Recognizing and Treating Delayed or Failed Lactogenesis II

Nancy M. Hurst, RN, DSN, IBCLC

Delayed or failed achievement of lactogenesis II—the onset of copious milk volume—occurs as a result of various maternal and/or infant factors. Early recognition of these risk factors is critical for clinicians who interact with breastfeeding women so that intervention and achievement of full or partial breastfeeding can be preserved. This article describes the maternal and infant conditions that contribute to the unsuccessful establishment of a full lactation. Treatment modalities that can maximize maternal lactation capacity and infant growth rates are offered. J Midwifery Womens Health 2007;52:588–594 © 2007 by the American College of Nurse-Midwives.

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One of the most frequently cited concerns voiced by new breastfeeding mothers is the question of whether she is providing sufficient milk for her infant.[1] This concern, coupled with the national initiative to increase breastfeeding rates,[2] points to the critical responsibility of clinicians who care for breastfeeding women to ensure proper instruction and follow-up when needed. The ability to recognize delayed or failed lactogenesis is essential so that clinicians can intervene to both assure that infants' nutritional needs are being met and to potentially preserve full or partial breastfeeding. This article describes the normal onset of lactogenesis and conditions whereby lactogenesis II is delayed or fails to completely attain sufficient milk volume levels to support adequate infant growth. Implications for clinical practice and suggested management strategies are reviewed.

PHYSIOLOGY OF LACTOGENESIS

In simplistic terms, lactation is the process of milk secretion, and it occurs as long as milk is removed from the breast on a frequent basis. However, the process of lactation and the act of breastfeeding is quite complex, because a range of factors in the mother's external and internal environment determines her breastfeeding efficacy. Her internal environment includes her physical and mental health, past experiences and intentions related to breastfeeding, and body image, all of which impact her breastfeeding experience. Her external environment, such as socioeconomic factors, her general physical environment, and spousal, family, and hospital staff support also influence breastfeeding success. And most importantly, the quality and quantity of maternal–infant interaction during the early postpartum period, sometimes described as the fourth trimester, sets the stage for a successful breastfeeding experience.

There are five distinct stages of human mammary gland development: embryogenesis, puberty, pregnancy, lactation, and involution.[3] The first two stages lay the groundwork for glandular growth; full development and maturation of the mammary epithelium await the hormones of pregnancy. By mid-pregnancy, the mammary glands have developed extensively and small amounts of secretion product are formed; however, the glands continue to develop until parturition, with the secretory process being held in check by the high circulating plasma concentrations of progesterone. The change that occurs between pregnancy and lactation is called lactogenesis.

Lactogenesis is a two-stage event. Lactogenesis I occurs during pregnancy and is the initiation of the synthetic capacity of the mammary glands. Lactogenesis II commences after delivery and is the initiation of plentiful milk secretion.[4] Concurrent with the increase in milk secretion associated with lactogenesis II are significant changes in several milk constituents, termed "biomarkers of lactation," as the transition from colostrum (high concentration of total protein, immunoglobulins, sodium, and chloride; low concentrations of lactose, potassium, glucose, and citrate) to mature milk (a reversal in concentration of these factors) takes place.[5] These changes in milk composition—coupled with a sudden feeling of breast fullness—identify the onset of lactogenesis II, which usually occurs between 30 and 40 hours following the birth of full-term infants.[3]

Lactation is influenced by a complex hormonal milieu including reproductive hormones (estrogen, progesterone, placental lactogen, prolactin, and oxytocin) and metabolic hormones (glucocorticoids, insulin, growth, and thyroid). The reproductive hormones act directly on the mammary gland, whereas the metabolic hormones act indirectly by altering endocrine response and nutrient flux to the mammary gland.[6] Ductal growth is primarily regulated by estrogen and growth hormone, and alveolar development requires progesterone, prolactin, and possibly placental lactogen.[7] During pregnancy, the high levels of circulating progesterone inhibit the secretory process of the mammary gland. Once the placenta is expelled after birth, progesterone levels decline rapidly, and increasing prolactin levels trigger the beginning of

Address correspondence to Nancy M. Hurst, RN, DSN, IBCLC, 2803B Houston Ave., Houston, TX 77009. E-mail: nmhurst@texaschildrenshospital.org

