Chapter 29

Rumination: A Critical Review of Diagnosis and Treatment

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INTRODUCTION

Rumination, an uncommon disorder occurring from infancy throughout adulthood, is derived from the Latin *ruminare*, "to chew the cud." Merycism, derived from the Helenic, is the act of post-ingestive regurgitation of food from the stomach back into the mouth, followed by chewing and reswallowing [1]. The two terms are often used interchangeably. Rumination is associated with medical complications such as aspiration pneumonia, electrolyte abnormalities, and dehydration [2] and is considered in the differential diagnosis of vomiting [3] and failure to thrive [4] in infants and young children. From latency through adulthood, rumination frequently has a benign course [5]. Recently it has been associated with bulimia [6,7], anorexia nervosa, and depression [5,105,109]. Past studies have ascribed the disorder to lack of emotional reciprocity and attunement between mother and child stemming primarily from maternal depression and anxiety [8-10]. Medical disorders such as gastroesophageal reflux and hiatal hernia [2,8,11,12], also are present in the population of ruminating children. Applications of formal behavioral contingencies in treatment have led to describing ruminatory activity as a habit disorder [13-15].

In DSM III [16] rumination is designated as a disorder of infancy [307.53]. The infant shows "a characteristic position of straining and arching the back with sucking tongue movements and the gaining of satisfaction with rumination" [16]. Diagnostic criteria include repeated regurgitation without nausea or associated gastrointestinal illness for at least one month following a period of normal functioning. Weight loss or failure to make expected weight gain occur often [16]. Irritability is noted between regurgitations and hunger is often inferred by the observer. Although the disorder occurs most frequently after 3 months of age, it has been reported in a 3-week old infant [17] and in the neonatal intensive care unit [4]. Consequent failure to thrive with malnutrition may produce severe developmental delays [15]. Rumination has been described in families over four generations, and learning to ruminate by imitation has been suggested [18].

Rumination may be underreported, with only complicated cases (malnutrition, electrolyte disturbances, hiatal hernia) referred to a gastroenterologist and minor cases treated by parent or primary physician. Rumination in anorexia nervosa and bulimia may be underreported due to omission of inquiry in the systematic medical history and reluctance of patients to volunteer specific clinical information [5,6,7,109].

The course of rumination may depend on the age of the patient and the severity of the complications. Mortality can be as high as 25% to 40% in infants [19]. Although the infant may manifest hyperphagia, post-ingestive regurgitation leads to progressive malnutrition (ie, a sham eating sequence). In the ruminating adolescent bulimia and affective disorder may be present [7]. Rumination in adults has been associated with gastric
The Eating Disorders

Table 29.1 Two vomiting syndromes of infancy

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Nervous Vomiting</th>
<th>Infant Rumination</th>
</tr>
</thead>
<tbody>
<tr>
<td>The nature of the vomiting</td>
<td>Involuntary</td>
<td>Voluntary</td>
</tr>
<tr>
<td></td>
<td>Visceral</td>
<td>Behavioral</td>
</tr>
<tr>
<td></td>
<td>Purposeless</td>
<td>Self-stimulation</td>
</tr>
<tr>
<td>Age of Onset</td>
<td>As early as newborn</td>
<td>After 3 months</td>
</tr>
<tr>
<td>Mothering</td>
<td>Attentive but dyssynchronous; increases rather than relieves tension.</td>
<td>Emotionally distant.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Little reciprocal interaction</td>
</tr>
<tr>
<td>Typical Circumstances</td>
<td>During the baby's response to environmental stimulation</td>
<td>In the absence of environmental stimulation</td>
</tr>
<tr>
<td>of vomiting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Management</td>
<td>Lessening excessive stimulation. Alleviating the tension-producing quality of mother-infant interaction</td>
<td>Increasing environmental stimulation.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Fleisher, 1979</td>
<td></td>
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</tr>
</tbody>
</table>

carcinoma [20] and anemia [21,22]. More frequent medical complications occur in the retarded [23], with a mortality rate of 12% to 20% [24].

Extended posttreatment evaluation of patients with rumination is rare. Investigators most often report a one- or two-year follow-up [3,4]. Kanner [19] stated that long-term follow-up of patients with rumination revealed a notable subsequent appearance of psychiatric disorder.

HISTORICAL PERSPECTIVES

Rumination was first discussed by Fabricius Ab Aquapendendente in an adult male with a hiatal hernia [25]. Lushka [26], and later Herbst et al [11], described rumination associated with hiatal hernia. Grulee [27] first suggested that attention be directed to the psychic condition of the child and that distracting the infant would diminish the rumination. Lourie [8] observed an abnormal mother-infant relationship and used a substitute caretaker for treatment. Since 1968, behavioral treatment techniques have included peripheral electric shock [14,28], lemon juice [29], and food satiation [24,30]. Chatoor and Dickson [31] proposed an integrated psychodynamic, biologic and behavioral etiology and treatment.

DEVELOPMENTAL AND PSYCHOSTRUCTURAL FACTORS FROM INFANCY THROUGH ADULTHOOD

Lourie [8] noted that both understimulation (maternal absence or neglect) and overstimulation (excessive inappropriate caretaking) were associated with rumination. These infants fail to develop basic trust in the maternal caretaker, resulting in a failure of attachment [32]. Such children have been characterized by Lourie as passive and sensitive to rejection, with rumination serving to relieve inner tension states. Also, a physical source of irritability such as hiatal hernia has been associated with rumination [11]. From a psychodevelopmental perspective, rumination might be viewed as voluntary self-feeding compensating for an inadequate maternal-infant relationship. Rumination becomes a defensive habit pattern with both functional autonomy and a pleasurable self-reinforcing effect. Precursors of pleasurable swallowing and "libidinization" of the esophagus may date from prenatal experiences of the ingestive passage of amniotic fluid [33].

