeed, the empirical treatment most often deemed beneficial involved nutritional fortification [12]. Moral weakness, perverted instinct, and psychological factors are implicated. The ambiguity conveyed in early clinical descriptions continues to the modern era, although several lines of rational investigation have emerged. (see table 1, Historial Perspectives.)

Current efforts to define and explain the phenomenon of pica include (1) developmental studies (vestigial instinct); (2) psychodynamic theories (deprivation conflict); (3) need-state hypotheses that propose nutritional deficit and homeostatic compensation; (4) sociocultural determinants that involve ethnic group traditions and beliefs related to rites of passage, health, and fertility (see chapter in this volume by Freeman on Transcultural Descriptions of the Eating Disorders); (5) consequences of erratic reinforcement in a chaotic unstructured environment (adjunctive behavior model) [12];

Table 30.1 Historical perspectives on the etiology of pica
Cooper (24) and Danford (1982) extensively reviewed the existing literature concerning pica. A summary of the literature pertaining to etiology follows:

Date	Author	Etiology
10 BC	Thompson	Clay lozenges ingested to treat illness and poisoning.
1000	Boezo	Avicenna treated pica with iron.
1542	Aetius	In pregnant women, suppressed menstrual flow rises to the stomach and causes bizarre cravings.
1562	Hubrigkt	Adulterated stomach fluid. Occurs in both sexes, especially women who "yield too much to the state of mind."
1638	Boezo	a) Fetus attracts purer blood causing foul remainder to corrupt the stomach, producing pica. b) A "vitriolic tartar" clings to the stomach lining. c) Visceral obstruction d) Suppression of menses e) Heredity
1668	Ledelius	a) Remnants of food in stomach cause fermented humors to corrupt the appetite b) "Natural appetite" – First of the "need-determined" theorists c) Psychological factors: anger, fear, sadness
1684	Van der Burgh	Passions of the spirit cause nervous excitation in those not strong enough to resist, therefore more common in women
1687	Betten	a) The guardian of the senses is perverted by the lust of the body b) Transmitted in utero from pregnant mother with pica
1692	Maler	Conflict of the spirit as cause of pica
1698	Dehne	An interplay of heredity, "fermental humors" and psychopathology
1719	Schrey	In either sex, in the mind that "judges food badly" perversion of perception of taste and aroma predisposes to pica, as may tendency of infants to mouth inanimate objects
1811	Craigin	In slaves, mistreatment causes a depression, which causes an addiction to earth eating.
1833	Mason	Poor diet and iron lack. The first to suggest iron deficit a) Innate instinct b) Imitation c) Emotional – homesickness, separation from family d) Underlying disease such as worms, yaws, pregnancy e) Poor diet
1840	Dors	An instinctive impulse analogous to that in animals who because ignorance or need fail in each season to procure sufficient nourishment
1845	Hille	Splanchnic nervous dysfunction which at advanced stages becomes "a true condition of the psyche."
1849	Varga	Pica in the insane is the result of "not knowing what else to do with the things they pick up."
1867	Foote	Pica in Jamaicans as a crude remedy for gastrodynia
1876	Gould	Iron deficiency
1879	Kovatsch	Pica caused by worm infestation
1899	Baccarini	Heredity
1900	Simonini	CNS disease
1903	Gros	Instinctive action
1906	Hooper/Mann	Anemia – notes that anemic people eat clay, which paradoxically worsens their anemia
1909	Raynaud	Poor diet
1931	Smith	a) Nutritional. Pica occurs when an organism most stressed nutritionally, during pregnancy, lactation, growth b) Psychological – a change of environment helps
1935	Major	Pica a symptom of anemia and pinworms, disappears when underlying disease treated
1942	Dickins/Ford	Nutritional – noted pica to be higher in children with less iron-rich foods in their diet
1948	Kanner	a) Mental retardation b) Faulty habit training c) Parental neglect
1952	De Castro	Pica a manifestation of "specific hunger"
1955	Arieti	Pica in terminal schizophrenics a behavioral manifestation of lower levels of integration
1962	Gutelius	No relationship between pica and iron deficiency anemia
1969	Coltman	Pica a symptom of iron lack
1977	Youdim	Iron as a cofactor in hypothalmic neurotransmission regulating appetite

and (6) neurobiologic bases of food selection and ingestive behavior in animal investigations (iron deficiency leading to pagophagia, labyrinthine stimulation, and pica [26,27] and iron deficiency and decreased dopamine receptor neurotransmission as etiologic factors in spontaneous pica [28]).

Psychodynamic theorists describe childhood pica as either resulting from maternal and/or paternal deprivation and a maternal fostering of oral defenses against anxiety [29,30] or resulting from excess oral stimulation coupled with aggression toward the mother after the introduction of solid food [30]. A mother may foster oral defenses against anxiety by late weaning, using the bottle as a pacifier, displaying pica behavior herself, or seducing her child into eating nonnutritive substances [29].