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lactogenesis II, which is the onset of copious milk secretion. Oxytocin is essential for milk removal from the mammary gland.[8] In response to infant suckling, afferent impulses from sensory stimulation of nerve terminals in the areola travel to the central nervous system triggering the release of oxytocin from the posterior pituitary. In turn, oxytocin is carried through the bloodstream to the mammary gland where it interacts with specific receptors on the myoepithelial cells located on milk-secreting cells (alveoli) and ducts, initiating contraction of the cells, which results in expulsion of milk from the gland.[9]

The main role of insulin appears to be in regulating nutrient fluctuation to the mammary gland by shunting nutrients away from traditional storage depositories, thereby making them more readily available for milk synthesis.[10] Thyroid hormones are essential for efficient milk production and, in animals, appear to be necessary for mammary responsiveness to growth hormone and prolactin during lactation.[11] One study in women with insufficient lactation found that the nasal administration of thyrotrophin-releasing factor (TRH) increased prolactin and daily milk volume.[12] In this randomized, double-blinded study, 19 women with insufficient lactation (<50% of normal milk yield) were allocated to receive TRH or a placebo nasal spray. At the end of 10 days of treatment, milk yield increased significantly ($P = .014$) in the TRH group from a mean of 142 ± 33.9 g/d to 253 ± 105.3 g/d compared to no change in the placebo group. Additionally, prolactin levels increased in the TRH group from a mean of 117 ± 45.2 μg to 173 ± 55.5 μg ($P < .001$), whereas in the placebo group prolactin levels decreased from 137 ± 69.5 μg to 82 ± 37.7 μg.

The early influence of these reproductive and metabolic hormones sets the stage for a transition to the autocrine function of the mammary gland. Autocrine control, also known as local control, refers to a mechanism whereby the gland regulates its own function through the local production of hormones and growth factors.[13] Evidence that the rate of milk secretion within individuals (and between breasts in the same mother) is directly correlated with the frequency of milk removal strengthens the theory of local control.[13,14] The mechanisms that regulate local control are not fully understood, but may include factors such as intramammary pressure,[15] milk removal,[4] bioactive factors in the milk that interact with milk cell membranes,[13,16] or a combination of these factors.

In addition to these anatomical and physiologic processes, breastfeeding is a process that also involves psychological and emotional responses in the mother. Many areas within the maternal brain, such as the amygdala, the striatum, the vagal motor and sensory nuclei, and preganglionic sympathetic neurons of the intermediolateral column of the spinal cord[17] undergo profound morphologic and secretory changes during lactation. Although prolactin and oxytocin are primarily secreted by the pituitary gland, both hormones are also secreted in these higher brain regions and have been implicated in promoting maternal behavior.[18] Furthermore, these lactogenic hormones can be released in the brain not only by suckling, but also by close physical contact such as that experienced during breastfeeding, thereby maximizing the neurohormonal response.[9]

OTHER FACTORS THAT INFLUENCE BREASTFEEDING SUCCESS

Consideration of the maternal neurologic, physiologic, and psychologic responses to lactation, collectively known as the mother's internal environment, gives an incomplete picture of the factors influencing lactation and breastfeeding. The external environment of the mother can provide a nurturing, supportive atmosphere or, in contrast, include obstacles that work to counter a successful experience. These external obstacles may result in behaviors and responses in the mother that either require extraordinary effort to overcome or that disrupt the breastfeeding relationship entirely. Positive social support, maternal confidence and attitude,[19] intent to breastfeed,[20] and knowledgeable/supportive health care providers[21] are associated with higher breastfeeding initiation and duration. Considerable evidence shows that older, non-smoking, employed women with more years of education, higher socioeconomic standing, and previous breastfeeding experience tend to initiate and continue breastfeeding for longer durations.[22] Perceived maternal barriers, such as a father's attitude toward breastfeeding, the quantity of milk produced, and time constraints associated with return to employment all impact the initiation and duration of breastfeeding.[23]

DELAYED AND FAILED LACTOGENESIS II

Delayed lactogenesis II denotes a longer than usual interval between the colostrum phase and copious milk production, but whereby the mother has the ability to achieve full lactation. Failed lactogenesis II is a condition wherein the mother is either able to achieve full lactation but an extrinsic factor has interfered with the process, or one or more factors results in failure to attain an adequate milk production. Failed lactogenesis can be described further in the context of two types of conditions: a primary inability to produce adequate milk volume, or a secondary condition as a result of improper breastfeeding management and/or infant-related problems.

