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Hyperlactation - How left-brained 'rules' for breastfeeding wreak havoc with a natural process

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"Hyperlactation: How Left-Brained 'Rules' for Breastfeeding Can Wreak Havoc with a Natural Process."

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Abstract

A variety of arbitrary and often unphysiologic rules for breastfeeding are frequently suggested to breastfeeding mothers. Many of these rules duplicate strategies commonly used to increase milk supply, and thus when undertaken by the many women who already have a generous milk supply, can lead to overproduction. Oversupply, or hyperlactation, is a frequent yet often unrecognized problem that can present with a variety of distressing symptoms for the breastfeeding mother and her infant. Infants may present with symptoms suggesting colic, milk protein allergies, or gastroesophageal reflux, or may present with unusually rapid or slow growth. Mothers may present with tender leaking breasts, sore infected nipples, plugged ducts or mastitis, or even the perception of insufficient milk supply. With an understanding of the pathophysiology of these symptoms, proper diagnosis and breastfeeding management can allow milk production to return to homeostatic levels and provide dramatic symptom relief.

Keywords: breastfeeding, hyperlactation, oversupply, breastfeeding patterns

Introduction:

Fifty years ago, women in the United States were arbitrarily told to limit breastfeeding to a four hour schedule. As a result, many women were unable to produce enough milk to feed their infants, and more and more women resorted to formula feeding. This was, of course, purely iatrogenic, but the cultural consequence was that many mothers and health care providers in the United States now still believe that insufficient milk production is a common and likely concern. Moreover, in the absence of a cultural history of easy and ubiquitous breastfeeding, and without an established understanding of the physiology of breastfeeding and lactation, health care providers now often pass on to mothers historical recommendations and rules about breastfeeding for which there are no clear physiologic rationale. Many of these rules—at least so many minutes on a side, always feed on both sides, always offer the full side—probably date back to those days of four hour feeds, and are essentially strategies for maximizing milk production.

Thus, as more and more women are breastfeeding in the United States, we are seeing more women who already have plenty of milk, trying to breastfeed according to these culturally defined rules. At the same time, we are now seeing both infants and mothers presenting with a whole series of new problems, and mother's milk is typically blamed. How can women's milk in the United States pose such problems when we see no such similar effects in the animal kingdom, or even in women in other parts of the world where breastfeeding is more common?

In our tertiary specialty practice, limited to breastfeeding medicine, we have seen approximately 2800 dyads in the past eight years. Of these, we estimate half have sought our help for mother and/or infant problems associated with hyperlactation. The infant feeding behaviors and gastrointestinal symptoms vary and are described in Table 1.

[Insert Table 1]

While we all recognize these as common infant problems, there are little data on their incidence or prevalence. A study by Adams & Davidson[1] on almost 1000 infants, found rates of colic to be similar among breastfed, formula fed or mixed fed infants, ranging from 19-21%. When a formula fed infant exhibits these symptoms the mother is frequently instructed to change formulas. But when it is a breastfed infant, the mother may be told she is overfeeding, underfeeding, that her milk is "too thin" or that something in her diet is causing the infant intestinal gas, or food allergy. Such suggestions can lead to severe elimination diets, formula supplementation or premature weaning. The difficulty maintaining a strict diet, the stress of caring for an uncomfortable infant, added to the concern that her milk is causing the distress, and the cost of medications and extra doctors visits, can often lead a mother to wean. Table 2 outlines common diagnoses and misdiagnoses which may be associated with symptoms of hyperlactation.

[Insert Table 2]

From our clinical practice experience, we maintain that many of these symptoms are not primarily caused by any individual mother's milk and only rarely are related to maternal diet. The purpose of this paper is to: describe the clinical symptoms we often see, and explore an alternative hypothesis for these symptoms and their etiology, describe the pathophysiologic basis as we understand it, and then offer our own physiologically based recommendations for management. These recommendations do not replace the need for accurate medical diagnosis and care and are not meant to encourage self-treatment in women and infants experiencing these symptoms. Very little has been published about maternal hyperlactation. What we describe here is based on our own experience in our tertiary breastfeeding medical practice, as well as our

understanding of the physiology of lactation. We've also drawn on the observations of Woolridge[2-4] and Livingstone[5] about this syndrome.