In a study of 95 children with pica, Millican et al [29] found 31.2% had a positive history for maternal facilitation of pica concomitant with paternal deprivation. However, 21.4% of a psychiatric comparison group (but only 3.7% of a normal comparison group) also had this dyad. Millican et al [29] emphasize that the critical factor for determining the choice of pica as a symptom is the "shunting" of the child to oral satisfaction. Pueschel et al [31] confirmed these findings in a group of lead-poisoned children with a constellation of inadequate mother-child interaction, paternal deprivation, culturally dependent maternal oral interests, and significant stress factors leading to pica. The remaining normal oral drive may vary in intensity and may be exaggerated when "there are extra pressures in this direction" and there are "inadequate patterns of control for these oral activities" [30].

Frustration of oral drive as a cause of fixation was not seen in the pica group. Oral deprivation may lead to varied developmental and psychostructural deficits (affective, motor, characterologic) that are predominantly nonoral in nature [32,33].

In addition to poor parental supervision and oral overstimulation, maternal pica (63% in Millican's pica group) and cultural acceptance of pica—especially common in families with African lineage and in southern communities [24,29,34], may represent the extra pressures that allow pica to become manifest in a child prone to intense oral focus of drive satisfaction.

A study of children with iron deficiency anemia pica and anemic children without pica suggests that certain psychosocial stressors are significantly associated with pica: maternal deprivation, joint family, parental neglect, child beating, impoverished parent-child interaction, and disorganized family structure [3].

Psychological stress or conflict situations were present in a group with pica compared with matched controls. Education, socioeconomic level, and the presence of neuroses as evidenced by psychometric testing gave equivocal differential results. Ingelligence testing showed no significant difference between the pica and nonpica controls. Both pica cases and controls displayed hypochromic anemia. Psychological stress and environmental disturbances appeared to more specifically contribute to the development of pica behavior than anemia [4].

Lourie's belief that pica is a predictor of later addictive behavior [35], is supported by a longitudinal study by McCord and McCord [36], which showed that a significant number of children with pica later developed alcoholism. Mitchell et al noted the same association and proposed an underlying susceptibility to visceral conditioning as the prevailing mechanism in both pica and alcoholism [37]. Rats exhibit pica in response to gastrointestinal distress in much the same way as other animals display emesis. That this effect is separate from a substance-induced toxic response was shown by the development of geophagia in rats in response to rotational stimulation [26,27]. After pairing saccharin with cyclophosphamide (which induces pica in rats), pica was elicited by the presentation of saccharin alone. A visceral conditioning process was evidenced by the presence of loose stools both in geophagic rats receiving cyclophosphamide and geophagic rats receiving saccharin alone. A physiologic basis for geophagia is implicated, namely gastrointestinal malaise (suggested by Foote in 1867 [38] and by Laufer in 1930 [39]), which persists after the physiologic cause has been removed. Thus the pica persists as the result of the physiologic conditioning.

Animal Models of Pica

There are reports of pica induced in rats by iron deficiency [40], a low-calcium diet [41], various toxins [42], or stress [26,27,25,43].

In the albino rat made iron deficient by venopuncture, a preference for ice eating (pagophagia), rather than water drinking, occurs. With iron repletion, pagophagia disappears [40].

Also, rats ingesting a low-calcium diet voluntarily ingested greater proportions of lead acetate solutions than did iron-deficient or controlled rats. Therefore, calcium deficiency may promote lead pica in rats on a low-calcium diet. These rats showed an increased toxicity to lead exposure manifested by increased body lead [44]. Burchfield et al [43] reported that rats made arthritic by injection of Freund adjuvant subsequently increased their kaolin ingestion. Furthermore, the geophagic arthritic rats may transmit the pica behavior (kaolin ingestion) to naive rats housed with them. Additionally, Mitchell et al [42] reported that rats poisoned with lithium chloride, red squill or cyclophosphamide prefer kaolin to food, again suggesting a toxic stress-induced

pica. Studies of potentially fertile rats engaging in amylophagia (starch eating) revealed lower conception rates. The amylophagic mothers neglected their offspring, contributing to a 100% 24-hour mortality [45]. Another stress, excessive turning (labyrinthine stimulation) of rats, also increased pica [26,27]. These mentioned dietary deficiencies, toxins, and stresses promoting animal pica acknowledge etiologies that may occur in human pica that deserve further experimental verification and replication.

Numerous writers have observed that the pica that is associated with iron-deficiency anemia ceases after treatment with iron [46]. In contrast, several authors have argued that the pica causes the iron deficiency [47-49]. Gutelius et al [50] proposes two types of pica that are recognizable syndromes: (1) children with severe anemia in whom the pica is terminated by treatment of the anemia, and (2) an anemic group with pica that persists after iron therapy. The existence of the latter group suggests the presence of a conditioned response similar to that in animal studies [37].

