Functional vomiting disorders in infancy: Innocent vomiting, nervous vomiting, and infant rumination syndrome

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Pediatric gastroenterologists have tended to view gastroesophageal reflux (GER) as a disease in and of itself—a disease that can be diagnosed "objectively" with use of numerical data from esophageal pH monitoring and cured with pharmacologic or surgical treatment. What is often forgotten is that the data derived from esophageal pH monitoring and other techniques may identify the presence of abnormal GER but tell nothing about its pathogenesis. The usual approach to infants who feed poorly, vomit, or fail to gain weight is to identify the presence of abnormal GER, rule out underlying organic causes of vomiting, and then diagnosis primary GER disease. The baby is then treated with pharmacologic, dietary, or positional therapy and, ultimately, if these therapies fail to eradicate the symptoms attributed to GER, surgical fundoplication, which stops vomiting regardless of its causes. The pediatric literature on infant vomiting and GER is almost devoid of research into the nature and possible relationships among infant stress, vomiting, feeding difficulties, and failure to grow. Clinically, the quality of the maternal-infant relationship is frequently approached superficially, with psychosocial aspects treated as less important in infants considered to have primary organic disease amenable to medical or surgical treatment. Psychosocial factors in the pathogenesis of the infant's symptoms are often not pursued beyond assessment for possible abuse or neglect. It has been known for centuries that stress or excitement affects gastrointestinal function and symptoms. Although the field of infant psychiatry has produced a substantial literature on the nature of stresses that affect both infants and mothers, the pediatric literature on vomiting and failure to thrive seldom acknowledges the existence or importance of these contributions. In clinical practice, failure to explore psychosocial aspects that may contribute to vomiting, feeding difficulties, or failure to thrive may result in missed opportunities for less invasive, more effective therapy at best, and countertherapeutic treatment at worst. This article describes three functional vomiting disorders of infancy, their distinguishing characteristics, hypotheses regarding their pathogenesis, and principles of comprehensive management. (J PEDIATR 1994;125:S84-94)

The management of infants who vomit and fail to thrive consists of identifying the presence of abnormal gastroesophageal reflux by esophageal pH monitoring and other methods, ruling out underlying organic etiologies of vomiting, viewing the GER as a primary disorder causing failure to thrive, applying pharmacologic measures to lessen reflux, and, if these approaches fail to effect a cure,

GER	Gastroesophageal reflux

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considering fundoplication, a surgical procedure that may stop vomiting regardless of its causes.

Boyle[1] categorized GER on the basis of published numerical data derived from esophageal pH monitoring of healthy and unhealthy infants and children. His four categories of GER are as follows: physiologic (i.e., within the normal limits of GER found in infants and children who do not vomit); functional (i.e., that found in healthy infants and toddlers whose vomiting neither results from nor causes organic disease, and usually remits between 6 and 18 months of age); pathogenic (i.e., GER of unknown origin that exceeds normal limits and causes organic disease that affects the esophagus or respiratory tract or causes growth impairment); and secondary (i.e., abnormal GER resulting from an identifiable primary disease intrinsic to or outside of the gastrointestinal tract, such as hiatal hernia, obstructive uropathy, or diseases of the central nervous system).

Although esophageal pH monitoring quantitates GER, it tells nothing of its cause or mechanisms.[2]

The purpose of this report is to focus attention on the infant-caregiver relationship in the clinical assessment of babies who vomit. Rene Dubois said, "Sometimes, the more measurable drives out the more important."[3] GER is measurable. Conversely, aberrations in the nurturing relationship are difficult to quantify, are subtle, and require interpersonal rather than technologic diagnostic procedures.[4, 5] Patients are described in whom malfunction of the maternal-infant relationship was of overriding importance in the pathogenesis of vomiting and failure to thrive.

The following descriptions of three functional syndromes of infant vomiting are based on clinical observation rather than esophageal pH monitoring criteria. They are "innocent vomiting," "nervous vomiting," and "infant rumination syndrome." The first, innocent vomiting, would seem to be the equivalent of Boyle's functional GER; the reason for calling this type of vomiting "innocent" will be explained during the discussion of its management. The importance of innocent vomiting is its ubiquity and its sometimes confusing similarity to abnormal types of vomiting. The latter two vomiting syndromes are caused by aberrations in the mother-infant relationship and are often accompanied by failure to thrive.

