WHO/FCH/CAH/00.13
ORIGINAL: ENGLISH
DISTR: GENERAL

Mastitis

Causes and Management

DEPARTMENT OF CHILD AND ADOLESCENT
HEALTH AND DEVELOPMENT

World Health Organization
Geneva
2000

Cover illustration adapted from a poster by permission
of the Ministry of Health, Peru.

Contents

Acknowledgements

The authors of this review were Ms Sally Inch and Dr Severin von Xylander, with editorial assistance from Dr Felicity Savage.

Many thanks are due to the following lactation experts for reviewing the document in draft and for providing helpful constructive criticism:

Dr Lisa Amir (Australia), Ms Genevieve Becker (Eire), Ms Chloe Fisher (UK), Dr Arun Gupta (India), Dr Rukhsana Haider (Bangladesh), Ms Joy Heads (Australia), Dr Evelyn Jain (Canada), Dr Miriam Labbock (USA), Ms Sandra Lang (UK), Dr Verity Livingstone (Canada), Dr Gro Nylander (Norway), Dr Marina Rea (Brazil), Ms Janice Riordan (USA), Dr Anders Thomsen (Denmark), Ms Marsha Walker (USA) and Dr Michael Woolridge (UK).

Ms Helen Armstrong (UNICEF) also reviewed the draft document and provided many helpful suggestions.

Thanks also to members of WHO's Technical Working Group on Breastfeeding for helpfully reviewing the manuscript: Dr Jose Martines, Ms Randa Saadeh, Dr Constanza Vallenas and Dr Jelka Zupan.

Mastitis:
Causes and Management

1. Introduction

Mastitis is an inflammatory condition of the breast, which may or may not be accompanied by infection. It is usually associated with lactation, so it is also called *lactational mastitis* (67) or *puerperal mastitis* (1). It can occasionally be fatal if inadequately treated. Breast abscess, a localised collection of pus within the breast, is a severe complication of mastitis. These conditions form a considerable burden of disease and involve substantial costs (43; 112). Recent research suggests that mastitis may increase the risk of transmission of HIV through breastfeeding (76; 150).

Awareness is growing that inefficient removal of milk resulting from poor breastfeeding technique is an important underlying cause, but mastitis remains synonymous with breast infection in the minds of many health professionals (11; 15; 93; 94). They are often unable to help a woman with the condition to continue to breastfeed, and they may advise her unnecessarily to stop (43).

This review aims to bring together available information on lactational mastitis and related conditions and their causes, to guide practical management, including the maintenance of breastfeeding.

2. Epidemiology

2.1 Incidence

Mastitis and breast abscess occur in all populations, whether or not breastfeeding is the norm. The reported incidence varies from a few to 33% of lactating women, but is usually under 10% (Table 1). Most studies have major methodological limitations, and there are no large prospective cohort studies. The higher rates are from selected populations.

The incidence of breast abscess also varies widely, and most estimates are from retrospective studies of patients with mastitis (Table 2). However, according to some reports, especially from developing countries, an abscess may also occur without apparent preceding mastitis.

2.2 Time of occurrence

Mastitis is commonest in the second and third week postpartum (29; 120; 122), with most reports indicating that 74% to 95% of cases occur in the first 12 weeks (49; 122; 140; 167; 170). However, it may occur at any stage of lactation, including in the second year (7; 140). Breast abscess also is commonest in the first 6 weeks post partum, but may occur later (18; 32; 43; 49; 71; 74; 109; 119; 157).

Table 1: Estimates of Incidence of Mastitis

Authors:	Year:	Country:	Method:	Number of cases:	Case definition	Observation period post-partum:	Population size:	Percentage of mothers breastfeeding at the time of assessment:	Percentage with mastitis:	Comments:
Fulton (49)	1945	UK	Population based prospective study	156	Definite evidence of suppuration	2 years and 4 months	41,000 1500 births	not indicated	9.33%	
Waller (168)	1946	UK	Retrospective questionnaire among post partum visit patients	3	Women reporting having had mastitis	0 – 4 weeks	52	42%	5.7%	
Hesseltine (62)	1948	USA	Prospective study in one hospital	121	not indicated	6 months	1,730	100%*	7%	* selection criterion
Marshall (100)	1975	USA	Prospective study on women delivering in one hospital	65	Actual or suspected breast "infection"	up to one year	5,155	49%	2.67%	* included only women returning to the same hospital
Prentice et al. (131)	1985	Gambia	Analysis of cases in a defined population	65	Diagnosis by a health professional	day 14 to cessation of breastfeeding	not indicated	100% (presumed)	2.6%*	* Mean monthly incidence
Hughes et al. (67)	1989	UK	Retrospective analysis of medical records	not indicated	Diagnosis of puerperal breast infection	not indicated	425	not indicated	4 - 10%*	* Annual incidence 1930 – 1988
Riordan & Nichols (140)	1990	USA based	Postal retrospective questionnaire among a breastfeeding support organisation*	60	Women reporting having had mastitis	the entire breastfeeding period for each child	180	100%	33%	* Non-representative population
Amir (6)	1991	Australia	self-report questionnaire in a breastfeeding clinic and health centres*	49	not indicated	week 1 to 2 years	98	100 %	50%	* Non-representative population
Kaufmann & Foxman (81)	1991	USA	Retrospective analysis of medical records	30	Physician's diagnosis	0 – 7 weeks	966	85%	2.9%	

