Chapter 2
Anatomy and Physiology of Breastfeeding

2.1 Anatomy

This overview is not meant to be comprehensive. Rather, it contains key factors that help provide a context in which to discuss breastfeeding. There are many books that describe breast anatomy and changes during pregnancy and lactation for those who would like to delve more fully into the subject. Some of those resources are listed at the end of this chapter.

2.1.1 Anatomy of the Infant

The anatomy of newborns is different from that of adults, or even of toddlers. These anatomical differences make breastfeeding possible.

All babies are born with a small degree of retrognathia, or physiologic jaw retraction. This results in a relative posterior positioning of the base of the tongue, and renders the newborn a predominantly obligate nasal breather. The tongue is also larger in neonates than adults relative to the jaw. The base of the tongue sits far back in the throat over the epiglottis. The newborn's larynx is in a higher position relative to an adult and sits near the soft palate (see Fig. 2.1).

This anatomy allows an infant: (1) to breathe even with a teat filling his mouth and his nose pressed up against the breast;

L. Dahl, *Clinician's Guide to Breastfeeding*,
DOI 10.1007/978-3-319-18194-3_2

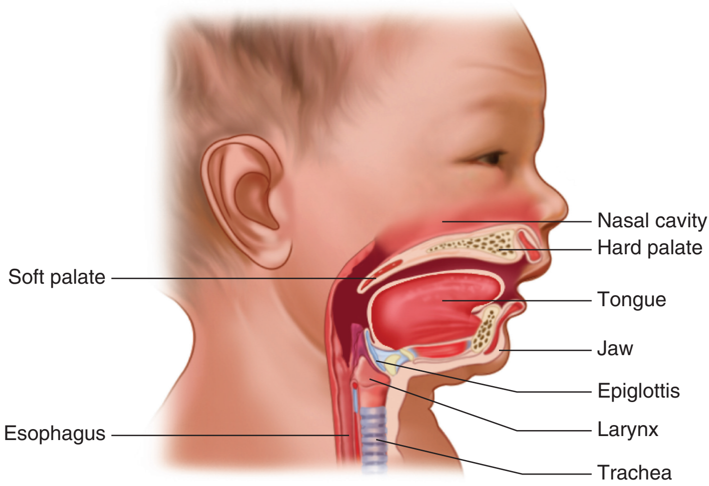

Fig. 2.1. Babies are born with a small degree of retrognathia, which results in a relative posterior positioning of the base of the tongue. The tongue is larger in neonates relative to the jaw, and the larynx is higher and sits near the soft palate.

and (2) to swallow milk without worry of having the liquid spill into the trachea. However, this also means that the newborn has to coordinate sucking, swallowing, and breathing in a very specific way with little margin of error (Sanches 2004).

2.1.2 External anatomy of the breast

On the exterior of the breast are the areola, nipple, and Montgomery glands. The areola is the circular, pigmented area of the breast. Centered in the areola is the nipple. The nipple is composed of horizontal and longitudinal smooth muscle fibers that contract in response to touch. Nipples carry the openings (galactophores) of the lactiferous ducts, which transport and store milk. There are 6–10 such openings in each nipple, corresponding to the 6–10 lobes of milk-producing glandular tissue in each breast (see Fig. 2.2: External and Internal breast anatomy).