INNOCENT VOMITING

Innocent vomiting is reported to occur in 20% to 50% of infants (T. Brazelton, personal communication, 1994).[1] It may begin as early as the newborn period and resolves within 6 to 18 months in most cases. The baby vomits without nausea, retching, or pain, with a frequency that ranges from several times a week to several times an hour, during or between feedings, while the infant is awake, and rarely during sound sleep. The vomitus is typically nonbilious gastric content that is ejected with little energy or in a projectile manner. By definition, innocent vomiting is not accompanied by features of GER disease (e.g., hematemesis, apnea, aspiration, bronchospasm, or chronic cough) and it does not impair weight gain. Presumably, its mechanism differs from the nausea and vomiting caused by vestibular stimulation, activation of the chemoreceptor trigger zone, or stimulation of visceral afferent nerves involving the vomiting centers in the medulla.[6]

Fig. 1. Barium study of antropyloric region that displays features of pyloric stenosis (patient 1).

Weakness of the lower esophageal sphincter mechanism and delayed gastric emptying predispose to GER.[7] Jolley et al.[8] studied esophageal reflux in infants who vomited and in healthy infants for 4 hours after feeding and found three patterns. In the pattern designated as *normal*, reflux was frequent during the first postcibal hour, then steadily subsided and was gone by the end of the second hour. Patterns designated as abnormal were of two types: *continuous* (undiminishing reflux throughout the 4 postcibal hours) and *discontinuous* (reflux decreased after feeding, but took almost 3 hours to subside completely). The continuous pattern was often associated with hiatal hernia. Infants with the

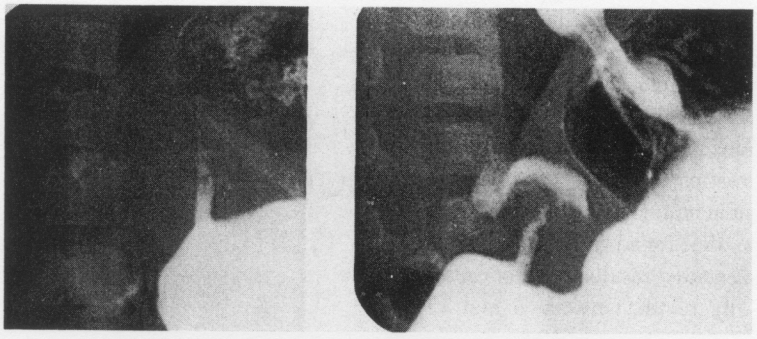

Fig. 2. Barium study shows elongation of pyloric channel suggestive of pyloric stenosis (patient 2).

discontinuous pattern had three characteristics: (1) their lower esophageal sphincter pressures tended to be above average; (2) their upper gastrointestinal contrast studies frequently demonstrated antropylorospasm; and (3) they tended to have diarrhea characteristic of the irritable bowel syndrome of childhood.[9] These data suggest the hypothesis that innocent vomiting is an upper gastrointestinal manifestation of the irritable bowel diathesis. Normal growth and absence of apparent distress in such infants could be attributed to the absence of nausea-related anorexia, which allows for retention of sufficient nourishment to sustain normal growth, provided they are fed to satiety.

Optimal management of innocent vomiting consists, in my opinion, of identifying the diagnosis and effectively reassuring the parents of its prevalence, safety, and prognosis for spontaneous resolution. Innocent vomiting is a better diagnostic term than "functional GER—a developmental disorder"[1] because the latter term implies the existence of an abnormal state rather than a variant of normal function. It "medicalizes" the physicians' approach to management, predisposing to unnecessary, invasive diagnostic procedures and the imposition of pharmacologic, positional, and dietary regimens that burden the parents and child while contributing little or nothing to resolution of the vomiting.[1] Innocent vomiting, like uncomplicated umbilical hernias, should be treated with effective reassurance that allows time to effect a cure. Calling it a disorder and prescribing antireflux treatments reinforce the parents' concern that their infant is abnormal and vulnerable.[10]

NERVOUS VOMITING

In the nervous vomiting disorder, vomiting accompanies other behavioral symptoms of infant stress and impairment of maternal-infant reciprocity.[11] It often results in failure to thrive. Nervous vomiting was first described in 1925 by H. C. Cameron,[12] Physician in Charge of the Department of Diseases of Children, Guy's Hospital:

> " . . . the infant who suffers from hyperemesis is almost always tense, nervous, emotional and more than usually unstable. . . . Sleep is almost always scanty and superficial. In response to the slightest stimulus, instantly the child is wide awake, the anxious strained expression flows back into the face, and the inevitable crying begins again . . . Opisthotonos is common, while the hands are nearly always held tightly clenched. Such infants may appear to suffer from a veritable gastric hyperesthesia. The presence of food in the stomach may evoke every appearance of extreme pain and discomfort may be followed by vomiting, whether the infant was fed upon the breast or the bottle."