Table 1 (cont.): Estimates of incidence of Mastitis

Authors:	Year:	Country:	Method:	Number of cases:	Case definition	Observation period post-partum:	Population size:	Percentage of mothers breastfeeding at the time of assessment:	Percentage with mastitis:	Comments:
Jonsson & Pulkkinen (78)	1994	Finland	Questionnaire distributed in a clinic, diagnosis made by health worker	199	Based on clinical presentation	5 – 12 weeks	670	85%	24%*	* Methodology not clear
Foxman (48)	1994	USA	Self-administered questionnaire distributed at discharge after birth.	9	Women reporting treatment for mastitis	0 – 9 days	100	100%*	9%	*Recruitment condition
Evans (43)	1995	Australia	Data collection from health facilities of mastitis cases in a defined area	402	Clinical signs of inflammation confirmed by a nurse	0 - 7* months	8175	50%**	4.92%	* upper limit not indicated **presumed form other studies
Nicholson and Yuen (118)	1995	Australia	Retrospective. Telephone contact at 3 months	not specified	Mothers reporting mastitis	3 months	735	54%	7.7%	
Inch (72)	1996	UK	Computerised analysis of medical records of primary birth visit of 4 general practitioners in Oxford	4	Medical diagnosis	at week 2	33	37%	12%*	*Average incidence 9%
				3			36	58%	8%*	
				1			54	81%	2%*	
				8			52	56%	15%*	
Kinlay (83)	1998	Australia	Prospective cohort study following up women who delivered in two obstetric service of one geographic area	219	Defined self-reported clinical diagnosis	0 – 6 months	1075	100%*	20%	*Recruitment condition

Table 2: Estimates of Incidence of Breast Abscess

Authors:	Year:	Country:	Incidence:	Comments:
Waller (169)	1938	UK	6.7 %	
Fulton (49)	1945	UK	8.9 %	
Waller (168)	1946	UK	1.9%	
Jeffrey (75)	1947	UK	4 % *	* of women with mastitis
Leary (95)	1948	USA	0.04%	
Newton & Newton (117)	1950	USA	0.47*	* Before the use of penicillin the incidence was 0.82%.
Devereux (32)	1970	USA	11 % *	* of women with mastitis
Marshall (100)	1975	USA	4.6 % *	* of women with mastitis
Niebyl et al. (119)	1978	USA	11.5 %	* of women with mastitis
Thomsen (161)	1984	Denmark	11 % *	* among breastfeeding women with breast inflammation
Cairns (19)	1996	Zambia	0.5 % *	* calculated on the average annual birth rate in the area served by one hospital

3. Causes of mastitis

The two principle causes of mastitis are *milk stasis* and *infection.* Milk stasis is usually the primary cause (67; 120), which may or may not be accompanied by or progress to infection.

Gunther in 1958 (55) recognised from clinical observation that mastitis resulted from stagnation of milk within the breast, and that the efficient removal of milk as it is formed could largely prevent the condition. She suggested that infection when it occurred was not primary, but resulted from the stagnant milk providing a medium for bacterial growth.

Thomsen and co-authors in 1984 (161) produced additional evidence for the importance of milk stasis. They counted leukocytes and bacteria in milk from breasts with clinical signs of mastitis, and proposed the following classification:

- milk stasis
- non-infectious inflammation (or non-infectious mastitis)
- infectious mastitis.

	Leukocytes < 10^6/ml milk	**Leukocytes > 10^6/ml milk**
Bacteria < 10^3/ml milk	Milk stasis	Non-infectious mastitis
Bacteria > 10^3/ml milk		Infectious mastitis

In a randomised study, they found that *milk stasis* (<10^6 leukocytes and <10^3 bacteria) improved with continued breastfeeding alone; *non-infectious mastitis* (>10^6 leukocytes and <10^3 bacteria) required treatment by additional expression of milk after a feed, and *infectious mastitis* (>10^6 leukocytes and >10^3 bacteria) was treated effectively only with both removal of milk and systemic antibiotics. Without effective removal of milk, non-infectious mastitis was likely to progress to infectious mastitis, and infectious mastitis to the formation of an abscess.

Thomsen at al also related cell and bacterial counts to clinical findings, and found that it was impossible to be certain from clinical signs whether or not infection was present.

4. Milk Stasis

Milk stasis occurs when milk is not removed from the breast efficiently. This may occur when the breasts are engorged soon after delivery, or at any time when the infant does not remove the milk that is produced from part or all of the breast. Causes include poor attachment of the infant at the breast, ineffective suckling, restriction of the frequency or duration of feeds, and blockage of milk ducts. Other situations which predispose to milk

stasis include an overabundant milk supply (107), or lactating for twins or higher multiples (118).

4.1 Breast engorgement

Observations on the connection between breast engorgement (Section 7.1) and mastitis have been made for many years, though the two conditions were not always clearly distinguished.

Historically, "milk fever", characterised by distended breasts and a high fever, was described in the 18th century. It occurred on about the third day after delivery, when the milk "came in", and it may have been the result of progression from engorgement (45). James Nelson in 1753 noted that the condition did not occur when infants were put to the breast immediately after delivery, so that milk stasis was avoided (115). The primary importance of prompt removal of milk in the early stages of mastitis, or congestion, to prevent progression of the disease and abscess formation, was also described by Naish in 1948 (114). She regarded suckling by the baby as the most effective means of milk removal.

4.2 Frequency of breastfeeds

In 1952, Illingworth and Stone formally demonstrated in a controlled trial that the incidence of engorgement was halved if babies were given unrestricted access to the breast (69). Over the years, a number of others also observed that when breastfeeding times were scheduled, engorgement, often followed by mastitis and lactation failure, was more common (14; 73; 168).

An association between restricted frequency or duration of breastfeeding and mastitis has subsequently been described by a number of authors (5; 8; 44; 49; 94; 100; 110; 139; 140). Many women have the experience that if they miss a breastfeed, or if their infant suddenly starts to sleep through the night and the time between feeds increases, mastitis may follow (94; 139).

4.3 Attachment at the breast

The importance of a baby being well attached to the breast for effective removal of milk was first recognised by Gunther (55). The process has been researched and further described subsequently (176) and clinical techniques are now well developed and described by other authors (42). Poor attachment as a cause of inefficient milk removal is now seen as a major predisposing factor for mastitis (5; 44). The technique for ensuring good attachment is described in Annex 1, and in WHO/UNICEF training materials (178).

Fissured or painful nipples are often found in association with mastitis. The commonest cause of nipple pain and trauma is poor attachment at the breast (177), so the two conditions may occur together partly because they have the same mechanical origin. Also, nipple pain may

lead to avoidance of feeding on the affected breast and thus predispose to milk stasis and engorgement (44).