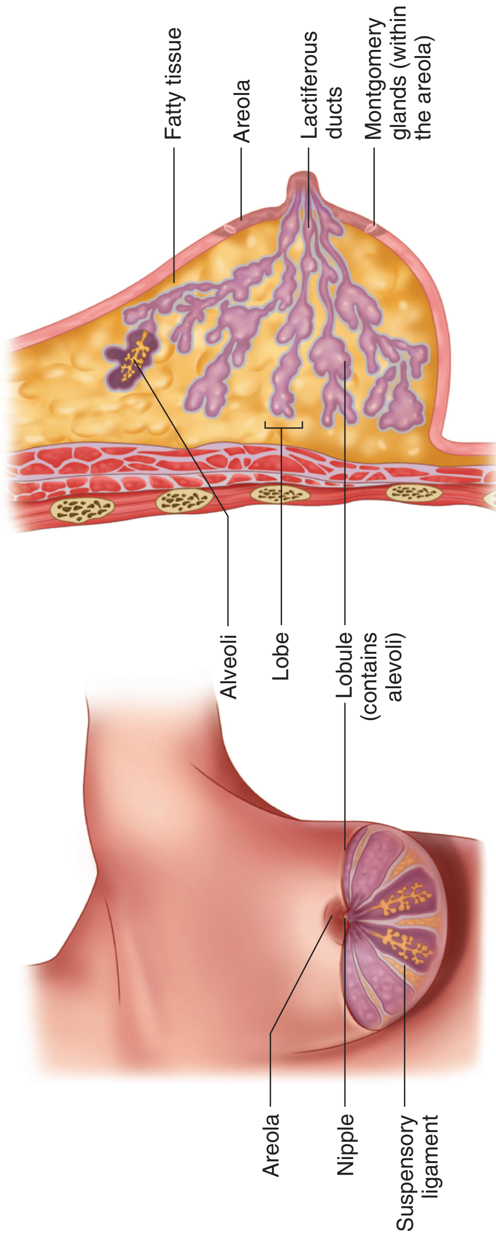

Fig. 2.2. External and Internal breast anatomy.

Surrounding the nipple, within the areola, are Montgomery glands, which secrete an oily liquid in the lactating breast. The yellow liquid creates a seal on the delicate nipple to prevent damage, but it may actually do much more. Decades of research have shown that the breasts of lactating women secrete compounds that affect newborn behavior. They can elicit arousal in sleepy babies (Sullivan and Toubas 1998; Russell 1976; Soussignan et al. 1997), and calm fussy ones (Schaal et al. 1980; Sullivan and Toubas 1998). The odor can also induce appetite (Russell 1976; Soussignan et al. 1997), directional crawling (Varendi and Porter 2001) and head turning behavior (Schaal et al. 1980; MacFarlane 1975; Makin and Porter 1989). The source of these compounds has been disputed, but Doucet et al. (2009) determined that these powerful compounds may come from the oily liquid secreted from the Montgomery glands.

2.1.3 Internal anatomy of the breast

In 1840, Sir Astley Paston Cooper published *On the Anatomy of the Breast*, the most advanced medical text of the time (Cooper 1840). Cooper was a passionate surgeon with a keen interest in dissection, going so far as to steal his neighbors' corpses in order to continue his self-directed education (Burch 2010). (He would also perform public dissections of executed criminals in a combination of egoism and showmanship.)

Cooper injected varying colors of wax into ducts and blood vessels of the breasts of recently deceased women to better understand the internal structures of the breast—including the lactating breast—and to visualize their relationships (see Fig. 2.3).

Cooper's study was so detailed that it became the definitive medical model of the breast, and remained so until 2005. Many physicians practicing today learned all they know about the breast from data that was more than 150 years old.

In 2005, researchers published the results of a study examining the breasts of lactating women using high-resolution

Fig. 2.3. Ducts and glandules. “Ducts and glandules,” from: *On the anatomy of the breast*, by Sir Astley Paston Cooper, 1840. Courtesy of Thomas Jefferson University, Archives & Special Collections.

ultrasound imaging (Ramsay et al. 2005). This study, as well as others that followed (Geddes 2009; Hassiotou and Geddes 2013), have highlighted inconsistencies in the anatomical literature that impact breast physiology and breastfeeding management, including differences from one of the most venerated texts, *Gray's Anatomy*. The information in this chapter is drawn from the newest research, based on state-of-the-art technologies.

The breast is composed of fatty and glandular tissues. Throughout pregnancy, estrogen causes the breast to change from predominantly adipose tissue to being made up primarily of glandular tissue. Studies have shown that a lactating breast transforms from having twice as much adipose as glandular tissue to having twice as much glandular tissue as adipose, although the percentage can vary greatly from woman to woman. These changes, called *lactogenesis I*, are complete by gestational week 22 in most women, but there is enormous variation in timing and degree (Cox et al. 1999). Mothers with preterm babies younger than 28 weeks may not have fully experienced the breast changes required for lactation. Therefore lactation may be delayed in preterm births (Cox et al. 1999).