Patient 1

A first-born female infant began vomiting at 3 days of age. She was hospitalized at 10 weeks because of almost continuous vomiting and increasing irritability, which did not improve with a milk-free diet. No weight lag or electrolyte deficits were present. Radiologic study showed normal peristalsis in the proximal stomach with insufficient opening of the pylorus and "hypertrophic muscle indenting the antral region and the base of the duodenal bulb" (Fig. 1). The radiologist's diagnosis was hypertrophic pyloric stenosis. Diagnostic interviews[13] conducted with each parent during the 12-day hospitalization revealed marital discord and emotional stress in both parents, which seemed to have intensified as their baby's vomiting increased. Pyloromyotomy was avoided because the baby's vomiting and irritability improved as the parents' fears, contentiousness, and exhaustion subsided. During an outpatient visit 17 days after hospitalization, the baby's vomiting had decreased further, her mood had improved, and she had continued her normal rate of weight gain. "She's like a different baby," the mother said. Nine months later, the mother reported, " . . . spitting up is almost completely stopped; she's a very happy baby."

Patient 2

A 7-month-old male infant was hospitalized for nonbilious vomiting and weight loss of 3 months' duration. Two upper gastrointestinal barium studies were performed and each showed pylorospasm with delayed gastric emptying (Fig. 2). A small amount of GER occurred when the stomach was filled. The radiologist diagnosed pylorospasm. Results of esophagogastroduodenoscopy were negative. Results of the biopsy of the esophageal mucosa showed minimal inflammatory changes. The family had moved three times during the preceding 2 years, most recently a few weeks before the patient's birth. The father, a young executive, had been assigned the task of making a large but marginal business profitable and had been working extraordinarily hard. The mother, a successful career person, now spent most of her time at home mothering her 7-year-old son and new baby. She lived far from friends and family and hadn't had time to establish new friendships. She was a perfectionist and felt at fault when anything went wrong at home. The relationships in this nuclear family had become critically stressed. The crisis in family life had not been apparent to them, partly because of their preoccupation with their increasingly sick infant and their frustrated attempts to find the elusive food intolerance that they believed caused the vomiting. The infant was hospitalized for 12 days, fed ad libitum, shielded from commotion, and comforted promptly whenever he fussed. Stressful diagnostic procedures were spaced widely. Vomiting decreased. The parents became aware of their state of emotional exhaustion during reflective discussions with their physician. Their stated fear of losing their child subsided as a weight gain trend began to emerge on the fourth hospital day. The patient was released from the hospital with no medications or special diet. Ten weeks later, the mother wrote, "He has gone for six days straight without even spitting up, much less vomiting!" Fig. 3 depicts the patient's weight before and after management of his vomiting disorder.

Patient 3

A 6-month-old first-born girl was hospitalized for vomiting, persistent crying since 6 weeks of age, and weight loss. She was weaned at 3 months because her mother felt her milk "drying up." Thereafter, fussiness persisted, the child vomited a few times a day, and she passed loose stools sporadically. Diagnostic studies of the digestive, central nervous, endocrine, immunologic, and urinary systems and a search for congenital and acquired infections were carried out during the 10-day hospital stay. Diagnostic procedures were spaced to minimize stress and allow observation of her symptoms. She rarely vomited in the hospital and passed three to six stools of varying consistency each day. She was fed the same formula as before admission, as much as she

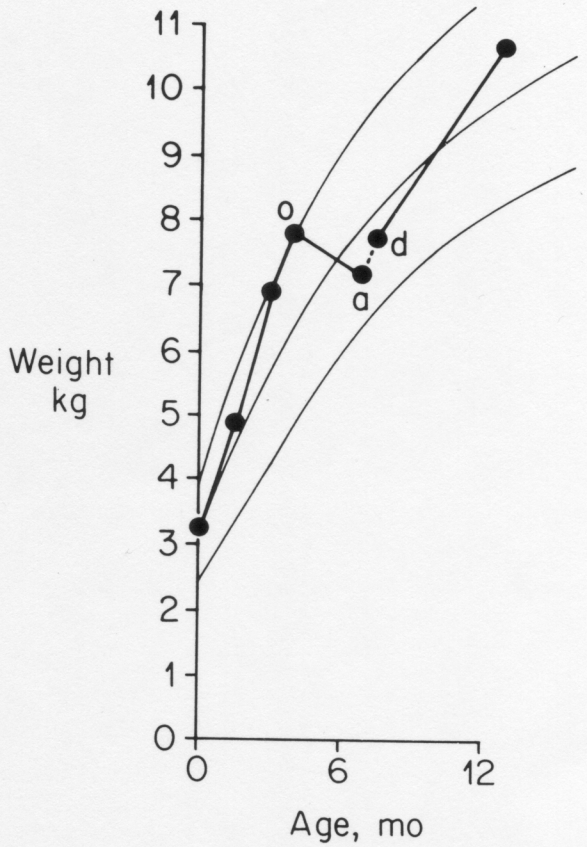

Fig. 3. Weight of patient 2 at onset of vomiting *(o)*, at time of hospital admission *(a)*, and at time of discharge from hospital *(d)*.