4.4 Preferred side and efficient suckling

Further evidence that an underlying cause of mastitis is milk stasis of mechanical origin comes from studies of which breast is affected most often, (43; 71; 140). It has been observed that many mothers find it easier to attach their infant to the breast on one side than on the other, and it was suggested that poor attachment, leading to milk stasis and mastitis might be more likely to occur on the side that was more difficult. It was assumed that this would be related to a mother's right or left-handedness. However, although several studies of the frequency with which each side is affected have been conducted, no consistent or significant difference has been observed. 37-52% of cases involve the right breast and 38-57% the left breast, with bilateral mastitis in 3-12%. The findings for breast abscess are similar (117).

Inch and Fisher (71) however noticed that women's preferred side for holding their baby was not necessarily related to their handedness. They recorded both the dominant hand and the preferred side for holding the baby for every woman who developed mastitis. No relationship was found between the dominant hand and the side affected, but in 78% of cases mastitis occurred in the breast opposite to the preferred side. This supports the contention that the underlying cause of the condition is likely to be mechanical.

4.5 Other mechanical factors

A short frenulum (tongue-tie) in the infant has been observed clinically to interfere with attachment at the breast, and cause sore and fissured nipple. It may also reduce the efficiency of milk removal, and predispose to mastitis (99; 101).

Use of a pacifier or a bottle and teat in a maternity hospital in Italy, was found to be associated with sore nipples at discharge (21). Pacifier use may also be associated with poor attachment at the breast, engorgement (138), and reduction in breastfeeding frequency and duration (164). Pacifiers may thus interfere with milk removal and predispose to milk stasis.

Tight clothing (26; 31; 44; 64; 74; 86; 92; 113) and *prone sleeping position* (5) are other mechanical factors that have been observed in connection with mastitis, and suggested as possible causes, though the evidence is mainly anecdotal.

5. Infection

5.1. Infecting organisms

The commonest organisms found in mastitis and breast abscess are coagulase-positive *Staphylococcus aureus* and *Staph. albus* (74; 100; 102; 103; 117; 119; 120; 140; 170). *Escherichia coli* (94; 161) and *Streptococcus* (α-, β-, and non- haemolytic) are sometimes found, (82; 119; 120; 137; 147; 161) and the latter has in a few cases been linked to neonatal streptococcal infection (82). Rarely, mastitis has been recognised as a complication of typhoid fever and other salmonella infections (51; 146). *M. tuberculosis* is another rare cause of mastitis. In populations where tuberculosis is endemic, *M. tuberculosis* may be found in about 1% of cases of mastitis (56) and associated in some cases with tuberculous tonsillitis in the infant. Candida and cryptococcus have been reported to cause fungal mastitis (60; 123; 165) but mycoplasma and chlamydia have not (162).

Bacteria are often found in milk from asymptomatic breasts, in both industrialised (144) and developing (184) countries. The spectrum of bacteria is often very similar to that found on skin (74; 100; 119; 170). Marshall (100), for example, found *Staph. epidermidis,* diphtheroids, and alpha-haemolytic and non-haemolytic streptococci. Bacteriological studies are therefore complicated by the difficulty of avoiding contamination from skin bacteria (160). Despite careful techniques for collection, only 50% of milk cultures may be sterile (109), while others show "normal" colony counts from 0 to 2,500 colonies per ml. (183).

Thus the presence of bacteria in the milk does not necessarily indicate infection, even if they are not contaminants from the skin. One way to distinguish between infection and simple bacterial colonisation of the milk ducts is to look for bacteria coated with specific antibodies. As with urinary tract infection, bacteria in breastmilk which are coated by immunoglobulins IgA and IgG demonstrate that a specific immune reaction to an infection has taken place (158; 160). However, facilities for such an investigation are not routinely available in many situations.

5.2 Bacterial colonisation of the infant and breast

Bacterial colonisation of the infant and breast is a normal process that takes place soon after birth. Both the mother's milk ducts and the infant's nasopharynx are colonised by a variety of organisms, some of them potentially pathogenic, such as *Staph. aureus* (38). However, their presence does not by itself cause mastitis (38; 102; 183).

If a mother is in close contact with her infant immediately after delivery, she transfers to the infant her own strain of respiratory and skin organisms. These organisms grow and populate her infant's gut, skin and respiratory tract. Once a flora of commensal organisms is established, the growth of pathogenic bacteria is inhibited. This process, known as *bacterial interference*, has been used deliberately in clinical settings to prevent and control outbreaks of infection by the more virulent forms of *Staph. aureus* (96; 151).

Thus in addition to facilitating breastfeeding and bonding, early skin-to-skin contact of a mother with her infant, and rooming-in, are the most natural and efficient ways to prevent the spread of infection, including the spread of organisms responsible for mastitis. It has long been recognised that infants who are kept with their mothers have lower rates of infection than those who are kept in a nursery (108). Colbeck in 1949 suggested that the most important single factor in the spread of infection was the number of infants per nursery. He even stated that "ideally, it would appear that the baby should remain with the mother" (24).

5.3 Epidemic puerperal mastitis

Problems may arise when first the infant and then the mother is exposed to unfamiliar or virulent organisms. This is most likely to occur in hospitals, from cross-infection or the development of resistant strains (38; 49; 109). An epidemic form of puerperal mastitis occurred frequently in hospital nurseries in industrialised countries from the 1930's to the 1960's (52; 74; 185). During this period, hospital deliveries became more frequent, breastfeeding was not promoted, and the antibiotic era was only just beginning. The dominant role of staphylococcal infections and transmission between nursery personnel, infants and mothers was repeatedly demonstrated (1; 24; 37; 109; 127; 135).

Epidemic mastitis has been regarded as a hospital acquired disease caused by highly virulent strains of penicillin-resistant *Staph. aureus* (52). It has become rarer since the advent of antibiotics and the use of more powerful bactericides for cleaning hospitals (105); but it has also become rarer since practices which favoured milk stasis have become less widespread, such as restrictive feeding schedules, and interruption of feeding from a breast with a fissured nipple; and since infants in hospital have roomed-in with their mothers instead of staying in nurseries.