Hyperlactation was first described in 1988 by Michael Woolridge[2] in a case report, actually describing an infant with failure to thrive. He was the first to introduce the concept that feeding management influenced the caloric value of the breastmilk delivered to the infant. Woolridge[3] had shown in 1982 that infants have the capability to self-regulate their caloric intake and in a subsequent review he concluded that cultural restrictions on the frequency and duration of feeds potentially compromises milk quality[4] pp.236-237). Livingstone[5,6] in 1996, described the maternal and infant hyperlactation syndromes, their pathophysiology and management. She focused on correct breastfeeding technique and feeding infants on cue, as well as fully draining a breast to allow for adequate higher fat milk intake.

Mothers experiencing abundant milk supply present to our office with a variety of symptoms in themselves and their infants. The constellation of symptoms will vary with the mother's anatomy, physiology, and vulnerability to cultural pressures, and with mother and infant's temperaments and interactions.

Symptoms

- Infant symptoms:

Babies can exhibit a variety of symptoms and often arrive with a variety of diagnoses. (Tables 1 & 2) These infants often "act hungry all the time", nursing very frequently, as if "starving". Yet, clinically they gain weight very well, frequently much faster than normal, crossing to higher weight percentiles rapidly in the first months of life. Rarely, an infant may fall below the expected growth curve for breastfed babies, and may be termed "failure to thrive"[2]. Spitting up is common; this and their visible distress make gastroesophageal reflux a

predictable misdiagnosis or secondary co-diagnosis. Symptoms of colic may also be primary or secondary. Colicky symptoms, combined with explosive, or green stools can lead to the diagnosis of “lactose intolerance”. Such symptoms combined with mucousy, heme-positive stools can lead to diagnoses of milk protein allergy.[7]

The infants present a wide variety of feeding styles at the breast. Some may gulp and “gobble” with visibly large swallows, appearing “gluttonous”. For those who swallow air, large burps are common. Infants may seem to struggle with the milk flow, sometimes choking or coughing at the breast. Some may pull and tug, appearing to fight at the breast. Others may pinch the nipple, despite a previous experience with comfortable latching. Still others may nurse with a loose mouth, described as a lazy or "weak” suck, and yet be gaining weight quite well. Many infants will demonstrate several of these patterns at different feedings. Mothers are often baffled by their infants' behavior at the breast, and may also report some feedings, or a particular time of day, when nursing is easy and without these problems.

- Maternal symptoms:

In this article we are focusing primarily on the infant's symptoms, but the mothers also can present with a variety of symptoms (Table 3). The mother’s symptoms relate primarily to the large amount of milk produced and her infant’s response to it. Infants who pinch to control flow can injure their mothers' nipples, leading to sore nipples and nipple infections. Rapid milk production can lead to milk stasis, so plugged ducts and mastitis are common. Because the infants tend to be unsettled and manifest excess hunger, many of these women actually believe they do not have enough milk, and may present to the clinician seeking methods for enhancing milk production. If they are already taking measures to increase milk production, they may find

their symptoms, or their infants' symptoms, actually worsening. Many women have no specific symptoms, but others report the symptoms outlined in Table 3.

[Insert Table 3]

It is not unusual for specific situations to bring on problems in a mother with a tendency for abundant milk production. Many mothers and babies present to us between three and six weeks postpartum, a common time for growth spurts, but many mothers describe some symptoms as early as ten days or two weeks postpartum. Infant growth spurts tend to exacerbate the pre-existing problems, as do periods of stress and hectic times like holidays, vacations, and relatives visiting.

- Possible causes of infant and maternal symptoms

We believe that these symptoms are caused by a vicious cycle of milk overproduction caused by interference with normal physiologic processes. As Woolridge[4] proposed, current culturally accepted arbitrary rules of breastfeeding management can interfere with ordinary homeostatic mechanisms. This can result in the iatrogenic production of increased volumes of lower fat milk. This change in the volume and caloric content of the milk, unchecked by ordinary homeostatic mechanisms, can lead to a vicious cycle of disturbing symptoms for both mother and infant, and a cascade of events that can then lead to further breastfeeding problems and premature weaning.

Before we describe the pathophysiologic basis for these symptoms, it is important to understand the normal physiology of milk production. Only with a solid understanding of the basic physiologic principles involved is it possible to understand what is going awry, so that practitioners may help the mother and infant establish a feeding rhythm that works for them and meets their needs.