wanted, whenever she wanted it, and she gained weight. Diagnostic interviews with each parent revealed severe marital discord, which worsened after the mother discovered her pregnancy with the patient. The father objected to the pregnancy and became increasingly unavailable. The mother handled her child hesitantly during the early days of hospitalization, but became more comfortable in caring for her as the infant improved. The baby was released from the hospital with no medications and the same formula that she had been offered before hospitalization. During the follow-up visit 2 months later, the mother reported that her infant's fussiness had greatly improved, although she still "spat-up" small amounts daily; this symptom resolved by 15 months of age. The mother said, "I feel a lot better now, knowing that nothing is really wrong with her. She's getting to be more fun." She was in the process of divorcing her husband. Fig. 4 depicts the patient's weight course.

Patient 4

A 6-month-old female first-born infant was hospitalized for evaluation of vomiting, episodic loose stooling, and

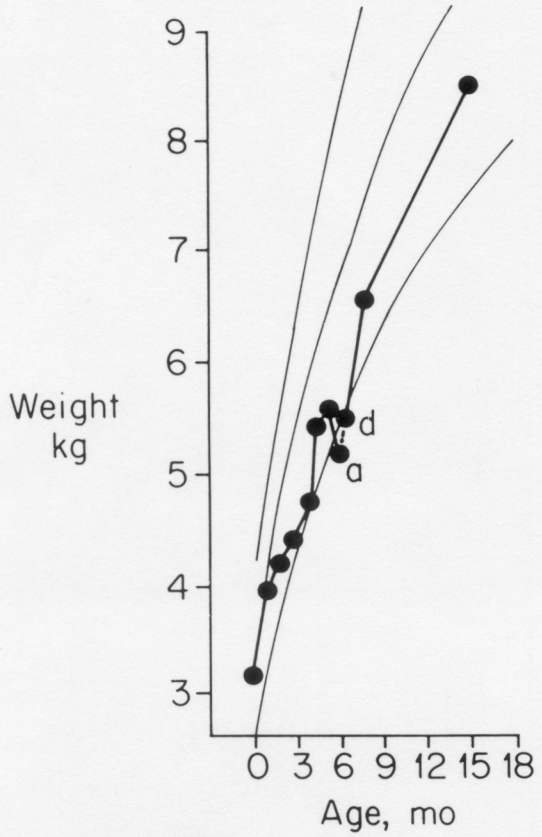

Fig. 4. Weight of patient 3 at time of hospital admission *(a)* and at time of discharge from hospital *(d)*.

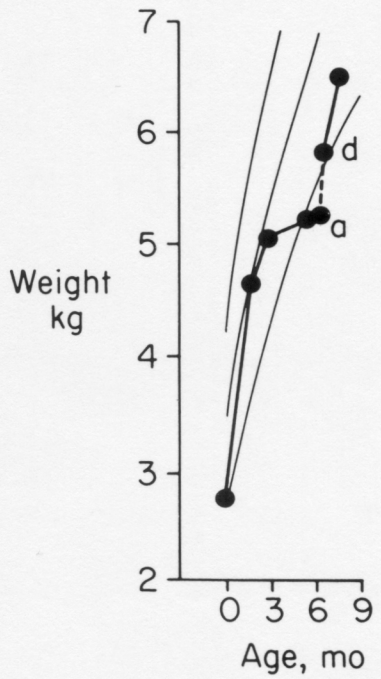

Fig. 5. Weight of patient 4 at time of hospital admission *(a)* and at time of discharge from hospital *(d)*.

weight lag. The patient had been unusually alert and responsive since birth. Six weeks after she was born the father was laid off his job, necessitating the mother's return to full-time employment at a very stressful job. At 2 months, the patient began "spitting up" after feedings. By 4 months, episodes of vomiting had increased to seven to eight times a day. The patient's stool pattern changed from infrequent pellet stools to loose stools passed as often as 12 times a day. Dehydration did not occur. The symptoms failed to improve with formula changes. During the 2 weeks prior to admission to the hospital, her stools changed from loose to firm pellets, but vomiting and weight lag persisted. Physical examination revealed a socially responsive, thin, but otherwise healthy-appearing infant. The patient was fed an unlimited diet containing milk, soy, and gluten. Diarrhea did not recur. Comforting was provided whenever the baby fussed and an effort was made to shield the patient from excitement during feedings and when she was put to bed. Diagnostic procedures were spaced to avoid prolonged stress and to permit observation of the patient's symptoms and daily discussions with the parents. An upper gastrointestinal barium study showed an irritable esophagus, but was otherwise negative. Esophageal manometry showed lower esophageal sphincter pressure to be at the lower limit of normal. Abnormal GER was demonstrated by esophageal pH monitoring. Diagnostic test results were otherwise negative. No medication, thickened feedings, or upright positioning techniques were used. The patient vomited an average of 3.2 times per day. Nevertheless, she began a catch-up pattern of weight gain. Interviews with the parents elucidated the severe stress they had experienced in caring for their new baby and the pressure imposed on the mother by her husband's unemployment and her own work. The parents' outlook improved as they observed their baby's weight gain (Fig. 5). On a follow-up visit 5 weeks later, the mother said, "She's back to her normal self again, playful and happy." Diarrhea had not recurred. On some days the baby regurgitated small amounts during postfeeding eructations " . . . especially if she plays hard and gets excited after meals." The mother had lessened her own job pressures and the father had been rehired. One year later, the mother reported that the patient no longer vomited and was growing normally.