5.4 Route of infection

How infection enters a breast is uncertain. Several routes have been suggested: through the lactiferous ducts into a lobe; by haematogenous spread; and through a nipple fissure into the periductal lymphatic system (18; 39; 52; 94; 137; 170). Nipple fissure has been reported with increased frequency in the presence of mastitis (41; 43; 71; 78; 100; 170). In a prospective, randomised clinical trial, Livingstone studied the effects of antibiotic treatment of women with nipple fissure from which *Staph. aureus* was cultured. She found that women who were treated with a systemic antibiotic were 4-5 times less likely to develop mastitis than women who were treated with a topical preparation, or with improved breastfeeding technique alone (97). So, in addition to the possibility that mastitis and fissure occur together because both can result from an infant's poor attachment at the breast, there is also the likelihood that a fissure provides an entry point for infection(101).

An association between *Candida* infection of the nipple and mastitis, particularly recurrent mastitis, has often been noted (6; 60; 94; 60). Mastitis due to *Candida* has occasionally been reported, particularly in diabetic women, but is extremely rare (123). It is more likely that nipple fissure resulting from candidiasis could provide an entry point for bacterial infection. It is also

possible that, when nipples are painful and damaged as a result of candida, a woman may use the breast less efficiently, leading to milk stasis. However, candidiasis often follows antibiotic treatment, and it may occur as an indirect consequence of mastitis rather than being a predisposing factor.

Deep burning breast pain occuring during and after feeds is often attributed to candida infection of the mammary ducts, but recently *Staph. aureus* has been identified as a pathogen in cases of deep pain as well as of nipple fissure (97; 159). Deep pain could be due to infection in the mammary ducts, but whether there is any connection with mastitis is unclear.

Many lactating women who have potentially pathogenic bacteria on their skin or in their milk do not develop mastitis.

But:

Many women who do develop mastitis do not have pathogenic organisms in their milk.

6. Predisposing factors

There are a number of factors which it has been suggested might increase the risk of mastitis. Evidence exists for some, but most remain anecdotal. Their importance is likely to be small compared with that of breastfeeding technique: good attachment and effective milk removal.

- *Age*
  One retrospective study showed that women aged 21-35 were more likely to develop mastitis than those under 21 and over 35 (78). Another retrospective study identified women aged 30-34 as having the highest incidence of mastitis, even when parity and full-time employment were controlled for (81).

- *Parity*
  Primiparity is found to be a risk factor in some studies (43; 49; 74; 109) but not in others (48; 81).

- *Previous attack*
  There is substantial evidence that a first attack of mastitis predisposes to recurrence (32; 43; 44; 48; 78; 109). In some studies 40 to 54 percent of women had suffered one or more previous attacks. This could be a result of uncorrected poor breastfeeding technique.

- *Delivery*
  Complications of delivery may increase the risk of mastitis (109), though the use of oxytocin does not do so (78).

- *Nutrition*
  Nutritional factors have often been thought to predispose to mastitis, including high salt intake, high fat intake, and anaemia, but the evidence is inconclusive (5; 31; 105; 106; 171). Poor nutrition has also been suggested, particularly poor micro-nutrient status. Anti-oxidants vitamin E, vitamin A, and selenium are known to reduce the risk of mastitis in dairy animals (149; 150). A micro-nutrient supplementation trial in Tanzania found that vitamin-E rich sunflower oil reduced signs of breast inflammation, although vitamin A from red palm oil did not (46).

- *Immune factors in milk*
  Immune factors in breastmilk may provide a defence mechanism in the breast. A study in the Gambia suggested that when levels of these factors are low, effective defence may be reduced, and the risk of recurrent mastitis increased (131).

- *Stress and fatigue*
  Maternal stress and fatigue have often been linked to mastitis but again there is little firm evidence (8; 31; 44; 94; 117; 140). Women who have pain and fever are likely to feel tired and want to rest, but it is not clear whether or not fatigue is a cause of the condition.

- *Employment outside the home*
  In a retrospective study in 1991 by Kaufmann and Foxman (81), full-time work outside the home was found to be associated with an increased incidence of mastitis. The suggested explanation was milk stasis caused by long intervals between the breastfeeds and lack of time for adequate milk expression

- *Local factors in the breast*
  Factors such as skin type, skin reaction to the sun, allergy, rashes, and exposure to cold have not been shown to affect the incidence of mastitis. Whether some procedures such as the use of nipple cream are able to prevent mastitis remains speculative (21; 44; 78). There is no evidence to support the suggestion that the size of the breast increases the risk of mastitis.

- *Trauma*
  Trauma to the breasts from any cause can damage gland tissue and ducts and this could lead to mastitis. A possible cause that should not be overlooked is domestic violence, which affects many women in all societies, and is likely to occur during lactation (179).

7. Pathology and clinical features

7.1 Engorgement

From the 3rd to the 6th day after delivery, when the milk normally "comes in", the breasts may be very full. This is physiological, and with effective suckling and removal of milk by the infant, rapidly resolves. However it may develop into engorgement, and the two conditions are often confused.

With *engorgement*, the breast is overfilled with both milk and tissue fluid. Venous and lymphatic drainage are obstructed, milk flow is hindered, and the pressure in the milk ducts and alveoli rises. The breasts become swollen and oedematous.

With both physiological fullness and engorgement, the whole of both breasts is usually affected. There are important differences however (178):

- a *full* breast feels hot, heavy and hard. There is no shininess, oedema, or redness. The milk usually flows well, and sometimes drips out spontaneously. It is easy for the infant to suckle and remove the milk
- an *engorged* breast is enlarged, swollen and painful. It may be shiny and oedematous with diffuse red areas. The nipple may be stretched flat. The milk often does not flow easily, and it may be difficult for the infant to attach to the breast to suckle until the swelling is reduced. The woman sometimes has fever. However, the fever usually settles in 24 hours.

7.2 Blocked duct

Localised milk stasis, affecting part of the breast such as a lobe, is often referred to as *blocked duct.* "Focal breast engorgement", "caked breast" or "plugged duct" are other terms which are sometimes used (91). The condition is assumed to be due to a solid obstruction, but may simply be due to inefficient removal of milk from that part of the breast.