Infancy and Early Childhood

Cameron [34] has delineated the classical description of rumination in infancy. He noted purposeful movements of the abdomen, mouth, and tongue resulting in the pleasurable ejection of previously ingested food. Rumination has been reported in infants with disorders including reflux esophagitis [2], hiatal hernia [11,12,35], necrotizing enterocolitis, malabsorption and malnutrition, failure to thrive, prematurity, severe bronchopulmonary dysplasia [4], growth failure [17], autism [31], infantile spasms [36], grand mal epilepsy [36], tuberous sclerosis [37], heroin withdrawal [38], barbiturate withdrawal [35], labile autonomic nervous system [31], object loss [8,39], and infection [18,35].

Repetitive self-stimulatory behavior (head banging, body rocking, and genital and anal-fecal play) resistant to maternal interruption has been observed in ruminating infants [10,31,35]. Rumination has been described in one member of a pair of both monozygotic [35] and dizygotic [17] twins.

Fleisher [40] has differentiated rumination from nervous vomiting occurring in tense, anxious, labile infants (Table 1).
Latency

Rumination is rare in latency-age children who are not retarded. The few cases that have been documented have been described adequately elsewhere [5,18,31,39,41]. In general, infantile predisposition to rumination and variation in both symptom frequency and intensity with emotional arousal are notable.

Adolescence

In nonretarded adolescents, rumination associated with anorexia nervosa [5,18], bulimia [6,7], anxiety and depression [5], and iron deficiency [22] has been reported.

Adulthood

Adult rumination is a chronic disorder [5], except when associated with bulimia [7]. The individual episode is postprandial, without nausea, effortless, and predominately involuntary. It may occur spontaneously after a hastily eaten meal, causing embarrassment or may appear seemingly voluntary and pleasurable [1,5,20]. The symptomatic presence of active ruminatory behavior varies from as little as six months to a lifetime [5,18]. Patients may complain of food returning to the mouth, belching, precordial distress (possibly due to esophagitis), indigestion, halitosis, and excessive dental deterioration [5,105].

Long [20] notes two uses of rumination: (a) as a sham eating technique; and (b) to eat and dispose of foods contraindicated medically (fatty foods, meat) yet having strong palatability and preference for the patient. Thus a patient with gallbladder disease would regurgitate and extrude fatty foods, preventing painful cholecystitis.

The presence of specific psychiatric disorder in adult rumination is undefined. Diagnoses noted in prior reports include:

1. Neuroasthenia [18]
2. Performance anxiety; somatic delusions of fatal illness [20]
3. Emotionally irritable, immature, and passive [45]
4. Schizophrenia hysterical traits [1]
5. Atypical personality [46]
6. Affective disorder [5]
7. Eating disorder-bulimia [5,6,7]

There were no structured psychiatric evaluations or uniformity of diagnostic criteria noted in the literature until Levine et al [5] evaluated nine patients with both psychiatric interview and questionnaire. Interviews revealed a family psychiatric history or disturbed family relationships in four of the patients; three patients had psychiatric histories (overdose, anorexia nervosa, brief reactive depression); patients had personalities that were anxious (seven of nine), obsessional (five of nine), or sensitive (six of nine); and four of five adult patients had psychosexual and marital problems. However, on formal mental status exam, only one of the nine patients had current psychiatric symptoms.

The results of questionnaires revealed that mild traits of anxiety, hysteria, and neuroticism were present. In only one patient did symptoms interfere with psychosocial functioning, and the group revealed no evidence of a current psychiatric illness. The authors concluded that substantive psychiatric disorder was absent. However, the findings are suggestive of affective spectrum disorder (depression, anorexia nervosa, overdose) in three of five adults and significant family history of psychiatric disorder in four of eight patients whose family history was accessible. An instrument such as the SADS [47] might have been a more significant diagnostic tool for detecting psychiatric disorder in the adult rumination group studies [105].

Fairburn and Cooper [7] report rumination lasting at least 12 months in 7 of 35 female bulimic patients. Three patients had postprandial effortless daily regurgitation. The patients complained of losing control of eating with shame about their rumination. All patients had disturbed eating habits, abnormal attitudes toward body and shape, and high psychiatric morbidities. In the subgroup of bulimic ruminators, compared with the bulimic nonruminators, a history of both anorexia nervosa and psychiatric treatment for an eating disorder was more prevalent. The habit by itself was difficult to stop, but successful treatment of the bulimia led to cessation of the rumination. Blinder [6] reported a subgroup of normal-weight bulimic patients with primary ruminatory behavior antedating bulimic symptoms. The patients were more likely to be polyphagic during binge episodes rather than demonstrating the more usual specific carbohydrate preference. Ruminatory behavior shifted to regurgitation during adolescence to aid in weight control. Ruminators may not show the pattern of impulsive behavior, affective disturbance, or family history of alcoholism seen in other patients diagnosed as bulimic.

The only report of psychoanalytic treatment emphasized unconscious anger toward authority figures who were aggressively ejected representationally by the ruminatory behavior. Interpretation of unconscious conflict led to cessation of the ruminatory behavior [48].

There may be two adult subgroups of ruminators—one group with minimal psychiatric problems and the second subgroup with an associated eating disorder, such as anorexia nervosa and bulimia [109]. Since patients are reticent about their illness, a diagnosis of psychiatric disturbance may be undetected [110].

Rumination Associated with Central Nervous System Disorders and Retardation

Rumination is associated both with CNS disorders and mental retardation [49]. Eating disorders are prev-
alent in the institutionalized retarded (pica 25%, anorexia 7%, and rumination 2.7%) [23]. Frequently individuals with pica also exhibit rumination [49]. Postigious gastroesophageal reflux has been associated with mental retardation, CNS lesions such as cerebritis, dilated ventricles, cerebral palsy, and sudden infant death syndrome, apnea, and laryngospasms [50]. Rumination has been associated with tuberose sclerosis [37], hypsarrhythmia [51], infantile spasms, and grand mal seizures [36].

Danford [23] summarized several clinical features in the retarded associated with the presence of rumination. These included male predominance, self-abuse, other food-related behaviors (pica, hyperphagia, anorexia), and medical complications.

Rumination in the retarded appears to be a self-stimulating behavior that relieves internal tension states that are blocked from social release because of marked communication deficit and inability to seek out external stimulation [51].

