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

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2 “Hyperlactation: How Left-Brained ‘Rules’ for Breastfeeding Can Wreak Havoc with a Natural  
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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

## 41 Introduction:

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

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

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

64 [Insert Table 1]

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

77 [Insert Table 2]

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

87 understanding of the physiology of lactation. We've also drawn on the observations of  
88 Woolridge<sup>2-4</sup> and Livingstone<sup>5</sup> about this syndrome.

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

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

## 102 *Symptoms*

### 103 • Infant symptoms:

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

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

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

123         • Maternal symptoms:

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

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

134 [Insert Table 3]

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

141 ▪ Possible causes of infant and maternal symptoms

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

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

154

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

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

160        • Maternal (Endocrine) Control of the Initiation of Milk Production

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

165        • Infant (Autocrine) Control of the Maintenance of Milk Production

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

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

181 [Insert Figure 1]

182 • Infant effects on maternal milk production

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

190 ○ Prolactin

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

196 ○ Oxytocin

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

199 milk ejection reflex. Under the influence of oxytocin, the myoepithelial cells that surround the  
200 alveoli in a basket-like arrangement contract to expel milk into the ductules.<sup>18</sup>

201       ○ Feedback Inhibitor of Lactation

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

210       • Interpretations of relevant research

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

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

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

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

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

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

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

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

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

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

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

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

288 • Explanation of this clinical picture:

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

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

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

303 • Normal physiology specific to the issues of abundant supply

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

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

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

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

328 ■ Pathophysiology in hyperlactation – explanation of the clinical picture.

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

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

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

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

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

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

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

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

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

393 • Clinical management:

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

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

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

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

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

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

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

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

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

444   • Counseling and education:

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

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

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

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

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

465     • Summary/Conclusions:

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

472           Breastfeeding, lactation, and the communication between mother and baby are, like all of  
473 the body's processes, mediated by neurohumoral and right-brained communications. It is very  
474 easy for left brain cognitive processing to interfere with what should be natural and instinctive  
475 behaviors. We believe this left-brained interference with mothers' neurologically based instincts  
476 is the major cause of the vicious cycle of the symptoms of hyperlactation. Learning to trust her  
477 body, to listen to her infant, and to let comfort needs guide behavior, can help restore comfort to  
478 the feeding situation, and in this way help them stop the vicious cycle of symptoms of abundant  
479 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”	

481

482 Table 2: Common diagnoses, misdiagnoses, and assessments which may lead to the suspicion of  
 483 hyperlactation. These diagnoses may be a) primary, causing hyperlactation; b) secondary to  
 484 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

485

486 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

487

Table 4

488	
489	Case Study
490	<b>First visit:</b>
491	5 1/2 weeks postpartum
492	<b>Presenting symptoms:</b>
493	<b>Mother:</b>
494	Tender, leaking breasts
495	Sore nipples, sensitive to touch, cloth, shower
496	<b>Infant:</b>
497	Gastroesophageal reflux diagnosed.
498	Colicky, gassy, fussy.
499	Hungry all the time, feeds constantly.
500	Chokes and coughs, gobbles and slurps at breast.
501	Fights, tugs at breast, latches on and off, pinches nipple. but miserable on the breast.
502	<b>Pertinent history:</b>
503	Mother nurses strictly by clock, 10 minutes each side.
504	Hx of mastitis in early weeks.
505	Infant: Rapid weight gain: Birthweight 8 lbs 13oz; 10 lb at 2 wks; 12 lbs 8 oz at 1 month.
506	8-10 watery yellow stools a day
507	<b>Mother's exam:</b>
508	Nipples pink, breasts with tender masses.
509	Compression stripe on nipple after nursing.
510	<b>Infant's exam:</b>
511	14 lbs 0.8 oz lbs, very tense muscle tone, fussy, calms to mother's voice.
512	Mouth without thrush.
513	<b>Breastfeeding observation:</b>
514	Tight latch, initial 5/10 pain reported, improved with feeding.
515	Infant nursed with nose in and chin out (a method of controlling flow).
516	Gulped and grunted on breast.
517	Repeatedly came off with milk spilling out of his mouth, then returned to breast.
518	Nursed briefly on left side only, taking in 2.4 oz in a short time, ending feeding hungry but too
519	distressed to return to breast.
520	<b>Assessment:</b>
521	Maternal hyperlactation caused by vicious cycle driven by infant appetite in response to clock
522	nursing .
523	Nipple infection secondary to infant pinching nipple to control flow.
524	Infant excessive hunger, rapid weight gain, colic, gastroesophageal reflux, and difficulty feeding
525	all caused by mother's high lowfat milk volume making it difficult for infant to satiate.
526	<b>Recommendations:</b>
527	Mupirocin and nipple care for mother.
528	Time out from breastfeeding until nipples heal.
529	Suggestions for daily pumping to alternate between
530	• pumping breasts well to avoid plugged ducts and get creamy milk,
531	• leaving breasts full to slow rate of production
532	Anticipatory guidance so that maternal and infant comfort guide the process.
533	<b>Resolution:</b>
534	Returned to breast ten days later, feeding much easier.
535	Reflux and colic fully resolved 2 weeks after retuning to breast.

536 Figure 1. The autocrine control of milk production by the healthy baby.

537

538 [Insert Figure 1 here]

539

540

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