Clinical signs are a painful lump in one breast, often with a patch of redness of the overlying skin. Only part of one breast is affected. The woman usually has no fever and feels well. Some women with blocked duct report the presence of particulate matter in their expressed breastmilk. In this case there may be true obstruction to a milk duct (86; 105; 119; 140). Symptoms are rapidly relieved when the hard particulate material is expressed, and milk is released from the affected part of the breast. White granules which may be found in accumulated milk are thought to be formed from a mixture of casein and other materials hardened by salts containing calcium (16; 27). Fatty- or stringy-looking material, sometimes brown or greenish, is also sometimes extruded from apparently blocked ducts, followed by relief of symptoms (53; 91; 152).

A related condition is the appearance of a *white spot* at the end of the nipple, usually about 1 mm in diameter, and which is associated with blocked duct (3; 17; 98). The white spot can be extremely painful during suckling. The obstruction is quickly relieved when the white spot is removed for example by use of a sterile needle or rubbing with a towel. The white spot is

thought to be due to an overgrowth of epithelium (forming a "blister"), or an accumulation of particulate or fatty material.

Another uncommon related condition is *galactocoele* (94). A galactoceole is a milk filled cyst, thought to develop from a blocked duct. It presents as a smooth rounded swelling in the breast, at first filled with pure milk, later with thicker creamy material as the fluid is absorbed. When the swelling is pressed, milky fluid may come out of the nipple. Diagnosis can be made by aspiration or ultrasound. Milk may be aspirated, but the cyst usually fills up again after some days, and repeated aspiration is required. A galactocoele can be removed surgically under local anaesthesia. Breastfeeding need not be interrupted.

7.3 Non-infectious mastitis

When milk is not removed from part or all of a breast, milk production slows and eventually ceases. However, this process takes some days and may not be complete for 2-3 weeks. In the meantime the accumulated milk may cause an inflammatory response (8; 55; 94; 160; 161; 162).

Cytokines, both inflammatory and anti-inflammatory are found normally in milk. *Anti-inflammatory cytokines* and other factors are thought to protect the infant (34; 153), but *inflammatory cytokines*, such as *interleukin-8* (IL-8), may be more important for protecting the breast against infection. Increased levels of IL-8 are found in the breast during mastitis, and are a sign that an inflammatory response is occuring (46; 175). As part of the inflammatory response, the paracellular pathways, that is the tight junctions between the milk secreting cells of the mammary alveoli, open, allowing substances from plasma to pass through into the milk, particularly immunoproteins and sodium. At the same time, the increase in the pressure of the milk in the ducts and alveoli may force substances from milk back into the surrounding tissue. Cytokines from the milk may induce an inflammatory response in the surrounding tissue, and it is also possible that other components induce an antigenic reaction.

The inflammation is responsible for the signs and symptoms of mastitis. Part of the breast is painful, red, swollen, and hard. Usually only one breast is affected. The woman often has a fever and feels ill. However, in two studies it was observed that one third to one half of women with mastitis had local signs only (7; 20).

The opening of the paracellular pathways results in changes in the composition of the milk. Levels of sodium and chloride increase, and the levels of lactose and potassium decrease (102; 126; 132). The taste of the milk changes: it becomes more salty and less sweet. Usually the saltiness is only temporary, lasting about a week (131). Sometimes the breast continues to be underused, and milk stasis and altered taste persist. This has been described as *unilateral chronic breast dysfunction* (25). However, the condition is reversible, and after the next pregnancy, normal function usually returns to the affected breast (130).

7.4 Immune factors in milk

A number of protective factors are normally present in milk including secretory IgA, lactoferrin, lysozyme, and C3 (a component of complement), and leukocytes (131; 160). Although more attention is usually paid to their importance for infant health, they may also help to protect the breast against infection by preventing *Staph. aureus* from becoming established (174). They have been shown to be an important defence mechanism in the bovine mammary gland (125). C3 and IgA promote phagocytosis of *Staph. aureus* by leukocytes in milk (13), and lactoferrin increases the adhesion of leukocytes to tissue at the site of inflammation (124). As part of the inflammatory response, additional immunoproteins from the serum, and increased numbers of leukocytes enter the milk (161).

Women in the Gambia who experienced repeated bouts of mastitis were found to have low levels of IgA, C3 and lactoferrin in their milk in comparison with other lactating mothers (131). This provides additional evidence that these factors constitute a defence mechanism, and that when levels are low, effective defence is reduced.

Increased immunoprotein levels occur during breast involution when breastfeeding is discontinued (58), and they may protect the breast at a time when milk stasis might allow bacterial growth.

7.5 Sub-clinical mastitis

Recently a condition called *sub-clinical mastitis* has been described (46; 175). Sub-clinical mastitis is diagnosed from the finding of a raised sodium-potassium ratio in the milk, and an increased concentration of interleukin-8, (IL-8) when there is no clinical mastitis. Increased sodium and IL-8 levels are thought to indicate that an inflammatory response is occurring, despite the absence of clinical signs. Sub-clinical mastitis has been found to be common among women in Bangladesh, Tanzania, Malawi and South Africa. An increased sodium-potassium ratio of breast milk has also been observed in association with poor weight gain of infants (46; 111), and when supplementary feeds are given to the infant, or when the number of breastfeeds is decreased, so that milk production falls below 400 ml per day (116). This suggests that sub-clinical mastitis may be associated with inadequate milk removal, and that it could be quite common in those situations. Morton in 1994 found that giving skilled guidance to mothers of infants up to one month of age, including helping them with attachment of the baby to the breast, was associated with improved lactation and reduction of elevated breast-milk sodium levels (111).

Sub-clinical mastitis is also associated, in HIV-positive women, with an increase in the HIV load in the breastmilk, and could be responsible for higher rates of mother-to-child transmission of HIV (148; 149; 150; 175). A 20-fold increased rate of mother-to-child transmission of HIV has been reported with clinical mastitis (76).