The theory that the eating of nonnutritive substances is a need-determined behavior is supported by studies of food selection in young infants. Of special interest is a child with rickets who selectively drank milk laced with cod liver oil until his blood calcium and phosphorus were normal and roentgenographic evidence of his rickets had disappeared [51]. Richter [52] demonstrated the ability of adrenalectomized rats to drink enough salt solution to remain symptom-free. This self-regulatory behavior was abolished by sectioning of the taste nerves, indicating the presumptive role of taste in dietary selection. In a later study, Richter [53] proposed that taste thresholds vary with internal needs, as adrenalectomized rats can distinguish far more dilute (1:33,000) solutions of salt than normals (1:2,000). Rolls's [54] studies of food selection suggest two adaptive mechanisms in the control of eating: (1) sensory specific satiety, a person's perception of a specific food as pleasant decreases with increasing intake of that food, while other foods not eaten increase in pleasantness as a function of time since last eaten. This parallels Richter's concept of changing taste thresholds. (2) Neophobia, the avoidance of food not in a person's current food repertoire. Both mechanisms have an adaptive value. Sensory specific satiety leads to increased variety, and neophobia insures against eating possibly dangerous or nonnutritive foods. Perhaps both mechanisms may be impaired or inoperative in pica.

An etiologic role for iron deficiency in clinical studies of pica has been long debated. Lanzkowsky [46] found cessation of pica after iron replacement in anemic dirt-eaters. Catzel [55] found cessation of pica with iron replacement before the peripheral manifestations of anemia and hemoglobin values had been corrected. Jolly [8] suggested that a rise in serum iron was "sufficient to remove the craving" found in pica. In 1984, Libnoch [56] described a woman with erythrocytosis requiring repeated therapeutic phlebotomy who developed pica (geomelophagia-raw potatoes) with normal hemoglobin values but low tissue iron stores. Upon administration of oral iron her serum ferritin, mean corpuscular volume, and serum iron returned to normal and her pica disappeared. The foregoing suggests a relationship between tissue iron depletion and pica. McDonald and Marshall [57] and McGehee and Buchanan [58] also support the role of iron therapy for relief of pica in anemic subjects.

Pica may be a cause of iron deficiency where the nonnutritive substance (clay, starch) interferes with dietary intake or absorption of iron [47,48,59]. However, iron deficit as a cause rather than a result of pica is most clearly seen in patients with iron deficiency and pagophagia (ice-eating). Ice displaces no known nutrients and does not alter the absorption of iron [9,10,60].

Olynyk and Sharpe [61], Von Bonsdorff [62], and Youdim and Greene [63] suggest CNS neurochemical iron-dependent appetite regulation. The studies of Quik and Sourkes [64] and Youdim and Greene [63,65] indicate that systemic iron deficiency results in no predictable alteration of iron-dependent enzymes or changes in CNS catechol neurotransmitter metabolites. Youdim et al [65,66] linked decreased brain iron specifically to decreased dopamine D2 receptors and consequent reduction of several CNS dopamine-driven behaviors. The foregoing studies suggest that further research on the neurobiologic basis of pica is clearly desirable.

Studies that cast doubt on iron deficiency in the etiology of pica include those by Morrow et al [67] and Gutelius et al [7]. Gutelius used a double-blind approach with well-matched, anemic controls and found that although pica decreased with iron replacement, hemoglobin levels rose in both the treated and nontreated subjects. Further, relapses of pica behavior were not associated with a drop in hemoglobin levels. Gutelius notes "the cure of pica, like the cause seems to be a complicated problem involving multiple factors in varying degrees" and "iron medication is not a specific therapy for pica," [7]. The attention shown the families during treatment and an increased awareness of the pica behavior and its antecedents may account for part of the ambiguity. The increase in hemoglobin levels in the nontreated subjects suggests that partaking in a study makes the mothers in both groups more conscious of nutrition despite the lack of any specific advice on the

matter.

The Incidence of Pica

The incidence of pica is difficult to establish due to differences in definition and the reluctance of patients, both male and female, to admit to abnormal cravings and ingestion.

In pregnancy, 51% of patients described cravings for foods, especially sweet foods. In the past up to 50% of southern black women ate starch and clay [1]. Other studies reveal a 33% incidence of amylophagia (starch eating), and Bruhn and Pangborn [68] report 38% of pregnant women experience pica. McGanity [69] stated 20% of 800 pregnant women had a history of pica.

In children ages 1-1/2 to 3 years, pica is considered normal with an incidence greater than 50%. However, persistence of excessive hand-to-mouth movements as in pica is abnormal in children older than three [30]. There is a racial difference in the incidence of pica because 30% of black children ages 1 to 6 have nonfood pica, while only 10% to 18% of white children in this age-group engage in pica. The incidence of pica is highest in psychotic nonwhite children, reaching 50% [1]. Barltrop [70] felt pica decreased with age, indicating about 10% of children beyond age 12 engaged in pica.