Overview – the physiologic basis for the regulation of human milk production

Human milk production is regulated by a supply and demand process that occurs through the interaction of infant and mother. Key to this process are a variety of factors: infant behaviors of appetite and satiety, maternal response to infant behavior, infant suckling, maternal pituitary hormonal response to infant suckling, and local alveolar conditions affecting response.

- Maternal (Endocrine) Control of the Initiation of Milk Production

Before the baby is born, and in the first few days postpartum, milk production proceeds without any input necessary from the infant. This early process is hormonally driven, controlled entirely by the mother's endocrine system. This milk secretion will occur whether or not the mother plans to breastfeed.

- Infant (Autocrine) Control of the Maintenance of Milk Production

However, once the volume of milk increases, the switch from the endocrine control of milk production (i.e. driven solely by maternal hormones) to autocrine control (driven by infant milk-removal), transfers the regulation of milk production from mother to infant[8,9]. From that point on, the mother's breasts and hormonal system are designed to shut down lactation, and it is only the infant's suckling, and the removal of milk, which is responsible for continued milk production. Indeed, the infant who has frequent access to the breast in the first few days postpartum can actually increase the volume of colostrum even before the more mature milk comes in. Autocrine control is the basis for the colloquially termed "supply and demand" response that allows the infant to regulate the production of milk to match his appetite. Breast milk synthesis is governed by the quantity and quality of infant suckling and milk removal; thus infant appetite drives milk production.[10-12]

The maintenance of established milk synthesis that is controlled by the autocrine system of supply and demand is termed galactopoiesis. (Figure 1: The autocrine control of milk production by the healthy baby). This occurs from approximately the second week postpartum through weaning.

[Insert Figure 1]

- Infant effects on maternal milk production

Milk production is directly stimulated by prolactin and indirectly by oxytocin; and it is the infant's appetite, or the removal of milk via some other means, that is the primary stimulus to maternal pituitary release of these hormones.[13,14] Moreover, the rate of milk production is inhibited by the presence of milk itself in the alveoli, which is why milk production stops in the absence of milk removal, for example with weaning or formula feeding. So the baby is not only responsible for stimulating pituitary release of the hormones that promote milk production, the infant is also responsible for regulating the factors that inhibit milk production.[9,14,15]

  - Prolactin

Prolactin is secreted by the maternal anterior pituitary in response to nipple stimulation and sucking stimulus. Secretion depends upon the frequency, intensity, and duration of nipple stimulation[13,14]. It has been hypothesized that the frequent removal of milk in the early weeks postpartum results in increased numbers of prolactin receptors in the glandular cells of the breast which can influence the total amount of milk the mother is able to produce.[16,17]

  - Oxytocin

Oxytocin is secreted by the maternal posterior pituitary in response to infant suckling, as well as in response to a variety of other neuroaffective and neurosensory factors, causing the

milk ejection reflex. Under the influence of oxytocin, the myoepithelial cells that surround the alveoli in a basket-like arrangement contract to expel milk into the ductules.[18]

- Feedback Inhibitor of Lactation

When the mammary alveolus is relatively full of milk, a decrease in the rate of milk synthesis has been observed at the local alveolar level. It has been hypothesized that a peptide on one of the whey proteins found in human milk probably serves as negative feedback to milk synthesis. Although not yet identified specifically, this peptide has been named the feedback inhibitor of lactation (or "FIL") and is believed to be the way that the baby's fluctuating appetite is able to control alveolar milk production to so exactly meet the infant's needs. When the alveolus is relatively full, less milk is made, but when the alveolus is relatively empty, and less of the FIL peptide is present, the rate of milk synthesis increases.[9]

- Interpretations of relevant research

Therefore, the early establishment of a good milk supply involves frequent, effective milk removal. The frequency of removal affects the rate of milk synthesis, while the amount of milk removed and how fully the breast is emptied together effect overall milk production.[9,19,20] Recent research[21] confirms this earlier work[9,13,14] that the fullness of the breast directly affects the autocrine mechanisms controlling the short-term rate of milk synthesis.