The glandular tissue is comprised of alveolar sacs (also called alveoli) and the milk duct system. These tissues are made up of two layers of epithelial cells. The inner layer contains cuboidal cells, which can differentiate into milk-secreting cells called lactocytes under the influence of progesterone and estrogen. The outer layer is made of myoepithelial cells, which have the properties of smooth muscle and contract in response to hormones. The alveolar and ductal tissues are supported within the breast by a loose framework of fibrous connective tissues called Cooper's ligaments, named for the same Sir Astley Cooper who was so fond of dissection.

In the lactating breast, tracing inward from the nipple, the terminus of each duct forms into alveoli, which produce and store most of the milk. Alveoli contain lactocytes in the luminal layer, and myoepithelial cells in the outer layer (Sternlicht 2006; Watson and Khaled 2008). Lactocytes are predominantly present in the alveoli and directional, in that they point toward the lumen where milk is secreted (Fig. 2.2: External and Internal breast anatomy).

During lactation, the milk is transported from the alveolar sacs via the ductal system to the nipple, where it is expelled through the nipple ducts.

Ductal structures are loosely associated into lobules, each of which contain between 10 and 100 alveoli (Hartmann 1991). In turn, these lobules are arranged into lobes. Each lobe is surrounded by dense fibrous connective tissue with embedded fat cells, referred to as intra-lobular stroma. This stroma contains mesenchymal cells that are highly responsive to hormonal cues (i.e., oxytocin), and which are associated with the development of lactating breasts (Bissell et al. 1999; Wiseman and Werb 2002). The lobes of glandular tissue are intertwining and of varying sizes, with up to 30-fold differences in volume (Moffat and Going 1996). It was formerly thought that alveolar sacs swell to form "pools" of milk in large sacs, but the size of the alveoli remain relatively consistent—about 0.12 mm in diameter each (Hartmann 1991). There are just more of them to contain more milk in lactating breasts.

Most of the glandular tissue is in the anterior part of the breasts, directly underneath the areola, and therefore more easily accessible to the infant.

In addition to these changes in glandular tissue, estrogen also causes an increase in blood flow to the breasts. By 24 weeks of pregnancy, blood flow is doubled compared to prepregnancy. This change persists throughout lactation. Most of the extra blood flow comes from the two main vessels supplying the breasts: the Internal Mammary Artery (IMA) and the Lateral Thoracic Artery (LTA). The breast is additionally fed by the intercostal arteries and the thoracoacromial artery. Superficial veins also become more prominent in pregnancy and throughout lactation. The ratio of blood flow to milk yield is approximately 500:1 (Linzell 1960; Christensen et al. 1989).

Nerve supply to the breasts comes from the second to sixth intercostal nerves, which divide into superficial and deep branches (Cooper 1840). The deep branches supply the glandular tissue and nipple, and the superficial branches supply the nipple and areola (although sensory innervation is predominantly found in the nipple). Nerves have been identified along major ductal systems, but not near smaller ducts. There is motor innervation of the smooth muscle of the areola and nipple, (Courtiss and Goldwyn 1976) and of the mammary arteries (Cowie 1974). But there is no motor innervation of the lactocytes or myoepithelial cells, which means they are under hormonal, not neurological control.

2.2 Physiology

2.2.1 Hormone Effects and Regulation

The main hormones responsible for the production and maintenance of milk supply are progesterone, prolactin, oxytocin, cortisol, and Feedback Inhibitor of Lactation (FIL).

During pregnancy, high levels of *progesterone* suppress the production of milk by inhibiting the release of prolactin. Within 48–72 hours after birth, progesterone levels decrease, triggering prolactin release (Suzuki et al. 2000; Czank 2007; Pang and Hartmann 2007).

Prolactin is stimulated by infant suckling and occurs 7–20 times a day. It is produced in the anterior pituitary gland, and is positively and negatively regulated (it can be inhibited by progesterone, estrogen, norepinephrine, and dopamine). Prolactin causes stimulation of mammary glandular growth and epithelial cell proliferation, as well as milk production (Neville et al. 2002).