Although several recent reports of behavioral treatment of retarded ruminants have appeared using aversive conditioning (localized electroshock [14], and lemon juice [29]), there is little discussion of environmental changes that could have precipitated the rumination. A retarded child may suffer significant object losses both when being taken from the family to an institution and when staff changes occur within the institution [53]. A case report [54] described a 10-year-old boy who lost weight and started ruminating following institutionalization and separation from family. Treatment by increasing environmental stimulation abolished the disorder; thus, prompt social stimulation and reinforcement may abort or terminate the rumination disorder related to institutional adjustments.

THEORIES OF RUMINATION

Behavioral

Since 1968 there have been many reports documenting behavioral treatment of rumination [13]. Behaviorists report effective treatments that diminish rumination within two weeks, in contrast to psychodynamic treatments requiring four to five weeks [55].

Behavioral theory explains rumination as a habit pattern. Reinforcement enhances and maintains a specific behavior that is temporally linked to its consequences. A positive reinforcement such as food, increases the frequency of an antecedent behavior. Maternal attention (especially following rumination) to a child who is receiving inadequate nurturing may increase (reinforce) the rumination [54]. Rumination also allows the child to obtain increased attention in the form of medical treatment. Wright and Thalassino [56] consider rumination to be a “learned illness behavior.” Remission of chronic ruminative vomiting occurs through a reversal of social contingencies. Behavioral theories focus on conditions that maintain rumination [13]. Lavigne and Burns [55] believe that rumination is an operant behavior maintained by its consequence and that it is a learned habit that can be extinguished.

A habitual response characteristic of rumination is suggested by a seeming voluntary quality, frequent waxing and waning with environmental stress, and extinction in response to aversive stimuli.

Association with Affective Disturbance

Four lines of evidence linking rumination and affective disorders will be presented. First, infants and children with rumination appear sad and withdrawn [8,9,31,35]. Lourie [8] described a ruminating child who developed features of an anaclitic depression due to the absence of a satisfactory love object (see section on Developmental and Psychodynamic Factors). Another child with rumination and hiatal hernia, age 7 months [8], was described as being withdrawn, expressionless, crying a great deal, irritable, and sleepless. Both cases demonstrate a passive (affective) reaction to helplessness in the face of psychic or physical pain. Several reports [8,10,17] described the emotional unavailability of a mother to her child because of maternal depression and feelings of rejection toward an unwanted infant. The child suffers a significant object loss (perceived or imagined) of the primary caretaker. This conceptualization also relates to Lourie’s notion of understimulation in infants with ruminatory disorder [8]. An animal model has also been observed [108].

Lourie [8] noted passivity and diminished affective expression of needs in infants with rumination. Such behavior could foster parental confusion in responding to the child’s immediate needs and lead to frustration, helplessness, and depressive affect. Lourie also noted that these children are markedly rejection-sensitive, a trait observed in atypical depression [57].

Second, there is a subgroup of children for whom object loss is a manifest onset condition for the appearance of ruminatory behavior [8,39,42,53,54,58-60]. A review of the literature reveals that object loss is the most frequent psychosocial onset event associated with rumination (Table 2).

A pleasurable self-stimulating component of ruminatory behavior may serve as a defense against the pain of object loss [33,61-63]. Protest, despair, and withdrawal, which are generally associated with object loss [32], may also be developmentally specific clinical features in the symptom context of rumination following loss.
**Third, observations linking ruminatory behavior in adults with depressive symptoms, anorexia nervosa, and bulimia have been described in the section on Biologic Determinants [5-7].**

**Fourth, maternal affective disorder may lead to both a genetic factor in the infant and deprivation consequences to nurturance contributing to increased risk to the infant for both mood vulnerability and ruminatory disorder. There may be a subgroup of infants and children with rumination who have an affective disturbance, rejection sensitivity, passivity, and increased incidence of psychiatric disorder [57].**

Diagnostic procedures measuring biologic state and trait markers for affective disorders might be useful in further defining the relationship of rumination subgroups to other specific psychiatric disorders [64]. Prospective follow-up of ruminators, noting whether a greater-than-normal incidence of affective or other psychiatric disturbance occurs, would clarify this posited association.

**BIOLOGICAL DETERMINANTS AND MEDICAL CONSEQUENCES**

**Reflux Subtypes**

Proponents of a biological etiology of rumination equate rumination with gastroesophageal reflux. Winter [65] found abnormal gastroesophageal acid reflux, esophagitis, and normal or diminished lower esophageal sphincter pressure in infant and child ruminators. Why children with gastroesophageal reflux develop rumination remains a mystery, although the psychological context is considered important. Conversely, other gastroenterologists [3,66] have evaluated ruminators uncovering no significant gastrointestinal structural or motility disturbances. There may be two subgroups of ruminators; one with significant gastrointestinal problems such as reflux or hiatus hernia, and another with no significant gastrointestinal problems.

Up to 20% of children who spit up food or vomit during the first year of life have gastroesophageal reflux defined as “a failure of the sphincter mechanism at the junction of the esophagus and stomach that allows acidic gastric material to flow into the esophagus,” (pg. 25) [12].

Reflux of acidic gastric material can cause peptic esophagitis with associated chronic blood loss, iron deficiency anemia, and possibly hematemesis. Esophagitis may diminish lower esophageal sphincter pressure and further increase reflux.

Reflux may be associated with vomiting and failure to thrive. Rumination is considered in the differential diagnosis of psychogenic vomiting and nonorganic failure to thrive (NFTT). Complications of gastroesophageal reflux, such as aspiration pneumonia and esophageal stricture, are often treated by surgery [2].

Reflux has also been associated with Sandifer’s Syndrome [67]. This disorder is especially interesting to psychiatrists because the patient who displays headcocking, abnormal movements of the head and neck, and unusual postures may be misdiagnosed as having a tic or dystonic disorder. These abnormal postures occur during gastroesophageal reflux in the child with hiatus hernia. Surgical repair of the hernia abolishes reflux, terminating the abnormal movements within several days postsurgery.