Comment

Patients 1 and 2 had features suggestive of hypertrophic pyloric stenosis. It may be difficult to distinguish pyloric stenosis from pylorospasm.[14-18] The temporal relationship between lessened parent-infant distress and the onset of

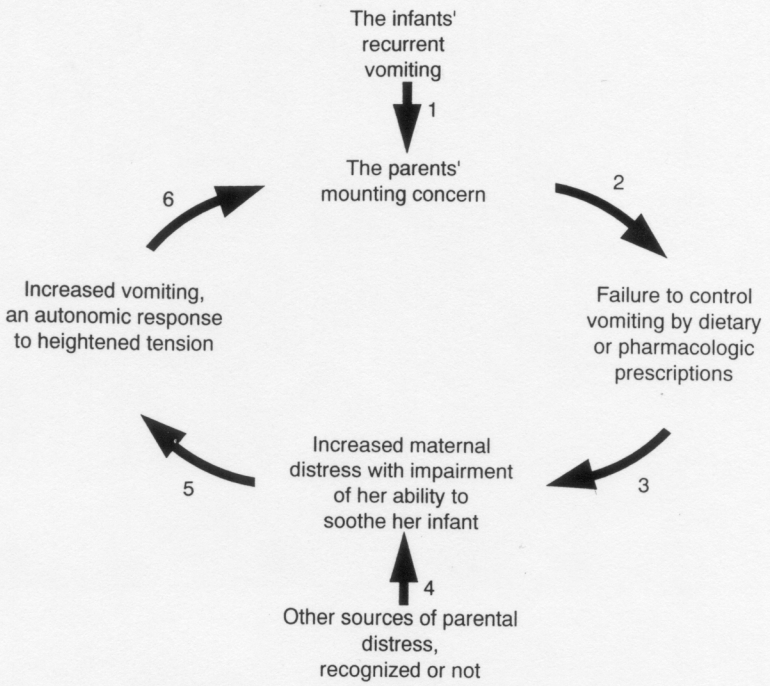

Fig. 6. The vicious cycle of nervous vomiting.

symptom improvement confirmed the presence and importance of the psychophysiologic component of the vomiting.[19]

Patient 4 exemplifies the error of dichotomizing infant vomiting into "psychologic versus organic" categories. It would have been a mistake to attribute this infant's vomiting and weight loss solely to the demonstrated weakness of the mechanical barrier to GER without also assessing the infant-mother relationship.[20]

Pathogenesis of nervous vomiting

It has been repeatedly demonstrated that emotional and other forms of stress affect gastrointestinal motility[21] and that gastric emptying is delayed by acute stress in healthy persons.[22] Maternal anxiety has been shown to cause increased muscle tension, which is transmitted to the infant, as evidenced by increased muscle tone and heart rate.[23] The field of infant psychiatry has developed a large scientific literature on the nature of infants, the reciprocal relationship of infants and caregivers, and the stresses that occur during normal and aberrant interactions between them.[11, 24-33] These physiologic and developmental concepts form the basis of a clinically useful hypothesis: nervous vomiting is a visceral reaction to stress and excitement manifested by GER, pylorospasm, or failure to thrive. It is perpetuated by the establishment of a vicious cycle (Fig. 6).

Diagnosis and management of nervous vomiting

One conceptual approach to the infant who vomits and fails to thrive is to diagnose abnormal GER, view it as a primary, idiopathic disease, and attribute the vomiting, fussiness, and feeding difficulties to GER disease,[34] which then results in failure to thrive. An alternative approach is to view that same infant as having symptoms caused by a breakdown in the nurturing relationship, which, in turn, causes irritability, vomiting, feeding difficulties, and failure to thrive. The former approach makes reflux-induced esophagitis or gastroesophageal hyperalgesia the focus of management.[35, 36] The latter approach makes the nurturing relationship the focus of management. Both approaches are valid to the extent that they are appropriate to individual patients. However, the pediatric literature on GER is almost devoid of references to research in the field of infant psychiatry.[37] The assumption that abnormal GER is a primary, idiopathic disease amenable to pharmacologic or surgical cure creates a bias in favor of technologic diagnosis and management[3, 38-40] and against assessment of psychosocial factors other than possible abuse or neglect. The danger of this bias is exemplified by the infant with functional GER who is categorized as having abnormal GER because he or she fails to thrive. The vomiting may be an epiphenomenon of failure to thrive.[33]