7.6 Infectious mastitis

Infective mastitis results if milk stasis remains unresolved, and the protection provided by the immune factors in milk and by the inflammatory response is overcome. Fresh human milk is not normally a good medium for bacterial growth (4; 90; 121; 128), and for infection to occur conditions must be present which prevent the breast from destroying and eliminating bacteria. The natural direction of the flow of milk along the ducts, when removed efficiently, would be expected to wash any organisms down and out of the breast. Inefficient milk removal, resulting in accumulation of milk, creates the conditions favourable to bacterial growth, and the anti-infective processes may be overwhelmed.

The signs and symptoms of infectious mastitis are, as discussed above, impossible to distinguish from non-infectious mastitis. Part of usually one breast becomes red, painful, swollen and hard, and there may be general symptoms of fever and malaise. An accompanying sign may be a nipple fissure.

Infectious mastitis has been classified by different authors in several ways. First according to its site: *superficial mastitis* occurring in the dermis and *intramammary mastitis* located either in the glandular tissue itself, (*parenchymous*) or in the connective tissue of the breast (*interstitial*). Second, according to the epidemiological patterns: *epidemic* or *sporadic*.

Attempts have also been made to link both classifications, as reflected in the accompanying box, but the usefulness of this remains uncertain (11; 48; 52; 55; 94).

Categorisation:	Intramammary Infectious Mastitis	
	parenchymous	interstitial
Location:	glandular tissue	connective tissue
Denomination:	adenitis	cellulitis
Symptoms:	localised and less severe	widespread and more severe
Pus present:	Sometimes	no
Manner of infection:	*epidemic*	*sporadic*

Cell counts and bacterial colony counts are useful to distinguish between infectious and non-infectious mastitis. Culture of the breastmilk may help to determine the infecting organism, if there is one, and its antibiotic sensitivity (102).

If culture is not possible as a routine, it can be done selectively:

- for hospital-acquired, or severe or unusual cases;
- if there is no response to antibiotics within two days;
- if there is recurrent mastitis;

Recurrent mastitis may be due to delayed or inadequate treatment of the initial condition (71) or uncorrected poor breastfeeding technique. Sometimes recurrence is associated with candidiasis (Section 5.4). Occasionally there is an underlying breast condition which causes persistently poor drainage of part of a breast, such as a ductal abnormality, a cyst or tumour, which should be identified and treated accordingly (94; 122). These conditions will not be further discussed in this review.

7.7 Breast Abscess

A lactating breast, as any other infected tissue, localises infection by forming a barrier of granulation tissue around it. This becomes an abscess capsule, which is filled with pus (137).

There is a severely painful swollen lump, with redness, heat, and oedema of the overlying skin. In neglected cases the lump may be fluctuant, with skin discoloration and necrosis. Fever may or may not be present (11; 62; 67; 77; 117). To confirm the diagnosis, pus can be aspirated with a syringe and wide bore needle. The differential diagnosis of breast abscess includes galactocoele (see Section 7.2), fibroadenoma, and carcinoma. These conditions are not discussed further in this review.

8. Prevention

Mastitis and breast abscess are largely preventable, if breastfeeding is managed appropriately from the beginning to prevent situations which give rise to milk stasis (32; 71; 102; 161; 167), and if early signs such as engorgement, blocked duct and nipple soreness are treated promptly. This is needed as part of normal maternity care, and as an ongoing part of both facility based and community care for women and children. Appropriate management in maternity wards is required by the Baby-Friendly Hospital Initiative, or BFHI (178).

8.1 Improved understanding of breastfeeding management

Women and those who care for them need to know about effective breastfeeding management, both to feed the infant adequately and to keep the breasts healthy. Important points are:

- start to breastfeed within an hour or so of delivery;
- make sure that the infant is well attached to the breast;
- breastfeed with no restrictions, in either the frequency or duration of feeds, and let the baby finish the first breast first, before offering the other;
- breastfeed exclusively for at least 4 and if possible 6 months.

Women and their carers also need to understand that the following may interfere with breastfeeding, limit or reduce the amount that an infant suckles, and increase the risk of milk stasis:

- using a pacifier;
- giving the infant other foods and drinks in the first few months, especially from a feeding bottle;
- taking the infant off the first breast before he is ready to make sure that he takes the other
- a heavy or stressful workload;
- missing breastfeeds, including when the infant starts sleeping through the night;
- trauma to the breasts, from violence or any other cause.

These should be avoided or women protected from them as far as possible; but when they inadvertently occur, a woman can prevent mastitis if she takes extra care of her breasts.

8.2 Routine measures as part of maternity care

The following practices are important to prevent milk stasis and mastitis. They should be routine in any setting where mothers give birth or are cared for before and after delivery: in maternity hospitals; in smaller facilities such as health centres; or at home when mothers deliver there, or when they return after delivery:

- Infants should have early contact with their mothers, and start breastfeeding as soon as they show signs of readiness, usually within the first hour or so;
- Infants should stay in the same bed as their mother, or close to her in the same room;
- Every mother should receive skilled help and support with her breastfeeding technique, whether or not she has breastfed before, to ensure good attachment, effective suckling and efficient milk removal;
- Every mother should be encouraged to breastfeed ‘on demand’, whenever the infant shows signs of readiness to feed, such as opening the mouth and searching for the breast;
- Every mother should understand the importance of unrestricted and exclusive breastfeeding, and of avoiding the use of supplementary feeds, bottles and pacifiers;
- Women should receive skilled help to maintain lactation if their infants are too small or weak to suckle effectively;
- When a mother is in hospital, she needs skilled help at the first feed and for as many of the subsequent feeds as necessary;
- When a mother is at home, she needs skilled help during the first day after delivery, several times during the first two weeks, and subsequently as needed until she is breastfeeding effectively and confidently.

8.3 Effective management of breast fullness and engorgement

If a woman’s breasts become very full or engorged during the first week, when her milk ‘comes in’, it is essential to ensure that her milk is removed and the condition allowed to resolve.