It is estimated that lead poisoning may be present in 5% to 10% of all children ages 1 to 5 and in 30% of children with pica [1]. Of children who have lead poisoning, 70% to 90% traced the source of pica to paint chips [1].

In the institutionalized mentally retarded, Danford et al [71] reported a 26% incidence of pica [72]. In contrast, McAlpine and Singh [73] reported an incidence of only 8.4% in the same population. Although both Danford and Singh note the highest incidence of pica at ages 10 to 19, Danford noticed an increase in pica beyond age 70, while Singh noted no occurrence of pica past age 45.

Ethnic differences occur in pica. The majority of cases of lead poisoning in New York were of Puerto Rican extraction. The incidence increases in summer, as vitamin D elevation by sunlight induced synthesis may promote an increased lead absorption [74].

Pica is endemic among sedentary Australian aborigines. As diet and eating customs changed with colonization by the Europeans, and monotonous foods replaced the once varied diet, people wished to return to the traditional folkways and customs. For example, clay has been eaten as a fertility food [75,76]. In Turkey, young women were encouraged to eat clay to enhance their fertility. The similar ideas shared in a black culture encouraged pregnant females, both in Africa and later in the United States, to eat various types of clay to enhance childbearing [24].

Demographic incidence information reveals that pica has been associated with diets that are low in iron, zinc, and calcium compared with a balanced controlled diet [77].

In the mentally retarded, there are changes in incidence of pica with age, IQ, medication, and manifestations of behavior and appetite. The majority of patients with pica are moderately underweight. Pica increases as the IQ decreases. Increased incidence of pica occurred in patients with CNS congenital anomalies and associated medical problems, such as diabetes, deafness, and seizures [19]. Pica increased in incidence in patients taking neuroleptics, which may be related to diminished postsynaptic dopamine (DS) receptor changes [65,28].

Behavioral problems in the retarded associated with pica include stereotypic behavior (52%), hyperactivity (39%), self-abuse (39%), and food-related abnormal behaviors including eating off the floor (73%) and chewing of objects (73%). Pica coexisted with rumination (53%), hyperphagia (47%), and anorexia. There were no racial or sexual differences in pica in the mentally retarded group [19].

MEDICAL COMPLICATIONS

Clinical Description

Literature reports describe specific types of nonnutritive ingestion that occur in different age-groups and social and cultural contexts. Geography, sociocultural factors, and developmental considerations all have been significant in determining the type of pica. Lead poisoning, lead intoxication in children, and social and epidemiologic antecedents as well as complications for development and medical hazards will not be discussed extensively in this section because it is reviewed elsewhere and constitutes a special focused area of study.

Lead Poisoning and Pica

Lead poisoning continues to be a hazard in young children. Inner-city children residing in the 49 million homes with lead-based paint have an incidence of excessive blood lead of 18.6% [78]. The persistence of hand-to-mouth movements in young children, especially from the age of 18 months to 3 years, results in the ingestion of lead-based paint [70]. Lead may enter the bloodstream by inhalation of particulate lead from automobile fumes and from nearby factories using lead-based materials.

Whole blood lead levels greater than 25 μg/dl (Pb25 μg/dl) indicate the child is in a contaminated environment. Elevated blood levels have multiple effects on cognition (including learning impairment and behavior), diminished attention span, and impulsive behavior.

When whole blood lead levels reach 70 PbB μg/dl, an insidious onset of anorexia, apathy, and poor coordina-

tion may occur. At lead levels above 90 μg/dl, lead encephalopathy manifested by gross ataxia, vomiting, lethargy and intractable convulsions may occur. Some children with high lead levels may have no symptoms, while other children may be symptomatic.

Neurologic complications of chronic lead poisoning may present as mental retardation, convulsive disorders, peripheral neuropathy, behavioral disturbance, or any combination thereof.

Treatment of lead poisoning includes stringent dust control by damp cleaning methods, treating a coexisting underlying iron deficiency, assuring an adequate dietary intake of protein and minerals, and careful follow-up of children until blood levels diminish. Chelation therapy with edathamil calcium disodium (CaEDTA), 2,3-Dimercapto-1-Propanol (BAL), and Penicillamine (PCA) can rapidly lower, but not completely eliminate, the lead content in the CNS. In the treatment of acute encephalopathy, and when Pb90-100μg/dl whole blood, immediate treatment with both BAL and CaEDTA is mandatory.

Since the more severe lead poisoning originates in multiproblem families, Chisolm recommends consultation with medical social service departments. Reduction in exposure is the cornerstone of any treatment program. In addition, renovation of substandard housing and systematic screening of children in high-risk areas are imperative [79].