Establishing a good milk supply depends on a variety of factors. As Hartmann et al.[10,11] view it, a woman's breasts' milk "storage capacity" is one factor which will effect the frequency at which her infant will need to nurse to achieve a given milk supply. As they interpret their own data, they believe that each woman has an inherent primary characteristic milk storage capacity of the breast, defined as the maximum volume of milk that can be stored in the breast between feedings. By their definitions, this capacity provides a baseline capacity that determines the

maximum volume available to an infant at a given feed. In their view, the infant's appetite determines the mother's total absolute milk production, and, given her inherent baseline milk storage capacity and the infant's caloric needs, the infant's appetite will determine how frequently the infant nurses at the breast.[11,22] Their interpretation of these findings is that infants whose mothers have smaller storage capacities will make up the difference by nursing more frequently than do those infants whose mothers have larger storage capacities.[11,22] They therefore suggest that it is the frequency of infant feeding and milk removal that indirectly affects the rate of milk synthesis as a function of the mother's storage capacity.[22]

However, in our own view, there is another way to interpret Hartmann et al.'s data. In this scenario, the mother's storage capacity may not be a primary characteristic exclusively inherent in her anatomy, but is also determined by the infant's feeding patterns. That is, we suggest that the feeding patterns may be the independent variable, which help determine the mother's storage capacity, the dependent variable, rather than vice versa. We propose this view of Hartmann's data because of what we have observed in our breastfeeding medical practice. Specifically, we have seen significant differences in feeding patterns and apparent storage capacities within the same woman at different times in lactation as well as in lactating with different children. For example, exclusively breastfed twins might feed infrequently whereas their older singleton sibling had been a frequent feeder. In addition, great variability has also been demonstrated across cultures related to breastfeeding frequencies and duration.[4]

Factors that effect infant feeding frequency or how well an infant empties the breast, thus could be seen as cause, rather than as effect, of the mother's storage capacity. To understand this, an analogy might be made to the formula fed infant's stomach capacity, which can enlarge to an unphysiologic eight or more ounces as the ***artificially fed*** infant is fed larger and larger

volumes less frequently than his breastfed peers. In a parallel fashion, the mother's ductal capacity may very well stretch to accommodate the larger volumes made by an infant emptying the breast quite well, but feeding less frequently than some of his peers.

A variety of factors might affect either the frequency of infant feeding or the degree of breast emptying, and these then would have an inverse effect on maternal storage capacity. Such factors might in some cases be maternal, and thus appear intrinsic, such as her understanding of how often and how "long" she "should" nurse. However, even these can change for an individual mother from one baby to the next. More often, infant factors will vary, and it is this variability that has made us look at Hartmann's data and come to different conclusions. Such variable infant factors include: (a) infant temperament; (b) infant age; (c) whether the infant is exclusively breastfeeding or also receiving pumped breastmilk or artificial baby milk; (d) the infant's total number and frequency of breastfeeds each day; (e) the relationship between the infant's appetite and behavior and his mother's response.

There is much that remains unknown about breast milk production. Nevertheless, we know that breastfeeding and lactation, like the other organ systems of the human body, represent processes that have maintained humans and mammals through the millennia, and thus can be presumed to "work," regardless of whether we understand every aspect of those processes. The processes of homeostasis allow the mother's breast physiology to meet the needs of her growing infant.

Whether it is breast milk storage capacity or infant demand that is primary, and whether it is milk production or infant behavior that is secondary, it is the homeostatic mechanisms that control these interactions that matter. It is the homeostatic response to variability that allows the baby to continuously effect his mother's milk production so that his own appetite and growth

needs can be met. It is only when arbitrary rules about breastfeeding interfere with this natural homeostatic process, when the infant's appetite or behavior is misunderstood, misinterpreted, or removed from this physiologic interaction with his mother, that an asynchrony can develop between mother and infant, and between milk production and infant needs (See Case Study Table 4).

The adequacy of an infant's milk intake can be assessed by a variety of methods. A high lipid meal provokes cholecystokinin, and consequent behaviors of satiety, which can be quite reliable indicators of good milk transfer. However, early on, these signs may be unreliable. In the very young baby, suckling may, via central oxytocin release, induce a transient but false satiety, with or without a lipid meal. Also, the dehydrated infant may be sleepy or slow to awaken in response to hunger; such an underfed infant might appear to the new mother to be content, and may not awaken despite hunger. Thus, early on, we encourage mothers to watch output—the frequency of urination as well as the frequency and consistency of bowel movements—to help assess the adequacy of intake. This early focus on adequacy of milk transfer, while important, may reinforce culturally based anxieties about the adequacy of the mother's milk production. Hill and Humenick (1989) report perceived insufficient milk supply to be a "universal" reason for early weaning and supplementation.[23]

Now that the normal physiology of milk production has been reviewed, a look at how these physiologic mechanisms might be disrupted, and a proposed pathophysiologic model for understanding hyperlactation and the symptoms will be described.