- The mother should be helped to improve her infant's attachment at the breast, to improve milk removal, and to avoid damage to the nipples.
- She should be encouraged to breastfeed as often and as long as her infant is willing, with no restrictions.
- If the infant's suckling does not relieve the fullness or engorgement sufficiently, or if her nipple is pulled so flat that the infant has difficulty attaching, she needs to express her milk. She should express enough to soften the breasts, relieve the discomfort, and enable the infant to attach and suckle effectively.
- Expression can be done either by hand or with a pump (178). If the breasts are very painful, another way to express milk is by using the hot bottle method (see Annex 2).
- After a day or two, the condition should resolve, and the milk supply and the infant's needs will match each other.

8.4 Prompt attention to any signs of milk stasis

A woman needs to know how to care for her breasts, and about early signs of milk stasis or mastitis, so that she can treat herself at home, and seek help quickly if the situation does not resolve. She should check her breasts to see if there are any lumps, if there is any pain, or hotness or redness:
- if she is affected by any of the risk factors, such as missing breastfeeds;
- if she has a fever or feels ill, for example with a headache.

If she has any of these symptoms, she needs to:
- rest, in bed if possible;
- breastfeed very often from the affected breast;
- put a warm compress on the affected breast, bathe it in warm water, or have a warm shower;
- *gently* massage any lumpy areas while the infant is feeding to help the milk to flow from them
- seek help from a health worker if she is not better next day.

These measures can often prevent milk stasis or early signs of mastitis from progressing and developing into severe mastitis.

8.5 Prompt attention to other difficulties with breastfeeding

A woman needs skilled help with breastfeeding at any time if she has a difficulty which may lead to milk stasis, such as:
- nipple pain or fissure;
- breast discomfort after feeding;
- nipple compression (a white line across the tip when the infant releases the breast);
- unsatisfied infant: very frequent, infrequent, or prolonged feeds;

- an unsatisfied infant: very frequent, infrequent, or prolonged feeds;
- loss of confidence in her milk supply, believing herself to have insufficient milk;
- early introduction of other feeds; or
- using a pacifier.

Midwives and other facility based health workers need appropriate knowledge and skills so that they can help mothers to establish breastfeeding in the early postpartum period, to continue breastfeeding subsequently, and to overcome early difficulties before they become more serious and put lactation at risk.

Knowledge and skills about ongoing support of breastfeeding need also to be available in the community, among community based health workers, TBAs, or peer counsellors, and women generally, so that women can help each other to prevent difficulties; and if problems arise, appropriate treatment can be started early.

8.6 Control of infection

While appropriate management of breastfeeding is fundamental for the prevention of mastitis, reduction of the risk of infection it is also important, especially in the hospital setting.

Thorough and frequent hand washing by both health workers and mothers is necessary (88). Health workers should wash their hands after every contact with a mother or infant, or with any possible source of pathogenic organisms. Plain soap is adequate to remove surface organisms, but for health workers in frequent contact with body fluids, an anti-microbial hand washing product is more effective, provided it is in contact with the skin for at least 10 seconds per wash (89). Peters showed that additional hand disinfection at the bedside of breastfeeding mothers in hospital reduced the incidence of mastitis from 2.8% to 0.66% (127).

Early skin-to-skin contact, followed by rooming-in of the infant with the mother are also important ways to reduce hospital infections, as discussed in Section 5.3.

Other measures and products have been proposed, but there is no evidence of their efficacy. These include breast massage, lotions, ointments and sprays, such as *Boracic lotion*, *Dettol 2.5%, penicillin ointment chlorhexidine (0.2%),* and *chlorhexidine spray* (12; 22; 61; 62; 68; 70; 78; 109).

9. Treatment

For treatment of any of the conditions discussed, it is necessary

1. To take a history from the mother, to learn if there are any obvious causes for her difficulties, or predisposing factors;
2. To observe a breastfeed, and assess if her breastfeeding technique and the infant's attachment at the breast are satisfactory, and how they might be improved.

9.1 Blocked duct

Treatment requires improved removal of milk, and avoiding any obstruction to milk flow.

- Ensure that the infant is well positioned and attached at the breast. Some authors recommend holding the infant with the chin towards the affected part of the breast, to facilitate milk removal from that section (91), while others consider generally improved attachment adequate (71; 167).
- Explain the need to avoid anything that could obstruct the flow of milk, such as tight clothes, and holding the breast too near the nipple.
- Encourage her to breastfeed as often and as long as her infant is willing, with no restrictions.
- Suggest that she apply wet heat (e.g. warm compresses or a warm shower) (91; 94; 139; 167).

Occasionally, these techniques do not relieve a woman's symptoms. This may be because there is particulate matter obstructing the duct. Massage of the breast, using a firm movement of the thumb over the lump towards the nipple may be helpful (3; 5; 65; 91; 133). However, this should be done gently, because when breast tissue is inflamed, massage can sometimes make the situation worse.

If there is a white spot visible at the end of the nipple, it needs to be removed, with the fingernails, a rough flannel, or with the aid of a sterile needle (17; 133).

Unfortunately, blocked ducts tend to recur, but once a woman knows what they are and how to treat them herself, she can start treatment early and avoid progression to mastitis.

9.2 Mastitis

If despite all efforts at prevention, mastitis occurs, it must be treated promptly and adequately. If treatment is delayed or incomplete, recovery is less satisfactory. There is an increased risk of breast abscess and relapse (32; 74; 102; 154; 161; 170).

The main principles of treatment of mastitis are:

- Supportive counselling
- Effective milk removal
- Antibiotic therapy
- Symptomatic treatment

- SUPPORTIVE COUNSELLING

  Mastitis is a painful and frustrating experience, and it makes many women feel very ill. In addition to effective treatment and control of pain, a woman needs emotional support (167). She may have been given conflicting advice from health professionals, she may have been advised to stop breastfeeding, or given no guidance either way. She may be confused and anxious, and unwilling to continue breastfeeding.

  She needs reassurance about the value of breastfeeding; that it is safe to continue; that milk from the affected breast will not harm her infant (see Section 10); and that her breast will

recover both its shape and function subsequently. She needs encouragement that it is worth the effort to overcome her current difficulties.

She needs clear guidance about all measures needed for treatment, and how to continue breastfeeding or expressing milk from the affected breast. She will need follow up to give continuing support and guidance until she has recovered fully.