Although actual rates of failed and delayed lactogen-

Nancy M. Hurst, RN, DSN, IBCLC, is the assistant director of the Texas Children's Hospital Lactation Program and Mother's Own Milk Bank in Houston, Texas, and an assistant professor of pediatrics at Baylor College of Medicine.

esis are unknown, estimates ranging from 5% to 15%, respectively, have been reported.[24] A variety of situations and conditions have been implicated as potential contributing factors to a delay or failure in the onset of lactogenesis II (Table 1). As previously stated, some hormones indirectly influence mammary gland responsiveness and thus maternal conditions with a hormonal etiology (e.g., diabetes, hypothyroidism, or obesity) may cause a delay in lactogenesis II. Additionally, some delivery modes and conditions that result in a delay in breastfeeding initiation and/or breast stimulation (e.g., preterm, cesarean, or a prolonged second stage of labor [>1 hour])[4,25] can also delay the onset of copious milk secretion. Examples of primary lactation failure include conditions in the mother such as anatomic breast abnormalities or hormonal aberrations. Insufficient mammary glandular tissue, postpartum hemorrhage with Sheehan syndrome,[26] theca-lutein cyst,[27,28] polycystic ovarian syndrome,[29] and some breast surgeries have been implicated as possible causes of lactation failure. While minor breast surgeries (i.e., lumpectomy) may have little effect on lactation, procedures that require invasive manipulation of the nipple/areolar complex,[30] such as the placement of breast implants, or reduction mammoplasty,[31] may disrupt normal lactation. Possible causes of secondary lactation failure include any condition in the infant that results in an ineffective/weak suck (i.e., prematurity, tongue-tie, palatal anomalies, or congenital heart defects); any condition in the mother that results in incomplete breast emptying (i.e., improper latch-on, timed/scheduled feedings, overuse of pacifiers, the unnecessary use of supplements); and some maternal medications (i.e., pseudoephedrine, progestin-only and/or estrogen containing birth control methods). It should be noted that any of the factors implicated in a delay in lactogenesis II can lead to a secondary failure of lactation if not effectively managed.

Table 1. Risk Factors for Delayed or Failed Lactogenesis II

Delayed Lactogenesis II
Primiparity
Psychosocial stress/pain
Maternal obesity
Diabetes
Hypertension
Stressful labor and delivery
Unscheduled cesarean section
Prelacteal feeds; delayed first breastfeed episode
Low perinatal breastfeeding frequency
Hormonal contraceptive administration first week postpartum
Failed Lactogenesis II and/or Low Milk Supply
Breast surgery/injury
Retained placental fragments
Cigarette smoking
Hypothyroidism, hypopituitarism
Ovarian theca-lutein cyst
Insufficient mammary glandular tissue
Polycystic ovarian syndrome
Postpartum hemorrhage with Sheehan's syndrome

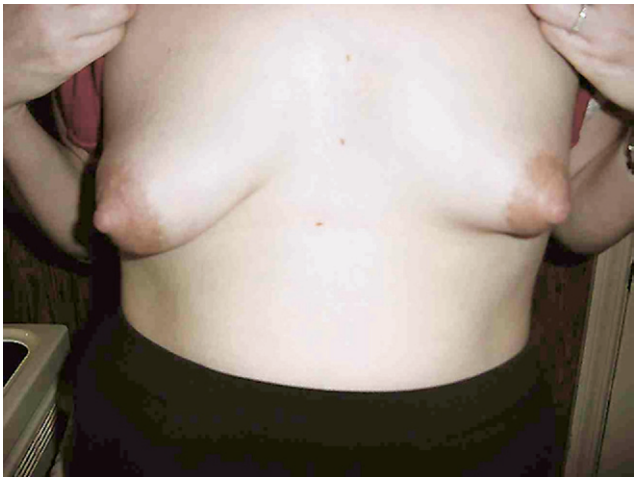

Figure 1. Hypoplastic breasts: Low milk volume. Reprinted with permission from Wilson-Clay B and Hoover K.[32]