Jolley et al [68] note three patterns of gastroesophageal reflux. Type I occurs in patients who have

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**Table 29.2 Onset of Rumination Associated with Object Loss**

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Clinical Features</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>5-1/2 mo.</td>
<td>M</td>
<td>Ruminated from age 4 months. Separation from primary caretaker. (grandmother). Depressed mother</td>
<td>Chatoor and Dickson (31)</td>
</tr>
<tr>
<td>6 mo.</td>
<td>M</td>
<td>Mother initiated full time work. Failure to thrive and rumination.</td>
<td>Murray et al (59)</td>
</tr>
<tr>
<td>9 mo.</td>
<td>F</td>
<td>Monozygotic twin; mother returns to work.</td>
<td>Chatoor and Dickson(35)</td>
</tr>
<tr>
<td>11 mo.</td>
<td>M</td>
<td>Developed rumination after father left home abruptly; mother depressed.</td>
<td>Lourie (8)</td>
</tr>
<tr>
<td>5 yr.</td>
<td>M</td>
<td>Retarded; developed rumination following death of 8-year-old sibling.</td>
<td>Menolascino, personal communication (60)</td>
</tr>
<tr>
<td>7 yr.</td>
<td>M</td>
<td>Developed rumination at 18 months shortly after family moved away. from caretaking grandmother</td>
<td>Griffin (39)</td>
</tr>
<tr>
<td>8 yr.</td>
<td>M</td>
<td>Object loss upon institutionalization was followed by rumination which fluctuated in conjunction with presence or absence of substitute caretaker. Rumination ceased upon return home.</td>
<td>Menolascino (53)</td>
</tr>
<tr>
<td>11 yr.</td>
<td>M</td>
<td>Developed rumination at age 6 after multiple object losses occurred following transfer from home to institution.</td>
<td>Wright and Menolascino (54)</td>
</tr>
<tr>
<td>13 yr.</td>
<td>F</td>
<td>Rumination developed at age 9 within one year of mother's death and father's remarriage.</td>
<td>Chan, personal communication (42)</td>
</tr>
</tbody>
</table>

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_Nutritional Characteristics & Consequences_
continuous postcibal reflux and large hiatal hernias, which frequently require antireflux surgical procedure. In type II, a functional motility disorder suggesting delayed gastric emptying appears to be important in infants with discontinuous reflux [69,70]. These infants had frequent gastroesophageal reflux for two hours postcibally, antral pylorospasm [69], increased lower esophageal sphincter pressures, high incidence of pulmonary symptoms, and nonspecific watery diarrhea. The mixed (type III) pattern of gastroesophageal reflux occurred in a small number of infants who exhibited features of both type I and II patterns. Dodds et al [71] noted the association of gastroesophageal reflux (GER) in adults with (1) continuous low esophageal sphincter pressure, (2) normal pressure with momentary drop in pressure, or (3) increased abdominal pressure.

Geffen [22] posited that rumination occurs because of an increased pleuroperitoneal gradient across the diaphragm with simultaneous relaxation of the cephapharyngeal and lower esophageal sphincters. Rapid gastric peristalsis with a contraction of the abdominal musculature, which is unconscious in certain patients, further increases this pressure gradient [106,107]. Incompetence of the lower esophageal sphincter, secondary to hiatus hernia, exacerbates this process. Herbst et al [11] discussed the mechanism of rumination observed during fluoroscopy in a 6-year-old child. The esophagus distended with barium up to the superior esophageal sphincter. When the child made sucking movements of the mouth and tongue, the superior esophageal sphincter opened and barium flowed into the mouth. Normal deglutition followed and initiated a peristaltic wave that emptied the esophagus [11]. The mechanism of rumination may not differ greatly from that of gaseous eructation [22].

Levine et al [5] speculated that an unconscious postprandial intraabdominal pressure occurs with coordinated relaxation of the upper and lower esophageal sphincter. Of the nine cases he reported, one patient had a large postprandial pressure wave starting first in her stomach, then spreading to the esophagus. He also posited that rumination was a benign habit disorder.

Esophageal motor dysfunction has been associated with reflux and rumination. A progressive esophageal peristaltic wave is normally present after the swallowing of food. In rumination, uncontrolled peristaltic movements are seen [72]. Esophageal contraction abnormalities [73] producing reflux are seen in children as well as adults [74]. Emotional stress in the infant or child may produce esophageal contraction abnormalities leading to reflux and rumination [75]. Prugh [76] finds a strong association between emotional stress, dyadic mother/infant disturbance, and upper gastrointestinal dysfunction [77].

Herbst et al [11] discussed three cases of hiatus hernia associated with rumination. The rumination terminated after surgical repair of the hiatal hernia. He suggests that the abnormal findings associated with rumination should be viewed as parts of an extended syndrome of presentation of gastroesophageal reflux [67].

A second type of gastrointestinal pathology in which the passage of food through the stomach to the duodenum is impaired is termed delayed gastric emptying (DGE) and is also associated with reflux. DGE, which is associated with antral dysmotility [69], pylorospasm, short segment pylorospasm [78], and pyloric stenosis has not been reported in association with rumination.

In two cases of adult rumination [79] no delay was noted in gastric emptying time as measured by radionuclide gastrography. The time course of gastric emptying did, however, affect the frequency and intensity of the ruminations. As gastric emptying progressed, rumination frequency diminished. Levine et al [5] also noted no delay in gastric emptying.

Increased pleuroperitoneal pressure gradient with incompetence or other abnormality of the lower esophageal sphincter may play a role in rumination with some adults. Rumination may lead to chronic esophageal irritation, the possibility of inducing metaplasia (Barrets esophagus) or frank neoplasia. A case of long-standing rumination reported with gastric carcinoma suggested that chronic irritation may have contributed to this malignancy [20]. Rumination associated with esophagitis [2] causes chronic bleeding, resulting in microcytic anemia.

Gastrointestinal Neurohormonal Substrate (Neuropeptides)

The role of neuropeptides (including opioids) in rumination remains to be precisely defined. Effects of upper gastrointestinal tract functions pertinent to postingestive rumination will be reviewed [80] (Table 3).