Serum prolactin levels are high during the first few weeks, but wane after that. After about week 3, prolactin returns to near prepregnancy levels. This is one of the reasons why early evaluation of potential breastfeeding problems is so crucial. During the first week, frequent stimulation of touch receptors on the breast stimulates an increase in prolactin receptors. With more available prolactin receptors, even as prolactin levels naturally decrease, milk production can be maintained for the duration of nursing (Jacobs 1977; Cox et al. 1996).

Initially, the circulating levels of prolactin vary throughout the day, with the highest level occurring about 1 h after sleep, and the lowest level in midmorning. Prolactin levels peak 30 min after the beginning of a feeding, preparing the breast for the next feeding.

If milk accumulates in the breast, the binding of prolactin is reduced. It is hypothesized that this is due to change in lactocyte shape when the alveoli are filled with milk, effectively deactivating the prolactin receptor (Berry et al. 2007). Full alveoli have lowered uptake of prolactin, and empty alveoli have higher binding affinity of prolactin (Cox et al. 1996; Cregan et al. 2002; Daly et al. 1993). When prolactin cannot bind, milk synthesis slows, so full breasts result in an inhibitory effect on milk production. As the alveoli empty, prolactin can again bind, which allows the alveoli to again fill with milk.

In addition to prolactin, infant suckling also stimulates *oxytocin*, resulting in milk ejection, or "letdown." It also results in uterine contraction to help the uterus shrink back down to pre-pregnancy size. By suckling, the infant stimulates touch receptors around the areola and nipple, which create impulses that activate the dorsal root ganglia, spinal cord, and hypothalamus, resulting in oxytocin release from the posterior pituitary gland. When oxytocin is released, it binds to receptors on the myoepithelial cells that line the alveolar ducts, causing these cells to contract and expel the stored milk from alveoli into larger ducts (Prime et al. 2007; Cowie 1974). This is called the milk ejection reflex.

The milk ejection reflex is transient, lasting between 45 s and 3.5 min. It is also pulsatile, meaning that oxytocin is secreted more than once, resulting in multiple ejections during nursing or pumping (Drewett et al. 1982; McNeilly et al. 1983; Uvnas-Moberg et al. 1990). Prior to the milk ejection reflex, very little milk can be removed, so it is critical for successful nursing (Young et al. 1996; Kent et al. 2008). This is why mothers often get less milk from pumping alone: because the touch receptors on the areola are not stimulated in the same way as by the baby's mouth so there is less oxytocin release and therefore less milk ejection. Oxytocin also prevents the unsuckled breast from ejecting milk to maintain positive pressure in that breast so the baby can nurse more easily from that side when the time comes.

Mothers can sense the milk ejection reflex happening with feelings of warmth, pain, tingling, pressure, and related sensations. The first letdown results in the strongest sensations and the largest volume of milk (Isbister 1954; Prime et al. 2007; Geddes 2009). Gardner et al. (2015) showed that milk ejection occurs asynchronously, suggesting that the timing of myoepithelial cell response differs, resulting in heterogeneous emptying of the breast.

It's important to note how amazing, reciprocal, and layered this oxytocin process is. In addition to milk ejection, oxytocin also induces a state of calm in the mother and promotes bonding between the mother and her infant. And this warm emotional state, in turn, affects oxytocin levels. While oxytocin is triggered by a suckling infant, levels are also influenced by the mother's subjective feelings and sensations. Smelling, touching, seeing, or even thinking lovingly about her baby or hearing her baby's cry can stimulate oxytocin release. Conversely, oxytocin is inhibited by emotional or physical pain.

Cortisol is a steroid hormone produced in the adrenal glands. It is secreted in response to stress, which includes pain. High levels of cortisol can delay the production and secretion of milk—another way that a mother's psychological and pain states can affect nursing ability.

In addition to these systemic controls, milk production is also regulated locally. A whey protein called *Feedback Inhibitor of Lactation* (*FIL*), first in identified in goat's milk, inhibits milk secretion in a reversible, concentration-dependent manner. FIL is produced by lactocytes along with other constituents of milk, and it reversibly blocks secretion by those same lactocytes. (This is called autocrine control.) FIL also affects other aspects of lactation: It disrupts membrane trafficking, resulting in down-regulation of prolactin receptors, which then causes a decrease in epithelial cell differentiation into lactocytes (Peaker and Wilde 1996; Wilde et al. 1998).