DIAGNOSIS

Risk assessment is important for identifying women with potential lactation insufficiency. A clinical lactation assessment (see Appendix) performed during pregnancy is useful to identify possible cases that may require closer surveillance following delivery. A history of breast surgery (e.g., augmentation or reduction), breast hypoplasia,[33] and previous insufficient lactation and/or slow-growth breastfed infant(s) should warrant concern that delayed or failed lactogenesis may occur. Wide-spaced, tubular in shape, underdeveloped breasts have been associated with failed lactogenesis[33] (Figure 1). Assessment of nipple type and protractility will alert the clinician to possible problems related to the ability of the infant to effectively grasp and maintain attachment to the breast, which could result in suboptimal stimulation and subsequent insufficient milk volume. Following delivery, additional information relevant to lactation sufficiency as previously discussed (e.g., labor and delivery course, maternal medications, etc.) should be obtained in order to provide an overall view of potential risks for lactation insufficiency.

The breast is the only organ in the body that does not have a diagnostic test to measure its adequacy.[4] This fact presents clinicians with the difficult task of using indirect measures to determine lactation sufficiency. Test weighing procedures, whereby the infant is weighed pre- and postfeeding to estimate milk intake during breastfeeding and biochemical markers can both be used as measures to document the onset of lactogenesis II. However, these procedures may be impractical, costly, and cumbersome to use routinely. In a study of 60 mothers following cesarean section, Chapman and Perez-Escamilla[2] defined

Table 2. Signs of Ineffective Breastfeeding

Infant weight loss >7%
Continued weight loss after postbirth day 3
No audible swallowing from the baby
<6 wet diapers per day after day 4
<3 stools per day after day 4
Infant is irritable/restless or sleepy
Minimal/no breast changes by day 5 after delivery
Persistent/increasingly painful nipples

delayed lactogenesis II as milk transfer <9.2 g/feeding at 60 hours and maternal perception of the lack of breast fullness, swelling, and leaking at 72 or more hours postpartum. They found maternal perception to be a valid indicator of delayed onset of lactogenesis II. Based on these findings, maternal perceptions are an effective starting point in diagnosing a delay in lactogenesis II. Once a delay is diagnosed early, and if aggressive intervention fails to result in a rapid regain in momentum of the lactation process, it should be assumed that a failed lactogenesis II exists.

INTERVENTION

The following interventions are intended to treat a suspected delay or early lactation insufficiency with the purpose of improving lactation performance. Most women are capable of producing some measurable milk volume; however, these suggested treatments are intended to stimulate the mother's full lactation potential. The challenge is determining the most effective plan of care based on the etiology of the problem, the ability of the mother to execute the plan, and at what point her full lactation potential has been reached. Treatment modalities to promote lactation sufficiency and breastfeeding success are designed to maximize breast stimulation and to assure adequate infant growth rates. Taking a "wait and see" approach may result in a delay in appropriate interventions of early breastfeeding problems. Simultaneous maternal and infant assessment to determine the possible etiologies involved will drive the proper treatment plan.

A delay in the onset of lactogenesis II warrants early recognition in order to provide timely intervention and prevent further lactation insufficiency. Recent trends prompting earlier discharge from the hospital/birthing center make it more difficult for clinicians to assess and recognize a delay in lactogenesis II and ineffective infant feeding ability. Early follow-up (during the first week post-birth) allows for the assessment of adequate milk intake and identification of possible signs of ineffective breastfeeding (Table 2). The presence of two or more risk factors in Table 1 should alert the clinician to the need for closer surveillance of breastfeeding in the first few days following hospital discharge until signs of adequacy of milk intake in the infant are demonstrated by frequent patterns of urination (about five–six wet diapers) and stooling (at least three–four yellow, seedy stools). Enlisting the expertise of a lactation consultant may be useful in developing an effective plan of care, offering support and instruction to the mother, and accessing other breastfeeding equipment/supplies (i.e., hospital-grade breast pump).[34]