Gastrin and motilin elevate lower esophageal sphincter pressure (LES). Glucagon, secretin, cholecystokinin (CCK) and vasoactive intestinal peptide (VIP) all lower LESP. VIP is considered the primary inhibitory gut neurotransmitter. VIP-containing nerve fibers, originating in the myenteric plexus of the lower esophageal sphincter, diminish LESP. VIP also promotes gastric emptying. In the stomach VIP inhibits gastrin release. Increased acetylcholine release from vagal stimulation is accompanied by increased VIP. Esophageal distention in animals increases VIP. Circulation clearance of VIP occurs within one minute of its release,
Table 29.3 Some agents influencing human lower esophageal sphincter pressure (LESP)

<table>
<thead>
<tr>
<th>Agent</th>
<th>Raise</th>
<th>Lower</th>
<th>No Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neuropeptide Hormones</td>
<td>Gastrin</td>
<td>Estrogen</td>
<td>Prolactin</td>
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<tr>
<td></td>
<td>Motilin</td>
<td>Progesterone</td>
<td>Somatostatin</td>
</tr>
<tr>
<td></td>
<td>Prostaglandin (PGE)</td>
<td>Glucagon</td>
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<tr>
<td></td>
<td></td>
<td>Secretin</td>
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<tr>
<td></td>
<td></td>
<td>Cholecystokinin (CCK)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Prostaglandin (PGE)</td>
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<tr>
<td></td>
<td></td>
<td>Vasoactive intestinal peptide (VIP)</td>
<td></td>
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<tr>
<td>Pharmacologic Agents</td>
<td>Sodium pentobarbital</td>
<td>Atropine</td>
<td>Diazepam</td>
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<tr>
<td></td>
<td>Metoclopramide</td>
<td>Theophylline</td>
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<tr>
<td></td>
<td>Bethanechol</td>
<td>Meperidine</td>
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<td></td>
<td>Histamine</td>
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<td></td>
<td>Edrophonium</td>
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<tr>
<td></td>
<td>Indomethacin</td>
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<td></td>
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<tr>
<td></td>
<td>Antacids</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other (Nutrition, substance use)</td>
<td>Protein in diet</td>
<td>Smoking</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Coffee</td>
<td>Fat in diet</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Alcohol</td>
<td></td>
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</tbody>
</table>

Adapted from Sleisenger, MH and Fordtran, JS (80)

- Gastrin, motilin, and prostaglandin (PGE) can raise LESP.
- Estrogen, progesterone, glucagon, secretin, cholecystokinin (CCK), prostaglandin (PGE), and vasoactive intestinal peptide (VIP) can lower LESP.
- Antacids can cause no change in LESP.

Supporting its role as a neurotransmitter, VIP may also have a paracrine function effecting relaxation of circular muscle cells immediately adjacent to the neurofibers of origin. Although VIP is structurally identical in both the CNS and gut, CNS VIP has not yet been found to affect gut function [80].

Opioid-containing neurons innervate circular smooth muscle of the lower esophageal sphincter (LES). Dynorphin exhibits preferential agonist effects at kappa receptors, and metenkephalin is agonistic at delta receptors. Mu and kappa receptor stimulation produce LES relaxation, while delta and sigma receptor stimulation produce LES contraction [81]. Opioids diminish acetylcholine release, produce transient smooth circular muscle contraction, and block inhibitory transmission to circular muscle.

Blinder et al [79] have shown that an opioid agonist (paregoric) totally inhibited postdigestive rumination in both a 33-year-old woman with a life-long history of rumination and a 23-year-old woman with rumination and bulimia. Naloxone administered intravenously inhibited this opioid agonist effect.

Premeal administration of both intravenous metoclopramide (50 mg) and oral Haldol (3 mg) also abolished rumination. This effect was blocked by intravenous naloxone. Since dopamine receptor blocking agents (haloperidol and metoclopramide) increase endogenous opioid neurotransmission, their inhibition by naloxone suggests a central or peripheral opioid mechanism in rumination characterized by opioid receptor insensitivity or reduction in endorphinergic neurotransmission. Recent studies in sheep have demonstrated control of ruminant stomach motility by opioid inhibitory and stimulating neurotransmission in the CNS involving mu, delta (inhibition), and kappa (stimulation) receptors [82].

Herman and Panskepp [83] note that the brain circuit for separation distress may be the evolutionary elaboration of an endorphin-based pain network. The extension of opiate receptors into the limbic system suggests an additional affective role for the endorphins. Their data, which demonstrate the effects of morphine (decreases distress) and naloxone (increases distress) on separation distress and approach attachment, suggest that one function of this system may be to modulate emotions arising from social variables. Attachment may represent an endogenous cellular addiction process in which an infant becomes physiologically dependent on its mother for endorphin stimulation.

Chatoor and Dickson [35], acknowledging the finding of Blinder et al [79] and noting the hypothesis of Herman and Panskepp [83] suggesting that attachment behavior is mediated by endogenous opioids, hypothesize that deficiency of attachment and the occurrence of separation may diminish endogenous opioid activity, thereby provoking rumination behavior in infancy. Subsequently, the ruminating activity may act as a compensatory mechanism increasing endogenous opioid levels creating a type of self-stimulating addiction. Adjunctive autoerotic behaviors in infancy that persist after loss and detachment may entail a similar mechanism [84,85].

Rumination and vomiting have been reported during the postnatal withdrawal phase in infants born of nar-
cotically addicted mothers [38,86]. Rumination has been noted in an infant born to a heroin-addicted mother. The child was small for a premature gestational age. Understimulation due to maternal deprivation and a hearing loss may have contributed to the ruminatory disorder along with the narcotic withdrawal [38,79]. Two infants who were in the intensive care unit with multiple medical and surgical problems did not terminate rumination in response to paregoric [4], which contrasts to the effectiveness of this drug in adults [79].