- Explanation of this clinical picture:

While normal variations in maternal anatomy and physiology and certain infant temperaments can certainly interact to create this clinical picture, more commonly the initial

cause of hyperlactation is cultural misinformation about optimal breastfeeding practices. Moreover, even when there are maternal or infant primary predispositions to rapid milk production, homeostatic mechanisms should normally lead to self-correction. But cultural ideas about breastfeeding can interfere with these physiologic mechanisms.

Switching sides arbitrarily by the clock, rather than switching for more physiologic reasons (e.g. on the basis of maternal comfort or infant behavioral cues) can result in the baby receiving excess lowfat milk and insufficient cream. Thus the infant, after feeding, has a full stomach of lowfat milk, yet is still hungry, and comes back for more, thus driving up the maternal supply. By the time the baby presents with symptoms, mother and baby are in the midst of a vicious cycle. The infant's appetite has created a large maternal milk supply, which in turn keeps the infant hungry, because the excess milk supply is primarily low calorie lowfat milk (See Case Study, Table 4).

- Normal physiology specific to the issues of abundant supply

To understand how this syndrome of abundant supply develops, it is helpful to understand the normal homeostatic mechanisms controlling milk production. In the brief overview of milk supply provided above, the emphasis is on infant appetite as the primary stimulus to maternal pituitary control of milk production. As presently understood, the lipid fraction is squeezed from the alveoli into the ducts with each milk ejection and diluted by the aqueous fraction of proteins, electrolytes and sugar.[24] Cregan & Hartmann[21] have demonstrated that the fuller breast delivers lower fat milk, while the emptier breast delivers creamier milk. This is because differing factors affect the rate of production of each of these fractions. Woolridge[4] states: "breast milk increases in caloric density during the feed as the volume available diminishes, so that calorie intake shows

a curvilinear relationship to volume intake, with the later stages of the feed making a disproportionate contribution to the baby's intake of calories" (p.223).

Suckling, as a major stimulus to oxytocin release, causes the milk released during the course of the feeding to be creamier than the milk immediately available at the beginning of a feeding. As present research suggests, our interpretation is that as a given feeding progresses, these boluses of creamier milk are diluted with progressively smaller aqueous volumes such that the milk available to the infant is creamier over time in smaller and smaller boluses. Under usual conditions, typically half the milk's calories are said to be in the milk fat, mostly in the creamy, slower flowing milk delivered at the end of the feeding.[21]

It is this increasing lipid content, transferred to the baby's gut, which stimulates cholecystokinin to produce a satiety that, together with the slower flow, permits the infant to relax and stop feeding. The frequency of maternal pituitary oxytocin release determines the frequency of these milk ejections. Other factors, including the time of day, frequency of feedings, infant behavior, the mother-infant relationship and maternal sense of well being, interact to affect the varying proportions between the lipid and aqueous fractions of milk.

- Pathophysiology in hyperlactation – explanation of the clinical picture.

If the mother switches from one breast to another prematurely, either by the clock or because she makes a left brained, cognitive decision that her breast is "empty", she overrules the right brained homeostatic mechanisms that allow the infant to adjust production to his thirst and appetite. Instead, she may be switching her baby to more high volume lowfat milk, just when the infant would have been getting to the lower volume cream. The baby's stomach may be full of lowfat milk, meeting thirst, but the infant is not satiated and remains hungry for the calories demanded for growth. So the baby suckles more, further stimulating maternal prolactin.

The increased milk removal decreases negative feedback from alveolar milk suppressor peptides (FIL) and the rate of milk production is accelerated. Each time milk fat is squeezed into the ducts, even with later letdowns, it is thus diluted with a somewhat larger volume of lowfat milk. Thus, despite the mother's frequent milk ejections, her infant receives primarily lower fat milk. In addition, although maternal oxytocin is released in response to infant suckling and other "warm and fuzzy" positive somatosensory cues, the hormone can be inhibited by pain, anxiety, and the adrenergic state.[25,26] Thus, maternal distress can result in less frequent milk fat release.