When lactogenesis II failure is suspected, the underlying cause will drive the plan of care. Definitive diagnosis and proper treatment of any hormonal imbalance (e.g., thyroid, pituitary, or diabetes) must occur before and/or concurrently with measures to stimulate and increase milk production. Removal of retained placental fragments[35,36] and normalizing of testosterone levels[27,28] (i.e., theca-lutein cyst) has resulted in normal onset of lactogenesis II. Depending upon the onset of polycystic ovarian syndrome and various treatment strategies, improvement of current or future lactation may be improved.[29] Early recognition of ineffective breastfeeding (Table 2) and assessment of risk factors identified from relevant information obtained in the prenatal and early postpartum period (see Appendix) will alert the clinician to the need for close surveillance in the early days and weeks following birth. A treatment plan for a delay or suspected lactation failure should include the following key elements: providing adequate infant nutrition, maximizing breast stimulation and complete breast emptying, strategies to measure milk intake during breastfeeding, written record of progression of feeding plan, and recognition of when maternal lactation potential is reached.

Provide Adequate Infant Nutrition

Determining the need for supplementation is essential in promoting adequate infant growth and energy levels. An infant who is malnourished will not have the energy to breastfeed effectively; recommending that the mother simply increase the number of breastfeeds per day to improve her milk volume and the infant's milk intake will not improve the situation when failed lactogenesis II is suspected. Delayed lactogenesis II, defined as little or no maternal perception of breast fullness or leaking ≥72 hours post-birth, may require only minimal, short-term supplementation. Ideally, expressed breast milk or formula should be given as a complement (immediately following the breastfeeding episode) rather than a supplement (in place of a breastfeed) in order to maximize maternal breast stimulation and to maintain infant breastfeeding ability. To determine the feeding method used to provide supplement/complement feedings (i.e., bottle, cup, syringe, or feeding tube devices), careful consideration should be given to the ability of the mother and infant to use these devices. A feeding tube device allows delivery of expressed breast milk or formula while the infant is at the breast. The Lact-Aid (Lact-Aid Interna-

tional, Inc., Athens, TN) and Medela Supplemental Nursing System (Medela, Inc., McHenry, IL) are two commercially available feeding devices. To use these devices effectively, the infant must be able to latch onto the breast and the mother must be able to manage the device. The advantage of using a feeding tube device is the ability to simultaneously provide breast stimulation and deliver supplemental milk feedings.

Maximize Breast Stimulation and Complete Breast Emptying

Any potential infant suckling problems, such as poor latch or tight frenulum, should be assessed and corrected. When breast engorgement or nipple trauma/pain is present, proper treatment to alleviate the symptoms and promote maternal comfort and milk flow should be employed. Mechanical breast pumping with an effective hospital-grade breast pump following each breastfeeding should be initiated whenever a delay or failed lactogenesis is suspected. This practice serves to increase breast stimulation and promote complete breast emptying. Use of various galactagogues (e.g., metoclopramide, domperidone) have been shown to increase prolactin levels and milk production in some cases.[37] Although no published studies exist on the effectiveness of herbals, there are anecdotal reports of increased milk production with the use of fenugreek (1000–1500 mg three times daily).

Objective Measurement of Milk Intake During Breastfeeding

Subjective estimation of the volume of milk the infant receives during a breastfeed is inaccurate.[38] Test-weighing procedures are an important diagnostic and management tool in the early management of suspected delayed or failed lactogenesis II, and are essential in determining the infant's ability to transfer the available milk during breastfeeding and manage extra milk feedings.[39] The test weighing procedure involves weighing the clothed infant under exactly the same conditions before and after feeding with an electronic scale (accurate to at least 5 g), then subtracting the prefeed from the postfeed weight. With this procedure, 1 g of weight gain approximates 1 mL of milk intake. In situations where renting a scale is cost-prohibitive, weekly provider visits to assess breastfeeding patterns, volume of supplement, and postfeeding pumping volumes are recommended. Providing mothers with the ability to accurately measure and monitor their progress will serve to guide appropriate levels of supplementation and assessment of measures used to improve maternal milk volume.