TREATMENT

Treatment in Infants and Children: Hospital Milieu and Family Collaboration

Since rumination in infants and young children may be life-threatening, a multidisciplinary approach is mandatory. The primary physician must decide whether hospitalization is indicated. The decision may be based on the chronicity of the rumination or the presence of significant medical complications (eg, failure to thrive, dehydration, electrolyte abnormalities) or gastrointestinal disturbances (eg, hiatal hernia). Hospitalization may also be indicated when the primary caretaker's ability is severely compromised. Since rumination often occurs in multiproblem families, careful evaluation of the child's psychosocial situation is mandatory. Although many authors have stressed deficient mother-infant interactions [8-10], recent reports [35,55] have noted a positive relationship between the mother and the infant. Rumination without severe weight loss or other physiologic alterations, in the context of a supportive family, may respond to outpatient treatment.

Hospitalization of the child is often a terrifying experience for the mother [9]. She may feel guilty, inadequate, and responsible for her infant's problem. She should be given permission to ventilate her fears and frustration that her child is not getting well immediately and to know that there are medical and psychological reasons for the rumination. An anatomical and physiologic description of reflux may be helpful.

Documentation of the staff's observations of the temperaments of mother and child and the degree of reciprocity should be obtained. Chess [87] suggests that a two- to three-hour home visit to determine the dyadic relationship of mother and child can be very helpful. Preferably, the observation period would be during a sleep and waking cycle. This amount of time is needed to put the patient at ease (as opposed to a brief structured laboratory observation, which could be stressful to both mother and infant and where the mother might present a facade of caring). The observer should refrain from taking notes and should be friendly and non-judgmental. If the mother is the primary caretaker, she can be seen alone. However, if there are other caretakers, such as a father or grandmother, they can be present. Levy [88] gives an excellent review of the mother-infant relations during feeding. He discusses specific questions such as, for example, is the baby allowed to be an active participant in his feeding, how the foods are presented, who decides when the feeding will end, and how pleasurable feeding is for both mother and infant. Some obsessive mothers may become distraught when the infant vomits near them [3].

A structured interview with the mother, father, or other primary caretaker is crucial. The mother's own developmental and personal psychiatric history may often contain determinants of current conflictual attitudes and behavior toward the infant.

Dickson emphasizes minimizing the mother's guilt. Rumination can be attributed to babies who have a problem with homeostasis and withdrawing into a maladaptive habit. The mother should be told she is both an expert with her child and an important colleague in the treatment process. Her fantasies about the child's rumination, associated failure to thrive, and what techniques have either been helpful in reducing the rumination or what events seem to have precipitated the rumination should be explored. The mother's fragile self-esteem and her feelings of incompetence should be acknowledged and countered by designating her an important colleague in the child's treatment.

In the hospital, the baby should be placed near the nursing station to increase the child's visual and auditory stimulation. There should be a specific nurse on each shift who will give primary care to the child. Frequently, a competent social worker who is involved will be able to pick an empathetic nurse who will be emotionally available during an eight-hour shift to spend much of the time with the child.

A nurse acts as a substitute (surrogate) mother with whom the baby can develop an attachment [8]. Where there is a failure of attachment, substitution of primary care may be critical. As this attachment develops, the child restores a stable object relationship. Mother later will become more involved with feeding. The child will transfer its attachment and thus develop a restorative object relationship with mother. The next therapeutic task will be interruption of the rumination. The child's unique ruminative pattern should be recorded, eg, occurring when the baby is alone or occurring when the mother pushes the baby away [89]. The nurse who is aware of this pattern should be present to interrupt the possible anxiety-producing situation and frustration that may precede rumination. The fourth therapeutic task focuses on the relinquishing of maladaptive ruminative and self-stimulatory patterns.
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Table 29.4 Suggested activities for a sensory stimulation program

**Visual**
- Place the infant in face-to-face contact with caretaker inside and outside of crib, particularly during feeding, diapering, etc.
- Place brightly colored mobiles about 7 to 12 inches above the infant’s face.
- Place face patterns on the sides of the Isolette or crib.

**Tactile**
- Skin-to-skin contact while being held.
- Gentle stroking to back, legs, and arms.
- Gentle patting on the infant’s back.

**Auditory**
- Frequent exposure to the human voice, particularly during routine care, and concomitant with eye contact with infant.
- Soft music from music box placed in Isolette or crib during alert periods.

**Kinesesthetic/vestibular**
- Gentle rocking while cradled in caretaker’s arms.
- Frequent changes in infant’s body position, ie, sitting in infant seat, lying prone, lying on side.
- Carrying infant around room in various positions, ie, on shoulder, cradled in arms, etc.

Sheagren et al [4]

Lourie [8] has reported that a number of ruminators have hypersensitivity to touch and sound [90]. Placing the child on a pillow with minimal touching, but with visual or auditory stimulation would be helpful. In one child who was not interested in people, a relationship was started by a nurse who interested him with brightly colored clothing and jewelry. These auditory and visual stimuli, combined with the crib rocking, initiated the attachment process. In another infant who withdrew from any social contact, placement in a crib with another baby was helpful in starting an object attachment. Later, holding both children on a nurse’s knee was useful in reinforcing the attachment process.

Hospital Milieu

Sheagren et al [4] discussed a multisensory stimulation approach (Table 4). He noted that in three cases using a limited number of nurses, and placing the child in an open crib, the children started to gain weight, developed a social smile, and had improved interaction with their caretakers.

The use of video tape may aid in analysis of the dyadic mother-child interactions by emphasizing tension states and separation-withdrawal in mother and infant and their link to ruminatory behavior.

The mother needs to recognize her child’s strivings for autonomy and the baby’s need to be a stimulated and active participant in the feeding process (many ruminant babies are quite passive). Feeding should terminate when the child is finished, and rigid schedules should be abandoned. The mother may need to stimulate the infant by increasing her eye contact, vocalizing, and smiling during feeding. She may have to work through her uncomfortable feelings that have been present during the feeding process [88].