In other words, removal of milk from the breast prevents collection of FIL, allowing milk production and secretion to continue. Thus, an empty breast produces milk faster than a full one. But if FIL is *not* removed—if the breast remains full—then the FIL concentration increases, preventing further milk secretion. The longer a breast is left full, the more FIL accumulates, inhibiting further milk production, thus helping mothers to avoid the harmful effects of engorged breasts, such as mastitis and plugged ducts. In this way, FIL fine tunes and regulates the volume of milk so the mother produces the amount of milk needed by her baby, without excess or shortfall (Daly et al. 1993). Interestingly, FIL controls milk production independently in each breast, so if a mother has difficulty nursing from one breast (due to injury, for example), she can continue to nurse from the other side without risk to either breast.

2.2.2 Lactogenesis

By mid pregnancy, breasts begin to differentiate into milk-producing glands by increasing alveolar cells, ductal proliferation, and myoepithelial cell hypertrophy. About 12 weeks before delivery, mammary tissues grow, and breast size and weight increase. For many women *colostrum*, or "first milk" production begins.

Colostrum is produced in small quantities, only about 20–40 ml/day, but it is very rich in nutrients. It contains the antibodies IgA, IgG, and IgM, white blood cells, and a higher concentration of minerals, protein, and vitamins than later milk. It is also low in carbohydrates and fat, and acts as a mild laxative, helping the neonate pass meconium. All these factors make colostrum ideal for the neonate's immature digestive system—it is all the nutrition an infant needs for the first few days.

By Day 3 after birth, progesterone levels are sufficiently low, so breasts begin making *transitional milk*. This is the onset of *lactogenesis II*. Compared with colostrum, transitional milk is lower in proteins and immunoglobulins, and higher in calories, fat, and lactose.

Initially, the volume of milk increases regardless of whether the mother is nursing. By Day 3 or 4, breasts produce around 300–400 ml/day of milk. By Day 5, most breasts produce about 500–800 ml/day of milk. While production increases, the amount actually secreted varies widely based on several factors, including maternal wellbeing and fatigue. Maternal stress and pain can cause increased levels of norepinephrine, dopamine, and cortisol, which inhibit the synthesis of prolactin and decrease overall milk supply.

By about 10–14 days after birth, the breast produces *mature breast milk*. Mature breast milk is often delineated into foremilk and hindmilk, but this delineation is misleading. Breasts only make one type of milk. However, when milk is produced, the fatty component sticks to the walls of the alveoli, while the liquid component travels down the ducts and mixes with any leftover milk from the previous feeding. When the infant begins a feeding, he first encounters the watery *foremilk*, which has a higher concentration of lactose and less fat (which was left behind in the alveoli). As the infant continues to nurse, the milk ejection reflex is triggered, and the fattier hindmilk is squeezed out of the alveoli into the ducts.

Therefore, the composition and amount of milk an infant transfers from the breast depends on the volume of milk mom produces, how long the baby nurses on each breast, and the amount of time between feedings. For example, if the mother produces a large volume of milk and the infant nurses for a short time, he may fill up on mostly foremilk before getting to the hindmilk. This can result in increased gas, colic, and lower weight gain.

Regardless of how healthy or unhealthy a mother's diet is, mature breast milk is generally made of the following breakdown of nutrients (Ballard and Morrow 2013):

2.2.2.1 Kcalories

60–75 kcal/100 ml

2.2.2.2 Water

90 g/100 ml water

2.2.2.3 Protein

0.8–1.3 g/100 ml of protein, which includes 30 % casein and 70 % whey proteins. Whey proteins include alpha lactalbumin, lactoferrin, lysozyme, serum albumin, secretory immunoglobulin

IgA (sIgA), insulin, epidermal growth factor (EGF), and many enzymes. These proteins serve many functions, such as host defense, nutrition, and enzymatic activity. Specifically oligosaccharides protect the gut from bacterial attachment; immunoglobulins, primarily sIgA, protect the gut lining from infection; and whey proteins kill bacteria, viruses, and fungi.