Special types of pica and their medical complications include paper pica, which may lead to mercury poisoning. Olynyk and Sharpe [61] reported a patient with paper pica who was found to have decreased serum iron and responded to iron therapy. In numerous clinical cases, pica was associated with occult iron deficiency, which resulted in the sudden appearance of eating nonnutritive objects such as match heads (cautopyreiophagia) [80] and raw potatoes (geomelophagia) [56]. Arcasoy and Cavdar [81] have shown decreased serum iron and zinc in association with geophagia in Turkish children who may manifest hypogonadism, hepatosplenomegaly and dwarfism. An identical syndrome has been described by Prasad [13] in Iranian children.

The term bezoar derives from the Persian word signifying antidote. These were concretions from the alimentary canal of animals and were thought to have both medicinal and magical properties [82]. Clinically, bezoars can be characterized as tricho (hair), phyto (plants), and gastroliths (mineral or chemical substances). Tricho and phyto bezoars account for over 90% of reported clinical cases [83]. Certain occupational situations (painters who swallow shellac, asphalt workers), medical procedures and treatments (oral contrast radiography, and medically prescribed special diets) may predispose to bezoar formation.

Grant et al [84} described a giant trichobezoar in a 17-year-old female with normal intelligence. The hair ball took up the entire stomach, and gastrostomy was required for its removal. McGehee and Buchanan [58] described trichophagia and trichobezoar in a 2-1/2-year-old female and a 19-month-old female, both with iron deficiency, and irritation and hemorrhage of the gastric mucosa. Singh [85] described severe fecal impaction in two school-age children resulting from sand eating. Gonzales-Espinosa et al [86] described trichophagia and trichobezoar resulting in blood loss, intestinal invagination, and the need for corrective surgery. Uretsky [87] described intestinal obstruction resulting from paper. Additional medical complications reported to result from nonnutritive eating have included intestinal perforation [88], dental complications [89], hyperkalemia associated with geophagia [90,91], hypokalemia and anemia [92], and parasitosis [93-97]. Nine of 23 children with toxocariasis had a history of pica [98]. Patients with pica should be screened for parasitism and other orally transmitted diseases.

Foreign body ingestion may be seen in delusional schizophrenic patients who may ingest glass, pins, or other nonnutritive items [16,18]. Arieti [16] noted that driven nonnutritive eating may be seen in disorganized schizophrenic patients.

Radiographic Diagnosis of Pica

Radiographic findings, which may assist in diagnosis, vary depending on the substance ingested. An abdominal flat plate may visualize chips of lead paint [99], radiopaque particles of clay or soil [100], or foreign objects.

If the ingested substance is sharp, intestinal perforation and the findings of both pneumoperitoneum and a radiopaque foreign body may be present [88]. In addition, a barium swallow may be useful in determining whether a large gastric mass is a larger bezoar, leiomyoma, or carcinoma [84]. Furthermore, of interest is a case of maternal pica of lead-based plaster resulting in an infant with radiographic findings of congenital lead poisoning [101].

Parotid hypertrophy occurs with starch eating [102]. Ulcerative colitis and iron deficiency have been described in an 8-year-old child who ingested masonry [103]. An interesting syndrome of nicotinism and myocardial infarction was described by Neil et al [21] in a psychotic delusional patient who ate tobacco. Neil notes that Kraeplin was the first to document an extraordinary array of inedible materials consumed by psychotic patients and felt that this behavior might be a vegatative sign of psychosis: "a perversion of the appetite."

Pica and Iron Deficiency

Numerous authors report pica associated with iron deficiency [46,104]. Ansell and Wheby [105] reviewed

numerous studies and reaffirmed this association. Gutelius [7] concluded in a double-blind study that intramuscular iron was no more effective than saline injection in reducing pica. However, patients in this study were evaluated at two-month intervals. Since iron generally abolishes pica in less than seven days [46,104], the preferential effect of iron in abolishing pica might not be apparent. Also, the educative approach over the relatively long period of the study may have facilitated the mothers' prohibitions against pica and may have improved mother-child interaction, diminishing stress promotion of pica. The most compelling argument for the association of pica and iron deficiency is suggested by the studies of Reynolds et al [104] and Coltman [9] on pagophagia: the obligatory urge to eat at least one tray of ice daily for at least two months. Ice eating does not reduce iron levels and is not a culturally determined pica. Reynolds [104] found pagophagia associated with low serum iron levels. Iron repletion abolished pagophagia before correcting the anemia. Furthermore, Coltman [9] reported cessation of pagophagia in 19 of 25 women with iron deficiency after iron supplementation for five days with intramuscular injection and 11 days with oral iron administration.

Ambiguity in the sequence of pica and iron deficiency is partly due to the situation in geophagia (clay eating). In geophagia of Turkish clays, a culturally determined behavior, gastrointestinal adsorption of both iron and zinc occurs, producing iron and zinc deficiency. Although the Turkish diet is low in protein and also contains phytate (which binds iron and zinc), the patient may be also iron-and/or-zinc deficient before the geophagia [59].