Dickson and Chatoor [89] feel that the primary purpose of therapy is to break the ruminative habit. Problems in the family may exist after discharge, although ruminination frequently will cease. The goal for the baby is to develop external object satisfaction by using a combination of negative and positive social reinforcers. As the child ruminates, the nurse will say “NO,” gently touch the mouth, and place the baby down. In two minutes she will check the baby. If ruminations has ceased she will again play with the patient. Ideally, the nurse will be able to play with and stimulate the baby during her entire shift, providing positive social reinforcement. At times the child may be pulled around and remain close to the nurse. This type of positive reinforcement may be more effective than the aversive behavioral techniques of squirting lemon juice and pepper sauce during rumination.

Maternal depression in association with infant rumination has been described frequently [8-10,31]. Therefore, a long-term goal may include individual treatment for a depressed mother. If the child continues to ruminate, placement outside the home may be necessary. As a part of discharge planning, home visits and increased support from mother’s friends and family are advised. The mother should be seen after discharge both individually as indicated, with the child at least once a week for psychological interactive management, and at least once a week by the pediatrician.

Since rumination in young children can result in death, the resistant mother may have to be confronted about the poor prognosis if her emotional state and environmental conditions are not modified.

Treatment in Adolescence

In a 13-year-old patient with a three-year history of rumination, a multidimensional approach to treatment was necessary, including individual psychotherapy to work through a mother’s death and the possible dissolution of a father’s current marriage, and family treatment to resolve tensions of marital discord. Behavior monitoring documented three specific ruminatory patterns: food rising but not going to the mouth, food regurgitated and reswallowed, and food regurgitated and extruded. A relaxation technique using a personalized tape developed by the therapist was employed with the patient alternately tightening and relaxing parts of the body. Deep breathing and imagery were also used [42].
Biological Treatment: Medical-Surgical and Pharmacotherapy

Herbst [2] believes that many cases of rumination are primarily related to excessive gastroesophageal reflux. In this view, the management of rumination is essentially the same as the medical management of reflux [12]. The initial treatment includes elevation of the infant to the position of a 30 to 45 degree angle; avoidance of juices; and small, frequent, thickened feedings. A six-week medical course is usually instituted. After each meal, aluminum hydroxide, an antacid, can be alternated with magnesium hydroxide. If symptoms persist [12] in an infant over one year of age, cimetidine, which blocks H-2 receptors, diminishes gastric acid production, is given. Bethanochol also may be administered. In older children, administering a dopamine antagonist such as metoclopramide (acting centrally and peripherally), or domperidone (acting peripherally) increases LES pressure, gastric tone and peristalsis, and improves gastric emptying [91]. Side-effects such as extrapyramidal reactions may occur with metoclopramide but rarely with domperidone [92]. If a six-week trial of medical treatment is ineffective, surgery may be indicated [12].

Winter [65] evaluated five cases of rumination in infants and young children who displayed pathologic gastroesophageal acid clearance. Treatment of reflux and esophagitis by positional therapy, antacids, and cimetidine abolished rumination and suggested a medical etiology. Nasogastric tube feeding may be used in severe reflux to prevent dehydration and electrolyte abnormalities [35].

Criteria for surgery are persistent vomiting after vigorous medical management, failure to thrive with nutritional depletion, gastroesophageal bleeding from esophagitis, aspiration pneumonia, and esophageal stricture. The surgical procedure of choice is the Nissen fundoplication [93].

Treatment of Adults

Treatment of adult rumination presents a different context from that of the child. Family physicians may not recognize adult rumination as a discreet phenomenon. Often patients are confused by explanations of the behavior that allude to stress or emotional disorder. They become guilty about their problem and need reassurance. Pope’s technique is to discuss gastroesophageal reflux using a diagram of the stomach and esophagus. Pictorial illustrations may be helpful. By explaining the mechanism of reflux to the patient, this problem is appropriately recognized, and the patient feels reassured and relieved [5,46].

Medical treatments with antispasmodics have been ineffective [1]. Because the esophageal contractile abnormalities producing reflux may be secondary to an agitated depression or anxiety disorder [73,74], some patients with rumination may have features of specific psychiatric disorder.

An experimental treatment using an opioid agonist (paregoric) and medications that enhance endogenous opioid transmission (metoclopramide, haloperidol) has been effective in diminishing ruminative behavior in two adult patients [79].

Preprandial hypnosis has been successful in an adult with chronic rumination [94].

Behavioral Therapy

Behavioral treatment to suppress rumination in the retarded encompasses physical aversive techniques including mild digital electroshock [14], oral application of pepper sauce [59,95] and lemon juice [29], and newer nonaversive modalities including food satiation [24], overcorrection [30,96], extinction [97], positive social reinforcement [31,35,98], withdrawal of music [99], or differential reinforcement of other behavior [100]. The general paradigm for behavioral treatment studies includes an analysis of the frequency, setting, duration and antecedent events of the behavior to the changed, followed by a procedural schedule of positive or negative reinforcement.

Behavioral treatment of rumination with physically aversive stimuli was first reported in 1968 [28]. Mild electroshock, an aversive stimulus to the finger, was successfully employed in a severely dehydrated child who did not respond to psychological management [14]. Winton and Singh concluded that “electroshock should be used only in life-threatening cases that have proved refractory to other forms of therapy and that its use should always be paired with positive reinforcement of appropriate behavior” [13].

Tart and bitter substances such as lemon juices [29] and pepper sauce [59] have been squirted into the mouth, diminishing rumination. Singh [95] found that pepper sauce was more effective than lemon juice (which can dissolve tooth enamel). Both substances, however, were difficult to apply effectively.

Subsequent approaches have deemphasized the physically aversive stimuli, which often led to staff resistance. For example, satiation techniques have been reported in which a subject is allowed to eat as much food as is desired, ultimately leading to suppression of rumination. A beneficial side-effect is an increase of weight for patients who are malnourished [24]. Duker and Seyes [101] used overcorrection, a procedure in which the patient is required to “clean up” after every episode of rumination. Fox et al [30] followed regurgi-
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Rumination may be on a continuum wherein a patient might have maximal gastrointestinal pathophysiology such as severe reflux with hiatus hernia and minimal psychological concomitants, or the converse wherein a patient could have minimal gastrointestinal pathophysiology or reflux but severe psychopathology or psychosocial stress. Proponents of the biologic theories believe that psychological factors definitely influence rumination.