Casein has low solubility and can turn into clots or curds. Whey proteins are soluble and remain in liquid form, making them more easily digestible. Human breast milk is lower in casein than most animal milks, such as cow's milk, which has 18 % whey and 82 % casein. Excessive casein can overwhelm the infant's maturing kidneys. Lactoferrin, sIgA, lysozyme, and alpha lactalbumin are found only in human milk. Human milk does not contain beta-lactoglobulin, found in cow's milk, which infants often cannot digest. For all these differences in composition, a cow's milk-based formula is a common cause of infant intolerance.

2.2.2.4 Carbohydrate

6.9–7.4 g/100 ml of carbohydrates, which is 90–95 % lactose. After colostrum transitions to mature milk, the lactose concentration remains constant. It is mainly used as an energy source, but a small amount is not absorbed and promotes softer stools. It also contains 1 g/100 ml of 100 or more oligosaccharides. These are carbohydrate polymers that control intestinal flora by promoting certain bacterial strains to grow in the gut, therefore protecting against infection.

2.2.2.5 Fat

3–5 g/100 ml of fat, mostly in the form of palmitic and oleic acids. Fats provide 50 % of the calories in human milk and therefore are a major source of energy. The fat content of milk is variable throughout the day and increases over the course of breastfeeding. It is present mostly in the alveolar sacs and distal ducts, so the infant must first drain the "foremilk" to get to this fat rich "hindmilk." Lipids are present in fat globules and many essential fatty acids in the form of triglycerides. Certain fatty acids are of interest—DHA (docosahexaeonic acid) and ARA (arachnidonic acid)—because they are found only in human milk. They are important in neurological and visual development of the infant.

2.2.2.6 Vitamins and Minerals

It contains 0.2 g/100 ml in total, including:

- 25 mg/100 ml calcium,
- 9 mg/100 ml phosphorus
- 200 mg/100 ml sodium
- 3.5 mg/100 ml magnesium
- 5 mg/100 ml vitamin C
- smaller amounts of iron, copper, zinc, vitamin A, pantothenic acid, nicotinic acid, iodine
- Trace amounts of vitamins K, D, E, and B

There is a high concentration of zinc initially, but it declines over the first 6 months of nursing. Also, there is a low concentration of iron. Infants are born with sufficient iron stores at birth, but they decline over the first 6 months of life. Similarly, vitamin D is present in low concentrations in human milk and is not easily supplemented to the mother. Current recommendations from the American Academy of Pediatrics (Perrine et al. 2010) include vitamin D supplementation for infants who are exclusively breastfed for the first 6 months of life. Solid foods are introduced at 6 months, resolving the need for iron and zinc supplementation.

2.2.2.7 Cellular Elements

Breast milk contains maternal cells (such as leukocytes) and epithelial cells, such as stem cells, lactocytes, myoepithelial cells, and progenitor cells. Neutrophils are present in greater concentrations in colostrum. They decrease in mature milk and are replaced by macrophages and lymphocytes to address the changing needs of the infant immune system (Hassiotou et al. 2013).

2.2.3 Galactopoesis (Maintenance of Lactation)

Very quickly after the baby begins nursing (around Day 2 or 3), milk supply transitions from endocrine to autocrine control. Therefore, production changes from being mostly under global hormonal control (via oxytocin and prolactin) to functioning mostly by supply and demand (with the help of FIL). Continual removal of milk from the breasts maintains the supply. More detailed explanation of maintenance of lactation is discussed in Chapter 4.

With so many factors in play, the timing, quality, and frequency of nursing in the first few weeks are critical to establishing a successful nursing pattern.

References

AAP: American Academy of Pediatrics: Vitamin D supplementation for infants. 2010. https://www.aap.org/en-us/about-the-aap/aap-press-room/pages/Vitamin-D-Supplementation-for-Infants.aspx. Accessed 21 Mar 2015.

Ballard O, Morrow AL. Human milk composition and bioactive factors. Pediatr Clin North Am. 2013;60(1):49–74.

Berry CA, Thomas EC, Piper KME, Cregan MD. The histology and cytology of the human mammary gland and breastmilk. In: Hartmann PE, Hale TW, editors. Textbook of human lactation. Amarillo: Hale; 2007. p. 43–54.