A practical method for determining the importance of aberrant nurturing as a cause of failure to thrive in the infant who vomits is to include a *therapeutic trial of comfort* as part of the diagnostic evaluation.[19] This includes simultaneous efforts at relieving both the infant's and the parents' distresses. The recommendations of Cameron[12] are still

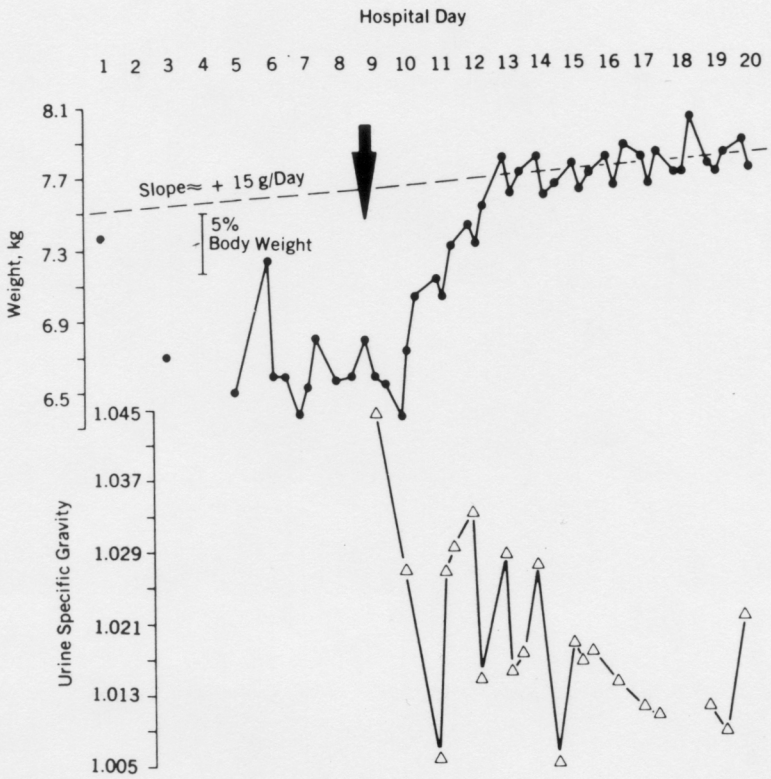

Fig. 7. Response of an 8-month-old boy with infant rumination syndrome to a therapeutic trial of special nursing. *Arrow* indicates the start of "holding therapy" by a mother substitute. (Modified from Fleisher D. Infant rumination syndrome. Am J Dis Child 1979;133:266-9. Copyright 1979, American Medical Association.)

useful as a guide to satisfying the infant's need for comfort.

> " . . . it is always an unwise and unsuccessful maneuver to attempt to allay the vomiting by prescribing a hunger period or by diluting the food or curtailing the total amount given. Such devices . . . only serve to make . . . vomiting of the nervously unstable child very much worse. With these restless, hypersensitive infants it is above all necessary to avoid the stimulus of excessive hunger . . . *Treatment, if it is to be successful, must aim, not so much at controlling the vomiting as at allaying the nervous unrest.*"

Although small, frequent feedings are widely recommended for infants with GER,[1, 2, 39, 41] there are no data that demonstrate that limiting feedings in patients with functional or abnormal GER and failure to thrive lessens the frequency of vomiting, accelerates weight gain, or yields benefits that outweigh the stress caused by interruptions of feedings before satiation.[37, 42]

Cameron's management of nervous vomiting was limited in that it was infant-centered.[12] Future revelations concerning the reciprocal nature of the infant-mother relationship were unknown to him and he therefore viewed nervous vomiting as the result of "nervous instability" inherent in the infant, rather than acquired or exacerbated by malfunction of the nurturing relationship.

Improving the infant's comfort depends on relief of the parents' fears concerning their child's morbidity and mortality, helping them recognize their emotional and physical exhaustion, and, once acknowledged, exploring its causes and possible remedies. This begins a therapeutic process that heals the parent-infant relationship and is necessary for continued improvement beyond the period of clinical management. Hirschberg[5] has written a succinct, comprehensive outline of the theory and practice of this therapeutic process. Healing may be summarized schematically in terms of interrupting the vicious cycle depicted in Fig. 6. The patient's vomiting must cease to cause worry in the parents (step 1). To bring about this attitudinal change, it is necessary to show that the infant's condition does not deteriorate when antireflux measures are dispensed with (step 2). This may require hospitalization for objective clinical observation that goes beyond the parents' subjective assessment. Interrupting the cycle at step 3 requires that the parents have the comfort of an alliance with a caring, accessi-

ble, nonjudgmental physician whom they perceive as expert, trustworthy, and committed to their child's recovery. Hospitalizing the infant provides a supportive environment in which the mother is relieved of burdensome outside responsibilities while focusing on the care and protection of her infant with the support of nurses and child-activities personnel. Hospital personnel must not exacerbate her irrational guilt for having "caused" her baby's illness.