Infant symptoms involve both direct reactions to the high milk flow at the breast, as well as subsequent response to the consumption of higher volume lower fat feeds. When there is an abundant supply, maternal milk ejections can be strong, overwhelming the infant. Depending on temperament and experience, each infant develops his own strategies for dealing with this rapid flow. Some infants will respond to these strong milk ejections and high milk flow by tugging and pulling at the nipple, apparently narrowing the milk ducts to decrease the flow. Other infants will pull off the breast when confronted with a high flow, possibly to be squirted with the spray. Some infants simply pinch the nipple to control the flow, injuring their mother's nipples. Other infants appear "lazy" as they hold their mouths loosely and receive the abundant flow.

These behaviors can often be magnified or diminished by the mother's responses. If a mother misinterprets her infant's behavior, tugging, fighting, and pulling off the breast, and believes that the infant "doesn't like" the breast, the milk, or mother herself, this will adversely affect the nursing relationship, and can further inhibit the frequency of milk release. On the other hand, a mother's calm reassurance with stroking and soft voice can often calm the infant to allow the infant to manage the flow.

Another distressing infant symptom is that of hunger, despite a "full" stomach. This is related to the large intake of lowfat milk in the absence of lipid-induced satiety. Without satiety, the infant remains hungry, distressed, and demonstrates the higher muscle tone seen with hunger. This full stomach, in the face of both hunger and persistent high abdominal muscle tone, can easily result in spitting up or symptoms of gastroesophageal reflux. The resulting distress can result in frequent comfort nursing, which actually could be therapeutic if the infant were able to nurse on an emptier, creamier breast. But further high volume feeds only exacerbate the symptoms. Moreover, because the infant is now drinking higher volumes of lower fat milk, with little lipid to slow digestion, the gut can easily be subjected to transient lactose overload, temporarily outstripping available lactase, creating the potential for symptoms of colic [25] and explosive or green stools (Table 1). These symptoms are most distressing to the parents, and infants are often diagnosed with reflux, colic or lactose intolerance. Because spitting up is common, this and their visible distress make gastroesophageal reflux a predictable secondary co-diagnosis. Symptoms of colic may also be primary or secondary (Table 2). In our experience, when the dyad learns how to manage their abundant milk supply, these infant symptoms usually disappear (See Case Study, Table 4).

Symptoms of colic, when combined with mucousy stools or blood in the stool, can often suggest allergy. At this time, too little research has been done in this area. The possibility exists that allergy may be either cause or result of hyperlactation, and it may also be possible that some apparent allergy symptoms may be purely the result of hyperlactation without any true allergy at all.

It is possible that rapid milk transit through the intestine, in the absence of the lipid that slows digestion, and combined with a relative lactose overload, may itself cause a mucosal tear

and microscopic blood, irritation, and a mucous response. Such a mucosal tear could also permit the passage of foreign proteins, setting up the potential for allergy. However, our understanding is that the pathogenesis of food allergy, or macromolecular transport in the gastrointestinal system, is still under study.[27,28] The distressed infant, whether distressed because of allergy, classic colic, or lactose overload, will seek comfort, and if this comfort involves suckling on an already full breast, a vicious cycle ensues.

The full exposition of the maternal symptoms of hyperlactation is beyond the scope of this paper. Leaking, engorged, and tender breasts, as well as problems with plugged ducts and mastitis can be explained by the excess milk volume and lack of adequate or complete drainage of the breasts by the infant. Sore nipples, nipple infections, or ductal candidiasis can be explained by the nipple trauma caused by the infant's attempt to regulate the flow and the continuous skin exposure to leaking breast milk.

- Clinical management:

For infants whose symptoms are relatively mild and of recent onset, the course is usually quickly reversed within a week or two by letting the baby stay on each breast for an entire feeding, and waiting until the next feeding before going to the alternate breast. Even after symptoms have resolved, usually the mother can expect that most feedings will continue to be one side at a time, but this should never be followed as a strict rule. Whenever a mother perceives that her infant is hungry "too soon," the softer, "emptier," or most recently used breast might be the first place to start. Again, this is a suggestion better left to comfort and instinct than to left-brained instruction.