Bissell MJ, Weaver VM, Lelievre SA, Wang F, Petersen OW, Schmeichel KL. Tissue structure, nuclear organization, and gene expression in normal and malignant breast. Cancer Res. 1999;59: 1757–64.

Burch D. Digging up the dead: uncovering the life and times of an extraordinary surgeon. London: Vintage; 2010.

Christensen K, Nielsen MO, Bauer R, Hilden K. Evaluation of mammary blood flow measurements in lactating goats using the ultrasound Doppler principle. Comp Biochem Physiol A Comp Physiol. 1989;92:385–92.

Cooper AP. On the anatomy of the breast. 1840. http://jdc.jefferson.edu/cooper/. Accessed 21 Mar 2015.

Courtiss EH, Goldwyn RM. Breast sensation before and after plastic surgery. Plast Reconstr Surg. 1976;58(1):1–13.

Cowie AT. Overview of the mammary gland. J Invest Dermatol. 1974;63:2–9.

Cox DB, Owens RA, Hartmann PE. Blood and milk prolactin and the rate of milk synthesis in women. Exp Physiol. 1996;81:1007–20.

Cox DB, Kent JC, Casey TM, Owens RA, Hartmann PE. Breast growth and the urinary excretion of lactose during human pregnancy and early lactation: endocrine relationships. Exp Physiol. 1999;84(2):421–34.

Cregan MD, Mitoulas LR, Hartmann PE. Milk prolactin, feed volume, and duration between feeds in women breastfeeding their full-term infants over a 24-hour period. Exp Physiol. 2002;87: 207–14. doi:10.1113/eph8702327.

Czank C. Hormonal control of the lactation cycle. In: Hale TW, Hartmann PE, editors. Textbook of human lactation. Amarillo: Hale; 2007.

Daly SEJ, Owens RA, Hartmann PE. The short-term synthesis and infant-regulated removal of milk in lactating women. Exp Physiol. 1993;78:209–20.

Doucet S, Soussignan R, Sagot P, Schaal B. The secretion of areolar (montgomery's) glands from lactating women elicits selective, unconditional responses in neonates. PLoS One. 2009;4(10):e7579.

Drewett RF, Bowen-Jones A, Dogterom J. Oxytocin levels during breast-feeding in established lactation. Horm Behav. 1982;16: 245–8.

Gardner H, Kent JC, Hartmann PE, Geddes DT. Asynchronous milk ejection in human lactating breast: case series. J Hum Lact. 2015. doi:10.1177/0890334414568120.

Geddes DT. The use of ultrasound to identify milk ejection in women – tips and pitfalls. Int Breastfeed J. 2009;4:5. doi:10.1186/ 1746-4358-4-5.

Hartmann PE. The breast and breastfeeding. In: Philipp EE, Setchell M, Ginsburg J, editors. Scientific foundations of obstetrics and gynaecology. 4th ed. Oxford: Butterworth Heinemann; 1991.

Hassiotou F, Geddes D. Anatomy of the human mammary gland: current status of knowledge. Clin Anat. 2013;26(1):29–48.

Hassiotou F, Geddes DT, Hartmann PE. Cells in human milk: state of the science. J Hum Lact. 2013;29(2):171–82. doi:10.1177/0890334413477242.

Isbister C. A clinical study of the draught reflex in human lactation. Arch Dis Child. 1954;29(143):66–72.

Jacobs LS. The role of prolactin in mammogenesis and lactogenesis. Adv Exp Med Biol. 1977;80:173–91.

Kent JC, Mitoulas LR, Cregan MD, Geddes DT, Larsson M, Doherty DA, Hartmann PE. Importance of vacuum for breast milk expression. Breastfeed Med. 2008;3(1):11–9.

Linzell JL. Mammary-gland blood flow and oxygen, glucose and volatile fatty acid uptake in the conscious goat. J Physiol. 1960; 153:492–509.

Macfarlane AJ. Olfaction in the development of social preferences in human neonate. Ciba Found Symp. 1975;33:103–17.

Makin JW, Porter RH. Attractiveness of lactating female's breast odors to neonates. Child Dev. 1989;60:803–10.