Coltman [9] proposed that in iron deficiency, iron-dependent peripheral tissue enzymes such as catalase, or cytochrome-c, were deficient. However, he could not explain why the changes in these enzymes would promote pica. Youdim [65] using rats made iron deficient, reported a reduction of specific dopamine receptor (D2) binding sites leading to a down-regulation of dopaminergic activity similar to that found in neuroleptic treated animals. The behavioral response to both pre- and post-synaptic dopamine-acting drugs was diminished.

Another form of pica, amylophagia or starch eating, has been associated with iron deficiency. Thomas et al [106], found that starch inhibited mucosal iron uptake. Keith et al [107] found more severe iron deficiency in pregnant women who engaged in amylophagia.

Table 2 illustrates a number of clinical situations of iron deficiency in conjunction with various types of pica. There are reports of eating match heads or ashes in patients who became iron deficient as a result of colonic carcinoma [80,108]. Thus pica may be a presenting symptom that can alert the clinician to anticipate iron deficit and pursue a careful differential diagnosis [109]. Pica frequently ceases after a few days of iron repletion, suggesting a role for iron loss in initiating and promoting pica.

TREATMENT APPROACHES

Behavioral Treatment

Several behavioral techniques have been used to diminish pica behavior exhibited by mentally retarded patients in a residential setting. Ausman et al [124] reported a time-out procedure to interrupt the pica response. A verbal reprimand was given to a 14-year-old male patient who ate food wrappers, erasers, and string, resulting in intestinal obstruction requiring surgical correction. A paradigm consisting of verbal reprimand setting, response interruption, generalization, and reward resulted in discrimination training. Overcorrection as a procedure was employed by Foxx and Martin [125]. A patient was forced to spit out the pica item immediately followed by administration of oral hygiene consisting of mouth flush, tooth brushing, and wiping of the lips. This procedure was effective in decreasing the occurrence of parasitosis in patients with pica. Matsen et al [126] and Madden et al [127] reported the effective use of overcorrection procedures with a 57-year-old retarded female and three female retarded patients, respectively.

In a critical review of pica treatment, Albin [128] pointed out conceptual differences in the definition of pica: non-food items ingested in contrast to eating food on the floor. He noted that use of experimental tactics such as baiting the patient and the absence of data about generalization and maintenance of improvement made comparative evaluation of different treatment techniques difficult. Furthermore, primarily developmental factors, particularly the perpetuation of a finger-feeding stage interfering with the use of utensils in retarded patients, may be related to the persistance of pica.

Physical restraint alone can control pica, as demonstrated in a series of studies by Bitgood et al [129,130], Bucher et al [131], and Winton and Singh [132-134]. Ten seconds of physical restraint appeared more effective than either 30-second or 3-second restraint. Physical restraint was easy to use, required minimal staff training time, and no specific equipment. Initiation of treatment in the number of different settings was important in promoting generalization of the behavior. Singh and Winton [133] and McAlpine and Singh [73] showed that physical restraint was more efficacious than overcorrection for the treatment of pica. Since institutions are often understaffed, an easily employable brief treatment is desirable.