Multiple stresses in children can produce similar symptomatic behaviors [104]. For the child, irritability and discomfort may result in feeling overwhelmed, anxious, or depressed or may be manifest as severe reflux with esophagitis. Inferred reflux esophagitis, treated either medically or surgically, may result in a feeling of well being and a termination of rumination [72].

Psychodynamically oriented therapy using a substitute caregiver may reduce rumination for two reasons. First, the child receives increased stimulation, which aids in trust and attachment. Second, this additional care is effective because the child is held upright during the period of stimulation, diminishing both reflux and esophagitis. The esophagitis, which subsides, augments lower esophageal sphincter pressure, further diminishing reflux. Diminished esophagitis results in reduced psychological tension, promoting feelings of well being in both mother and infant.

Maternal anxiety may promote secondary physiologic changes in a child. For example, a mother feeling overwhelmed by a stressful situation or feeling anxious secondary to her child’s persistent vomiting and weight loss may exhibit increased muscle tension. This is transmitted to the child, who becomes tense and develops a more rapid heart rate [77]. The increased autonomic response may alter neuroendocrine controls and VIP, producing lower esophageal sphincter relaxation and increased reflux. Thus, the tendency of the child to ruminate may be increased by an anxious mother. Psychiatric disorder has been associated with both reflux and esophageal contractility abnormalities [73]. Transmitted maternal stress could result in infant gastroesophageal contractile dysfunction, promoting reflux and rumination.

Two proposed biopsychosocial sequences of rumination in an infant are presented in Table 5 (predominance of interactive psychopathology or gastroesophageal abnormality). A close interrelation occurs between mother and infant with various pathophysiological and emotional stresses. Diagnosis and treatment based on evaluation of both the psychological state of the mother

BIOPSYCHOSOCIAL SYNTHESIS IN RUMINATION

The etiology of rumination is unclear. Physiologic, psychodynamic, and behavioral theories have been discussed. Rumination is a psychobiological disorder in which psychological and physiological abnormalities combine in various degrees to produce the ruminary behavior.

In summary, each author advocates the efficacy of his own technique. However, methodologic inadequacies include small sample size, lack of comparison of different behavioral techniques in a given population, absence of control groups, and lack of long-term follow-up comparing relative effectiveness.
Table 29.5 Biopsychosocial sequence in rumination with maternal infant interactive disturbance

I. Child-natural tendency for vomiting and reflux.
II. Psychosocial stress.
   1. Understimulation — mother depressed.
   2. Maternal overstimulation — mother anxious or involves infant in excessive inappropriate involvement.
   3. Object loss of parent.
   4. Excessive family discord.
III. Neuroendocrine dysregulation related to II.
   1. Abnormal esophageal contractions.
   2. Endogenous opioid deficit (neurotransmission/receptor insensitivity central or peripheral).
IV. Increased reflux and rumination.
V. Augmented maternal anxiety due to:
   1. Fear baby will die.
   2. Helplessness over an inability to feed baby.
   3. Further detachment due to mother's disgust with vomiting.
VI. Increased rumination.
VII. Possible death due to dehydration, starvation, aspiration.

Biopsychosocial sequence in rumination with primary functional or anatomic pathophysiology

I. Natural tendency toward vomiting and reflux.
II. Gastroesophageal reflux.
   A. Etiology #1 — Gastroesophageal pathology, eg:
      1. Hiatal hernia.
      2. Esophageal motor dismotility — excess uncoordinated tertiary peristaltic waves, esophageal contractural abnormalities, atonic distal esophagus.
      4. Excessive VIP (speculative).
      5. Subsensitive opioid-receptors or insufficient opioid gut transmission (speculative).
   B. Etiology #2 — Gastroduodenal pathology. Delayed gastric emptying due to:
      1. Antral dismotility.
      2. Pylorospasm.
      3. Short segment pyloric stenosis.
III. Rumination. Healthy mother becomes anxious due to loss of weight and vomiting of infant.
IV. Maternal anxiety transmitted to child.
V. Increased rumination.

and infant and the infant's gastrointestinal function is indicated [111].

SUMMARY

Rumination is an uncommon disorder occurring from infancy through adult life. It consists of regurgitat-

and then reswalling partially digested food. Rumination may result in considerable morbidity in infants and young children. Adult ruminators may have a benign course with embarrassing involuntary reflux or may have an associated eating disorder (bulimia or anorexia) or depression.

Biologic theories of etiology associate rumination with gastroesophageal reflux, hiatus hernia, and delayed gastric emptying. Psychological theories discuss infants who have severe failure to thrive and often appear depressed. Severe dysynchrony between mother and infant and maternal psychopathology consisting of anxiety, depression, and inability to adequately nurture the child may be present. Behavioral theory discusses the self-reinforcing aspect of the ruminatory behavior. Theories of neuropeptide and opioid regulation posit central and peripheral deficits of endorphinergic neurotransmission and receptor sensitivity. Rumination associated with interactive psychopathology may be an affective disorder variant.

Treatment approaches reveal pharmacologic or surgical treatment of reflux, psychological treatment of the infant-mother dysynchrony (with the use of substitute caretakers), and behavioral treatment using aversive stimuli (lemon juice, pepper sauce) or positive social reinforcement.

Since rumination may have a biologically or psychologically predominant context, a biopsychosocial theory and sequence have been elaborated. Therefore, a multidisciplinary approach to diagnosis and treatment that uses available appropriate treatment modalities is imperative to treat this disorder comprehensively and effectively.

REFERENCES

25. Fabricius A. Tractatus de gula ventriculoet intestinis. Padua 1618.
52. Woolston JL. Eating disorders in infancy and early childhood. In this volume.


89. Dickson L, Chatoor I. Personal communication, 1984.


98. Barmann BC. Use of contingent vibration in the treatment of self-stimulatory hand-mouthing and rumina-