Interrupting the cycle at step 4 involves diagnostic and therapeutic interviews.[13] Interrupting the cycle at steps 5 and 6 occurs when the parents' optimism returns as they see their baby begin to gain weight and fuss less, and as diagnostic studies confirm the absence of organic disease. The number and types of technical medical procedures necessary for diagnostic clarity is based on the evidence suggestive of organic etiologies and, equally important, the infant's course after recognition and relief of physical and emotional discomfort; a prompt response to the therapeutic trial of comfort may lessen the need for testing.

It is very helpful, in patients whose illnesses are unexplained and may have a significant psychologic component, to enlist the participation of a mental health professional. However, this may derail progress when parents are averse to or cannot see the relevance of psychologic services in the management of their infant's physical symptom. In such cases, rather than permit the parent's refusal of mental-health participation to be a roadblock, the physician may use the rapport developed during management of the infant's vomiting to concurrently assess and manage the psychologic components of their infant's illness. He or she is enabled to do this by avoiding the dichotomy of physical versus mental disorder and, instead, taking a personal interest in the infant and family. Such an "undifferentiated" clinician, by avoiding psychologic jargon and other connotations of the mental health professional, is able to approach mental health issues in families who would have rejected such attention were it offered by a psychiatrist. Resistance to a mental health referral is, in itself, a mental health issue that the physician may help them overcome in time.

INFANT RUMINATION SYNDROME

Infant rumination is a potentially lethal kind of vomiting that begins when reciprocal interaction between the infant and the caregiver fails to take place.[43] The caregiver is, for any number of reasons, emotionally distant, unable to sense the baby's needs, and unresponsive to his or her signals.[44] The baby learns to bring up gastric content into his or her mouth for the purpose of self-stimulation and need-satisfaction that would normally be supplied by the caregiver.[45-49]

The classic description of infant ruminating behavior

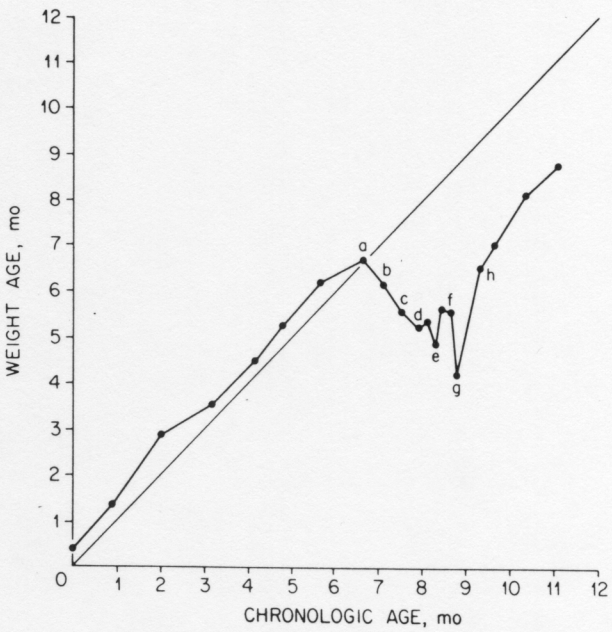

Fig. 8. The overall weight pattern in a patient with infant rumination syndrome at the onset of vomiting *(a)*, before his fourth and final hospital admission *(b-e)*, at his fourth hospital admission *(f)*, at the start of special nursing by a mother surrogate *(g)*, and at discharge from the fourth hospitalization *(h)*.

was, again, provided by Cameron.[12] The features of infant rumination syndrome[50] include the following: (1) Onset after 3 months of age, most commonly between 3 and 8 months. Its diagnosis in infants younger than 3 months is suspect because young infants have not yet reached the level of development enabling them to sense that they and mother are physically or emotionally separate persons participating in a relationship.[51] (2) Emesis of swallowed food leads to inanition. (3) The failure to thrive does not improve with antireflux management, formula changes, arm restraints, or gastrostomy feedings. (4) Rumination is an acquired skill and it is done without distress. (5) Vomiting does not occur during sleep or when the baby is actively interested in objects or people in his or her environment. It typically occurs while the baby is awake, quiet, and self-absorbed. There may be other self-stimulating behaviors (e.g., head rolling, hand sucking, or sound making). (6) The typical act of rumination begins with rhythmic contractions of the pharynx, tongue, and abdominal musculature that culminate in regurgitation of gastric content into the mouth and spillage to the outside. Whatever remains in the mouth is mouthed and reswallowed. (7) The mother has obvious difficulty in sensing what produces comfort and satisfaction in her baby. (8) The weight loss and rumination respond to nurturing that is sensitive and interactive. (9) The inability to nurture is one manifestation of more pervasive difficulties the par-