The general plan is for each breast to be alternately well emptied, and then subsequently left full for longer than before. In this way, the infant is able to drink the creamy milk that

promotes satiety and longer periods between feedings, while the "unnursed" breast stays full longer, allowing the negative feedback that can slow the rate of production. Thus the goal is to restore a relaxed feeding situation that both mother and baby can enjoy, increasing the rate of maternal milk ejection while slowing the rate of aqueous production.

For more entrenched symptoms, we often take a different approach, individualized to the particular circumstances. We must modify the plan if the mother has secondary plugged ducts, nipple trauma, or infection. It is beyond the scope of this paper to address these maternal issues. However, the general plan is always to help the breasts alternate well between quite "empty" and quite full, while letting maternal and infant comfort guide the moment to moment decisions about the process. Depending on the situation, for these more longstanding or more extreme symptoms, we will usually suggest that the mother use a pump to help her make this alternation between "empty" and "full" more exaggerated. For several days, or even a week or so, each breast is "emptied" extra well at least once a day, usually by pumping immediately before or after nursing. If the milk at any of these sessions is particularly thin, as is often the case with very high volumes pumped in the morning, the first most watery ounce of milk might be set aside, so that the rest of the pumped milk is then that much creamier than it would have been.

The rest of the day the mother nurses as usual, probably alternating breasts, while at times giving the infant the milk she has pumped earlier. In this way she allows her breasts to stay full for a little bit longer than usual. When exactly that pumped milk is fed will vary, as we find it best to individualize each plan to a particular dyad's circumstances. But in all cases the rationale is to allow the breasts to stay comfortably full long enough to permit that negative alveolar feedback necessary for the rate of production to decrease. During this time, most of the pumped milk should not be stockpiled but should be fed to the infant at some point each day.

Pumping well at least once a day permits the infant the opportunity to nurse on a less full breast, and helps protect the mother from the development of plugged ducts during this process. In addition, the mother can pump, nurse, or hand express to comfort over the course of the day to help prevent plugged ducts during this process.

This entire process for slowing production can sometimes take awhile. Pseudophedrine has recently been shown to decrease milk production and has been proposed as a treatment for hyperlactation.[29] Although published clinical evidence or ethno-botanical information is lacking, we have found that herbal remedies, such as sage tea, are a useful adjunct to breastfeeding management of hyperlactation.[30]

Over time, as the milk production slows, the feedings at the breast will be getting easier, as the mother finds herself pumping smaller volumes of creamier milk at the few times a day that she is pumping. However, because the pumped milk is not needed as often to finish the feeds, mother and baby gradually transition off this plan, by listening to what works.

A key component of this management is helping breastfeeding become enjoyable and comfortable for both mother and baby. This can allow the baby more relaxed feedings, and more frequent maternal oxytocin release, yielding smaller, more frequent milk release, which should result in both creamier milk and smaller volumes with each milk ejection.

- Counseling and education:

Thorough counseling and education are important so that the mother understands the process and can adjust the plan as needed**.**

The mother should NOT stick to the plan rigidly. It is important that she let her own comfort, and her baby's comfort be her guide. That is, there cannot be a lot of rules and instructions. With a few general guidelines and expectations about how to approach feeding

issues, this must return to a right brained process. If the left brain is permitted to control the process, we will continue to interfere with the right brained processes that are inherent in how our bodies maintain homeostasis. Regardless of the plan, if her baby gets frustrated or fidgety, or if the mother herself is uncomfortable, she should not continue to keep the baby in an uncomfortable position. She can switch to the other breast, or to her shoulder, or do whatever she can to calm the baby. After the infant is calm, she can let the baby stay where he is, or if he still seems hungry, she may want to return him to one breast or the other.

If necessary, when she starts the new side, she may want to pump or express just a little of the lowfat milk off first, if she otherwise expects the baby would choke and sputter. This shouldn't be done as a general rule or expectation, but only as needed, in a decision of the moment, so that it is comfort, i.e. the right brain, that is making this decision.

The mother may also be offered anticipatory guidance with regard to the normalized sensations of reduced breast fullness and milk ejection. Careful clinical management is critical to prevent plugged ducts and the risk of mastitis, and to prevent increasing production on one side as we decrease it on the other.

- Summary/Conclusions:

Hyperlactation is an under-recognized problem that often goes misdiagnosed. Even when secondary co-diagnoses are correctly identified, their treatment is complicated by failure to recognize and treat the underlying hyperlactation. Hyperlactation itself is not something inherent in the mother's anatomy or physiology, or caused by the infant's feeding style, but is rather a vicious cycle of behaviors initiated and reinforced by cultural expectations and rules for feeding which overrule basic instincts towards homeostasis.