Maintaining Feeding/Pumping Record

Having mothers maintain a simple record of daily feeding, pumping, and infant stooling and urination patterns may be useful to monitor their progress. This record can guide the plan of care to allow for modifications as breastfeeding improves (or not). For instance, the mother may see an increase in the amount of milk transferred by the infant during breastfeeding (by test weights) but no increase in postfeed pumped volume. Without the use of test weights, the mother might conclude that the postfeed pumping is not effective; however, her pumping efforts have served to stimulate milk production and breast emptying, and as a result, more available milk for the infant. As discussed previously, it is important to determine the ability of the mother to manage a specific plan of care. Whereas some mothers may find a simple feeding diary useful in monitoring her progress, others may find it cumbersome and stressful. As clinicians, it is important to recognize the most effective management strategy and individualize the plan of care based on each mother and her abilities.

Recognizing When Maternal Lactation Potential Has Been Reached

Helping the mother recognize the point at which she has reached her lactation potential is useful in providing a reassessment of her long-term breastfeeding goals. Recognizing when that potential is reached is the challenge. Assuming that the appropriate plan of care for the underlying cause(s) of a delayed or failed lactogenesis has been followed, an assessment of planned outcomes reached will guide continued management. Assessment of proportion of daily breastfeeding, supplements, and expressed breast milk volumes obtained from pumping will provide the clinician with the necessary information to determine when lactation potential has been reached. For example, when mechanical pumping postbreastfeeding and/or galactagogue administration results in no appreciable increase in milk production, the clinician can interpret that the mother has achieved her full lactation potential. Determining the extent to which each mother is able to provide her infant's feedings at breast or with expressed breast milk will give them a realistic plan as their infant develops. The clinician should be prepared for an emotional session with the mother at this point, especially for those who had a goal of exclusive breastfeeding. However, when the mother recognizes that all interventions have been exhausted and she has put forth the best effort possible, she is less likely to feel a sense of failure and loss and more likely to eventually feel successful.

SUMMARY

Understanding the mechanisms in which various factors influence lactation and breastfeeding is required as a basis for analyzing the possible effects on this process in situations where delayed or failed lactogenesis is suspected. Recognizing when and how to intervene in order to properly detect and assess the degree to which lacta-

tion is compromised will allow for individualized interventions and appropriate follow-up. A collaborative effort between nurses, midwives, physicians, and lactation consultants will serve each mother with a coordinated and individualized plan of care for her unique situation. Finally, helping the mother recognize her full lactation potential, even when it falls short of exclusive breastfeeding, can result in a feeling of success and accomplishment.

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Appendix. Clinical Lactation Assessment

Breast History *(Check if applicable.)*

A. Breast surgery: ☐ Yes ☐ No

If yes, what type?: ☐ Biopsy ☐ Augmentation ☐ Reduction ☐ Radiation ☐ Other_____

Which breast was involved?: ☐ Both ☐ Left ☐ Right Year of procedure:_____

If augmentation, type of incision used:

☐ Periareolar ☐ Submammary ☐ Axillary ☐ Transumbilical

If reduction, type of procedure used: ☐ Free nipple transplantation ☐ Pedicle technique

Loss of nipple sensation? ☐ Yes ☐ No

B. Did your breasts enlarge during puberty?: ☐ Yes ☐ No

C. Did your breasts change during this pregnancy?: ☐ Yes ☐ No

D. Have you breastfed before?: ☐ Yes ☐ No If yes, how many times?_____

How long with each child?: _____

E. Did you supplement with formula?: ☐ Yes ☐ No

Reason(s) for supplementing: _____

Breast Assessment

General breast shape:

- ☐ Round, normal lower medial and lateral quadrants
- ☐ Hypoplasia of the lower medial quadrant
- ☐ Hypoplasia of the lower medial and lateral quadrants
- ☐ Severe constrictions, minimal breast base

Breast symmetry: ☐ Symmetrical ☐ Noticeable asymmetry

Intramammary distance: ☐ $<$1.5 inches ☐ $\geq$1.5 inches

Nipple type (appearance at rest): ☐ Everted ☐ Flat ☐ Inverted

Nipple protractility (movement with areolar compression): ☐ Evert ☐ Flatten ☐ Retract inward