Table. Three functional vomiting syndromes of infancy

Characteristic	Innocent vomiting	Nervous vomiting	Infant rumination
Nature of vomiting	Involuntary Visceral Purposeless	Involuntary Visceral Purposeless	Voluntary Behavioral Self-stimulatory
Age of onset	As early as newborn period	As early as newborn period	After 3 mo
Mothering and mother-infant reciprocity	"Normal"	Attentive, but dyssynchronous; increases instead of relieves tension	Emotionally distant; little reciprocal mother-infant interaction
Typical circumstances	During wakeful state	*During* the baby's response to environmental stimuli	In the *absence* of environmental stimuli
Prognosis if untreated	Normal growth and development	Failure to thrive	Failure to thrive and possible death
Treatment	Effective reassurance that allows time to be used for "cure"	Lessening excessive stimulation; alleviating the tension-producing quality of mother-infant interaction	Increasing environmental stimulation; satisfaction of the infant's needs by mothering

ents have with interpersonal relationships. The parents usually have a history of emotional deprivation during childhood and marital dysfunction during adulthood. (10) Once the rumination and failure to thrive have been overcome, they typically do not recur.

The diagnosis of infant rumination syndrome is prompted by direct observation of the ruminating behavior. Cameron's[12] advice in this regard remains valid:

> "... rumination is very apt to be mistaken for- ... vomiting due to other causes, and it may require careful observation to make the distinction evident. Nor are such babies easy to observe ... Only when the child is alone and in a drowsy, vacant state, while nothing distracts attention or excites curiosity, does the act take place ... rumination can only be observed by stealth, by peering through peep holes, between screens and so forth."

The diagnosis of infant rumination syndrome is supported by diagnostic interviews that reveal the mother's history of emotional trauma during childhood, difficulties in her adult relationships, and family dysfunction.[52] The diagnosis is confirmed by the infant's response to treatment.

Although behavioral therapy employing aversive techniques (e.g., electric shock, aversive taste stimuli, and punishment) and positive reinforcement[53] may be useful in suppressing rumination in highly motivated adults or children with mental retardation and neurologic handicaps,[54] the application of such techniques in infant rumination syndrome may be harmful. This is especially true if the therapeutic goals are limited to suppression of the symptom without addressing the infant's unmet needs for mothering and the mother's inability to engage him or her in a mutually satisfying relationship. Fundoplication, if successful, also will stop rumination. However, if fundoplication is used as the principle therapy without addressing the underlying psychosocial abnormality, it burdens the infant and parents with a painful surgical procedure that is quite unnecessary.

The most humane, developmentally appropriate, and comprehensive management involves reversing the baby's weight loss and tendency to ruminate by providing a temporary mother-substitute, helping the mother to change her feelings toward herself and her baby, and improving her ability to recognize and supply her infant's physical and emotional needs.

A therapeutic trial of comfort in infant rumination consists of employing a nurse or other competent mother-substitute to hold, socially interact with, and feed the infant during the hours he or she is awake. The nurse should be relaxed and maternal enough to enjoy this work. Social stimulation need not be continuous or forced. The nurse should be sufficiently empathetic and observant to know when the baby enters the self-occupied state of withdrawal that fosters rumination, and she should respond to it promptly, by making social contact with the baby. The results of this procedure in one reported case are shown in Fig. 7, and the baby's overall weight course is shown in Fig. 8.[50] Similar results with use of holding therapy have been reported by others.[44, 47, 55] It is crucially important to foresee the response mothers have when a mother-substitute is used. Predictable feelings of guilt and anger, and her assumption that the physician judges her to be inadequate, should be anticipated. Ask the parents' permissions for this nursing procedure, the purpose of which is to help with data collection and child care. The nurse's role should be perceived to be that of helper to the physician and parents,

not didactic teacher of superior mothering technique. Diagnostic/therapeutic interviews with each parent strengthen rapport, provide opportunities to elicit some recognition of their emotional pain, and explore their willingness to accept more formal psychotherapeutic help.

SUMMARY

The table summarizes the similarities and differences between innocent vomiting, nervous vomiting, and infant rumination syndrome. Reflux occurs in esophagi; vomiting disorders occur in patients. The two should not be considered synonymous. Management of infant vomiting can be enhanced by recognition of clinically described functional vomiting syndromes and by assessing not only the vomiting but also the infant and family who seek medical help for it.

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