Breastfeeding, lactation, and the communication between mother and baby are, like all of the body's processes, mediated by neurohumoral and right-brained communications. It is very easy for left brain cognitive processing to interfere with what should be natural and instinctive behaviors. We believe this left-brained interference with mothers' neurologically based instincts is the major cause of the vicious cycle of the symptoms of hyperlactation. Learning to trust her body, to listen to her infant, and to let comfort needs guide behavior, can help restore comfort to the feeding situation, and in this way help them stop the vicious cycle of symptoms of abundant milk supply.

480 Table 1: Infant symptoms which may occur as a result of feeding mismanagement.

Feeding behaviors at the breast	Gastrointestinal symptoms after feeding
Hungry “all the time “	Burping, spitting up
Gobbles and slurps	Fussy, crying
Chokes and sputters	Gassy, colicky
Tugs or “fights” the breast	Explosive or green stools
Clicking, pinching	Mucousy or blood streaked stools
“Lazy”, “loose latch”	

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Table 2: Common diagnoses, misdiagnoses, and assessments which may lead to the suspicion of hyperlactation. These diagnoses may be a) primary, causing hyperlactation; b) secondary to hyperlactation; or c) misdiagnoses.

Health care providers: Diagnoses	Lactation consultants: Assessments
Colic	Overactive MER
GER (gastroesophageal reflux)	High need or fussy baby
Allergies	Bad latch in later weeks
Lactose Intolerance	Plugged ducts
Not enough milk	Yeast
OB's: mastitis	Not enough milk

Table 3. Maternal symptoms which may occur as a result of feeding mismanagement.

Milk Volume	Sore Nipples	Sore breasts
Leaks, sprays, and pours	Pinched	Tender, overfull breasts
Rapid flow	Injured	Plugged ducts
OR Perception of “not enough”	Infected	Mastitis

Table 4

Case Study

First visit:

5 1/2 weeks postpartum

Presenting symptoms:

Mother:

Tender, leaking breasts

Sore nipples, sensitive to touch, cloth, shower

Infant:

Gastroesophageal reflux diagnosed.

Colicky, gassy, fussy.

Hungry all the time, feeds constantly.

Chokes and coughs, gobbles and slurps at breast.

Fights, tugs at breast, latches on and off, pinches nipple. but miserable on the breast.

Pertinent history:

Mother nurses strictly by clock, 10 minutes each side.

Hx of mastitis in early weeks.

Infant: Rapid weight gain: Birthweight 8 lbs 13oz; 10 lb at 2 wks; 12 lbs 8 oz at 1 month.

8-10 watery yellow stools a day

Mother's exam:

Nipples pink, breasts with tender masses.

Compression stripe on nipple after nursing.

Infant's exam:

14 lbs 0.8 oz lbs, very tense muscle tone, fussy, calms to mother's voice.

Mouth without thrush.

Breastfeeding observation:

Tight latch, initial 5/10 pain reported, improved with feeding.

Infant nursed with nose in and chin out (a method of controlling flow).

Gulped and grunted on breast.

Repeatedly came off with milk spilling out of his mouth, then returned to breast.

Nursed briefly on left side only, taking in 2.4 oz in a short time, ending feeding hungry but too distressed to return to breast.

Assessment:

Maternal hyperlactation caused by vicious cycle driven by infant appetite in response to clock nursing .

Nipple infection secondary to infant pinching nipple to control flow.

Infant excessive hunger, rapid weight gain, colic, gastroesophageal reflux, and difficulty feeding all caused by mother's high lowfat milk volume making it difficult for infant to satiate.

Recommendations:

Mupirocin and nipple care for mother.

Time out from breastfeeding until nipples heal.

Suggestions for daily pumping to alternate between

- pumping breasts well to avoid plugged ducts and get creamy milk,
- leaving breasts full to slow rate of production

Anticipatory guidance so that maternal and infant comfort guide the process.

Resolution:

Returned to breast ten days later, feeding much easier.

Reflux and colic fully resolved 2 weeks after retuning to breast.

536 Figure 1. The autocrine control of milk production by the healthy baby.
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538 [Insert Figure 1 here]
539

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