Table 30.2 Pica and Iron Deficiency Clinical Features and Response to Treatment

Patient Age/Sex	Type of Pica	Associated Clinical Features	Response to treatment	Source
43 YO F	Pagophagia	Anemia 2o to menorrhagia.	Iron Tx abolished pica in 3 wks.	Altafulla et al (115)
7 females & 5 males	Geophagia	Ingested sand and soil. 10 had worms (ascaris). Hb 3g to 10.9g severe anemia.	Intramuscular iron dextran (200-400 mg) cured pica 1-2 wks after treatment.	Lanzkowsky (46)
46 YO F	Geophagia	Ingested chalk and plaster for 2 yrs. Hemorrhages.	Pica abolished in 8 days by daily iron injections (100 mg)	Duc et al (121)
45 YO F	Geophagia	2 abortions, 23 blood donations. Snuffed clay dust every 10 min. and ate clay dust for 2 yrs. Hb 9.9 g.	Pica abolished after 20 iron injections. (1250 mg)	Hadnagy et al (122)
33 males & 35 females ages 3-24	History of geophagia 3-15 yrs.	Growth retardation & hypogonadism. Hb 5.7 + .2g hepatosplenomegoly	Iron deficiency anemia was corrected by intramuscular iron dextran. Of 68 patients 22 received additional zinc sulfate (120) mg) daily for 6 mo.	Cavdar et al (59)
17 YO F	Geophagia	Hb 4g, Hypokalemia	Anemia responded to intramuscular iron dextran Tx. Pica abolished after 8 mo. continued treatment.	Mengel et al (92)
32 YO F	Geophagia	Hb 10.5 2o menorrhaghia, soil ppt. asthmatic attack	Anemia responded to oral iron.	Krengel & Geyser (123)
53 YO F	Tomato seed craving	15 yrs ago partial gastrectomy, esophageal web. mechanical dilation relieving dysphagia. Hb 7 g. Hysterectomy for menometrorrhagia 4 yrs. ago.	Parental iron abolished pica	Coleman et al (119)
5 males ages 18-51	Pagophagia	Anemia due to GI bleeding	Iron Tx abolished pica in 3 of 4 men. Pica reappeared in 1 male due to bleeding ulcer. Iron Tx and surgery abolished pica.	Reynolds et al. (104)
18 females	Pagophagia	Ingested 2-11 glasses of ice daily. Anemia due to menorrhagia. Hb 5-10.2g Serum iron 0 to 52 mg.	Pica abolished by oral iron	Reynolds et al. (104)
25 females	Pagophagia	Ingested at least 1 tray ice cubes daily. Severe menorrhagia except for 3 patients.	Completely resolved by treatment with iron insufficient in amount to correct either the anemia or iron lack.	Coltman (9)
46 YO F	Pagophagia	Ingested 20-40 ice cubes daily, anemia 2o gynecologic loss.	Iron abolished pica in 4 days.	Sacks (111)
35 YO F	Pagophagia	Ingested 6-8 trays of ice cubes daily for 5 months.	Oral iron abolished pica.	Coltman (10)
33 YO F	Pagophagia during pregnancy. Concurrent cigarette ash eating & geophagia.	Anemia 2o to carcinoma of ascending colon	Oral iron & blood transfusions abolished pica.	Desilva (113)
9 females ages 19-78 & 4 males	Amylophagia (in form of rice, potatoes & bread)	In women, anemia 2o to gynecologic loss. In 3 men, anemia 2o to GI bleeding. In 1 man anemia 2o to hemorrhoids. Hb 5.0-10.2g	Pica abolished by iron Tx.	Reynolds (104)

Table 30.2 Pica and Iron Deficiency Clinical Features and Response to Treatment (continued)

Patient Age/Sex	Type of Pica	Associated Clinical Features	Response to treatment	Source
41 YO F	Amylophagia (in form of cornflakes craving)	Anemia 2o to malnutrition & menorrhagias	Iron abolished pica	Altafulla (115)
44 YO F	Lectophagia	Ingested 4-5 heads lettuce daily for several months. Anemia	Oral iron Tx abolished pica in 1 mo. Hb14.0.	Marks (116)
78 YO F	Pagophagia to lectophagia	Hematocrit 27, enzyme changes consistent with MI. At discharge began to consume 3 trays ice cubes daily. Cut to 2 cups/day; began to eat many heads lettuce/day. Neoplasm and lesion of colon.	Responded to units of packed cells. Gave total remission of signs & symptoms	Moss et al. (108)
26 YO F	Geomelophagia (craving for raw chilled potatoes)	Symptomatic erythrocytosis requiring phlebotomy. Menorrhagia. Hb 17g Serum iron 67.	Oral iron Tx. Returned	Libnoch (56)
68 YO F	Geomelophagia	Anemia 2o to GI bleeding. Lung cancer. Hb 9.3g. Serum iron 13.	Iron Tx (3x300 mg/day) abolished pica after 1 wk.	Johnson and Stephens (117)
42 YO F	Olive craving	Ate approximately 5-7 jars green olives/wk for 4 mo.	Oral ferrous sulfate (3x300mg/day) diminished craving in 1 wk. In 2 wks down to 1 jar/wk. After 1 wk pica cured	Chandra and Rosner (118)
33 YO F	Sodium chloride craving	Hb 9.2 g. Serum iron 28	Iron Tx abolished pica in 2 wks.	Shapiro & Linas (110)
33 YO F	Tea leaves craving	Ingested 250g/day Anemia 2o to gynecologic loss	Iron Tx abolished pica after 6 days	Sacks et al. (111)
46 YO F	Paper craving	Mercury poisoning. Blood level 251 ppb Hg	Iron Tx abolished pica in 1 wk	Olynk & Sharpe (61)
37 YO F	Paper craving	Anemia 2o to chronic menorrhagia, ageusia, dysomia	Oral iron sulfate (300mg) and zinc (24mg) abolished pica 1 mo. after treatment	Chisholm and Martin (112)
48 YO F	Cautopyreiophagia	Ingested ashes of 15 burnt matchbooks daily. Hb 6.5g Serum iron 14. GI bleeding due to lesion in cecuma and adenocarcinoma of left lobe of liver.	Iron Tx, operations & transfusions abolished pica	Perry (80)
76 YO F	Magnesium carbonate	Micocytic hyperchromic anemia; consumed 60-80g/day 3 yrs prior to admission. Hb 5.7g	Repeat admissions about each mo. Given oral & parenteral iron. No blood loss site found. Only improved in hospital.	Leming et al. (47)
21 YO F	Paradichloro benzene craving	34 wks pregnant. Hb 6.6g	Treatment parenteral iron, folic acid abolished pica.	Campbell & Davidson (114)
17 YO F & 34 YO F	aspirin craving	Ingested 4-6g aspirin/day. Anemia 2o to metrorrhagia and menorrhagic respectively	Pica abolished by iron Tx after 8 days	Sacks et al. (111)

Often the control of pica in mentally retarded adults with complicated histories presents a clinical problem that must be approached through individualized design and the presence of aversive consequences. Friedin and Johnson [135] described the treatment of copraphagia by linking it to aversive consequences such as delay of a shower. Most recently, Singh and Winton [134} have demonstrated that aversive oral hygiene when used alone was as effective as physical restraint and overcorrection. The oral hygiene technique was easily taught to staff and could be applied early in a variety of circumstances.