McNeilly AS, Robinson ICAF, Houston MJ, Howie PW. Release of oxytocin and prolactin in response to suckling. Br Med J. 1983; 286:257–9.

Moffat DF, Going JJ. Three dimensional anatomy of complete duct systems in human breast: pathological and developmental implications. J Clin Pathol. 1996;49(1):48–52.

Neville MC, McFadden TB, Forsyth I. Hormonal regulation of mammary differentiation and milk secretion. J Mammary Gland Biol Neoplasia. 2002;7(1):49–66.

Pang WW, Hartmann PE. Initiation of human lactation: secretory differentiation and secretory activity. J Mammary Gland Biol Neoplasia. 2007;12(4):211–21.

Peaker M, Wilde CJ. Feedback control of milk secretion from milk. J Mammary Gland Biol Neoplasia. 1996;1(3):307–15.

Perrine CG, Sharma AJ, Jefferds MED, Serdula MK, Scanlon KS. Adherence to vitamin D recommendations among US infants. Pediatrics. 2010;125(4):627–32.

Prime DK, Geddes DT, Hartmann PE. Oxytocin: milk ejection and maternal-infant well-being. In: Hale T, Hartmann PE, editors. Textbook of human lactation. 1st ed. Amarillo: Hale; 2007. p. 141–58.

Ramsay DT, Mitoulas LR, Kent JC, Larsson M, Hartmann PE. The use of ultrasound to characterize milk ejection in women using an electric breast pump. J Hum Lact. 2005;21(4):421–8.

Russell MJ. Human olfactory communication. Nature. 1976;260: 520–2.

Sanches MT. Clinical management of oral disorders in breastfeeding. J Pediatr (Rio J). 2004;80(5 Suppl):S155–62.

Schaal B, Montagner H, Hertling E, Bolzoni D, Moyse R, et al. Olfactory stimulations in mother-infant relationships. Reprod Nutr Dev. 1980;20:843–58.

Soussignan R, Schaal B, Marlier L, Jiang T. Facial and autonomic responses to biological and artificial olfactory stimuli in human neonates: re-examining early hedonic discrimination of odors. Physiol Behav. 1997;62:745–58.

Sternlicht MD. Key stages in mammary gland development: the cues that regulate ductal branching morphogenesis. Breast Cancer Res. 2006;8(1):201.

Sullivan RM, Toubas P. Clinical usefulness of maternal odor in newborns: soothing and feeling preparatory responses. Biol Neonate. 1998;74:402–8.

Suzuki R, Atherton AJ, O'Hare MJ, Entwistle A, Lakhani SR, Clarke C. Proliferation and differentiation in the human breast during pregnancy. Differentiation. 2000;66:106–15.

Uvnas-Moberg K, Widstrom AM, Nissen E, Björvell H. Personality traits in women 4 days post partum and their correlation with plasma levels of oxytocin and prolactin. J Psychosom Obstet Gynaecol. 1990;11:261–73.

Varendi H, Porter RH. Breast odour as the only maternal stimulus elicits crawling towards the odour source. Acta Paediatr. 2001; 90:372–5.

Watson CJ, Khaled WT. Mammary development in the embryo and adult: a journey of morphogenesis and commitment. Development. 2008;135(6):995–1003.

Wilde CJ, Addey CV, Bryson JM, Finch LM, Knight CH, Peaker M. Autocrine regulation of milk secretion. Biochem Soc Symp. 1998;63:81–90.

Wiseman BS, Werb Z. Stromal effects on mammary gland development and breast cancer. Science. 2002;296(5570):1046–9.

Young 3rd WS, Shepard E, Amico J, Hennighausen L, Wagner KU, LaMarca ME, McKinney C, Ginns EI. Deficiency in mouse oxytocin prevents milk ejection, but not fertility or parturition. J Neuroendocrinol. 1996;8(11):847–53.

http://www.springer.com/978-3-319-18193-6

Clinician's Guide to Breastfeeding
Evidenced-based Evaluation and Management
Dahl, L.
2015, XVI, 168 p. 13 illus. in color., Softcover
ISBN: 978-3-319-18193-6