Behavioral treatment approaches in pica involve careful observational analyses and the application of consistent contingent responses by a trained staff.

Nutrient Approaches

Nutrient treatment of pica has been reported for almost 1,000 years. (Table 1) In the tenth century, Avicenna added iron to wine as a treatment for earth eating [1,24]. There are numerous case reports [46,104] indicating that iron treatment will abolish pica. Cavdar et al [59] reviewing a geophagia syndrome (iron-deficiency anemia, hepatosplenomegaly, hypogonadism, and dwarfism found in Turkey and Iran) reported that iron administration successfully treated both anemia and pica, but zinc was necessary for linear growth and pubertal advance. Zinc deficiency may also be associated with pica. Hambidge and Silverman [136] reported a 10-year-old boy with sickle cell disease who ate kitchen cleanser. Following oral administration of zinc, serum levels of the metal almost quadrupled, and pica was terminated [137]. A critical review of clinical reports reveals that the majority of authors verify the efficacy of iron repletion in abolishing both food and nonfood pica associated with iron deficiency states of different etiology. This suggests a common central pathway such as mediation by decreased CNS dopamine neurotransmission reported to be a specific result of iron deficiency states [65,28]. In animal studies Youdim [65] reported a 50% diminution of CNS dopamine (D2) receptors with iron depletion.

Pharmacologic Treatment of Pica

No specific pharmacologic treatment studies of pica exist. Jakab [138] reported that thioridazine reduced pica as well as a number of other problematic behaviors such as aggression in hospitalized mentally retarded patients. However, Danford and Huber [19] noted an increased pica incidence (39%) in a subgroup of institutionalized retarded patients receiving neuroleptic medication in contrast to a 25% incidence of pica in a medication-free group. Does diminished dopaminergic neurotransmission promote pica? Decreased dopamine transmission resulting from both iron deficiency and administration of neuroleptics may be a critical determinant in the appearance and maintenance of pica. A pharmacologic approach that increases dopaminergic transmission (bromocriptine, ritalin) may be worthy of investigation in a subgroup of patients in whom pica is both refractory and hazardous.

Psychosocial Treatment

Lourie [139] recommended a psychoeducation treatment approach. Mothers would be instructed about the danger of pica that could result in lead poisoning. Social workers would provide a social support system for mothers who may be depressed, meet their dependency needs, and help mothers be more available to the children. Strategies of prohibiting pica would be taught to mothers so that they would spend more time with their children and interrupt pica behavior. Lourie et al [30] suggested that identifying families at high risk for pica could function as a primary prevention.

CONCLUSION

Pica in man is indeed a complex behavior with multiple determinants ranging from demands of tradition and acquired tastes in the cultural context to presumptive neurobiologic mechanisms (iron deficiency, CNS neurotransmission, physiologic conditioning). Clinical consequences of pica may have broad epidemiologic implications as in lead intoxication and geophagia in children leading to severe impairment of intellectual and physical development. Acute and chronic medical complications may pose surgical emergencies (intestinal obstruction from bezoars) as well as more subtle encroaching symptoms in parasitosis, intoxications, and resulting nutritional deficits.

Although pica as a naturally occurring behavior in animals has apparent utility in aiding digestion or overcoming nutritional deficit, its presence in man appears to be the result of culturally contrived or pathophysiologic circumstances, and any adaptive value remains obscure. The occurrence of pica in pregnancy [40], mental retardation, schizophrenia, and autism suggests a psychobiologic significance to link a disturbance in food selection to other complex neuroendocrine medicated responses.

Treatment approaches have been primarily preventive, educational, and directed toward modification of pica behavior. Iron repletion has dramatically reversed pica for those patients whose clinical symptoms were more clearly coincident with iron deficiency from nutritional or covert medical causes [141,142,143].

Further investigation of pica may clarify the normal

psychobiology and developmental progression of food selection in man, the intricate role of sociocultural influences, and the significance of appetite and ingestive disturbances in neuropsychiatric disorders. Special focus should be given to the high incidence of pica in the mentally retarded and to the role of iron deficiency (the single most prevalent nutritional deficiency in world population studies) in the etilogy and perpetuation of eating disorders.

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