- EFFECTIVE MILK REMOVAL

  This is the most essential part of treatment (71). Antibiotics and symptomatic treatment may make a woman feel better temporarily, but unless milk removal is improved, the condition may become worse or relapse despite the antibiotics.

  - Help the mother to improve her infant's attachment at the breast (see Annex 1).
  - Encourage frequent breastfeeding, as often and as long as the infant is willing, without restrictions.
  - If necessary express breast-milk by hand or with a pump or hot bottle, until breastfeeding can be resumed (Annex 2).

- ANTIBIOTIC THERAPY

  Antibiotic treatment is indicated if either:
  - cell and bacterial colony counts and culture are available and indicate infection, or
  - symptoms are severe from the beginning, or
  - a nipple fissure is visible, or
  - symptoms do not improve after 12-24 hours of improved milk removal (2; 6; 71; 102).

  An appropriate antibiotic must be used (Table 4). To be effective against *Staph. aureus* a β-lactamase resistant antibiotic is needed. For gram negative organisms, cephalexin or amoxicillin may be the most appropriate. If possible, milk from the affected breast should be cultured and the antibiotic sensitivity of the bacteria determined.

  The chosen antibiotic must be given for an adequate length of time. 10-14 days is now recommended by most authorities (94; 167). Shorter courses are associated with a higher incidence of relapse.

- SYMPTOMATIC TREATMENT

  Pain should be treated with an analgesic (5; 167). Ibuprofen is considered the most effective, and it may help to reduce inflammation as well as pain. Paracetomol is an appropriate alternative.

  Rest is considered essential (94; 167) and should be in bed if possible. As well as helping the woman herself, resting in bed with the infant is a useful way to increase the frequency of breastfeeds, and may thus improve milk removal.

  Other measures which are recommended are the application of warm packs to the breast, which both relieves pain and helps the milk to flow; and ensuring that the woman drinks sufficient fluids.

Other Therapeutic Approaches

A number of other forms of treatment of mastitis are sometimes suggested, but there is little evidence of their effectiveness.

1. *Pus stripping.* This technique was proposed by Bertrand and Rosenblood in 1991, following a form of veterinary practice (11). In a study of 475 women with clinical mastitis this treatment was interpreted as having a favourable outcome. However the study was uncontrolled, and few milk cultures were positive for pus forming bacteria. The procedure is reported to be very painful, and there is no reason to suppose that it is more effective than improved physiological removal of milk.

2. *Cabbage leaves.* The use of chilled or room temperature cabbage leaves is sometimes recommended for relief of symptoms of engorgement (142; 143). They were found to be as effective as cold packs in the relief of pain, but there is no evidence that they shorten the duration of the condition.

3. *Dietary measures.* Avoidance of drinks such as coffee which contain methylxanthines; and lowering fat intake are considered helpful by some authors (5; 31; 94; 156), but the evidence is mainly anecdotal.

4. *Herbal treatment.* In traditional Chinese medicine plant extracts (*Fructus gleditsiae*) are used (66), apparently successfully.

Table 4. Antibiotics for treatment of infectious mastitis

Antibiotic:	Dosage:	References:
Erythromycin	250 – 500 mg 6 hourly	(5), (23), (36), (163)
Flucloxacillin	250 mg 6 hourly	(23)
Dicloxacillin	125-500 mg 6 hourly by mouth	(94), (122)
Amoxacillin	250-500 mg every 8 hours	(94)
Cephalexin	250-500 mg 6 hourly	(5), (23), (36), (50), (122), (163)

Penicillin and ampicillin, though often used in the past, are not currently regarded as appropriate antibitoics, in view of the increasing resistance shown by *Staph. aureus* isolated from women with puerperal mastitis (37; 84; 102). Methicillin and gentamycin resistance also has been reported since the 1960s (63; 87; 129; 155). An increasing number of oxacillin-resistant *Staph. aureus* (ORSA) infections are being reported (28; 94). Most strains of *Staph. aureus* remain sensitive to fucidic acid, rifampicin and vancomycin, but vancomycin-resistant strains are emerging (40). Several antibiotics, including ampicillin, gentamycin, tetracyclines and chloramphenicol, have been shown to have anti-inflammatory properties (33). They might therefore reduce symptoms of mastitis even in the absence of an active infection (71). Such an effect might be interpreted as evidence of infection, and distract attention from the need to improve milk removal.

9.3 Breast Abscess

- SURGICAL TREATMENT

If an abscess has formed, the pus must be removed. This can be done by incision and drainage, which usually requires general anaesthesia (77; 145), but it can also be done by aspiration, guided by ultrasound, if available (35; 36; 47; 59; 79). Ultrasound is a useful diagnostic tool for breast abscess, and a thoroughly performed, ultrasound guided aspiration of the pus may be curative. It is less painful and mutilating than incision and drainage, and can be done under local anaesthesia (35; 36), often as an outpatient.

Systemic treatment with antibiotics appropriate to the organisms' sensitivity is usually required in addition (10; 67; 77). However, antibiotics alone, without removal of pus, are unlikely to be of value. An abscess wall creates a barrier that protects the pathogenic bacteria from the body's defences, and makes it impossible to achieve effective antibiotic levels in the infected tissue (10; 84; 134; 137).

- SUPPORT OF BREASTFEEDING

Whether breastfeeding continues or stops after an episode of mastitis or breast abscess, and how long it continues, depends partly on the counselling and help that a mother receives (9). If she is given adequate guidance and clinical and emotional support, she should recover fully, and experience no problems with subsequent lactations. If she receives poor guidance and support, she may never breastfeed again.

Doctors and other health workers who care for a woman with a breast abscess should reassure her that she can continue to breastfeed, that this will not harm her infant, and that she should be able to breastfeed any other infants that she may have. They should explain how this can be achieved, and what the management of the condition will be.

To ensure continued satisfactory breastfeeding, management should be as follows:

- The infant should be kept with the mother both before and after surgery.
- The infant can continue to feed from the unaffected breast.
- While the mother undergoes surgery, if she is likely to be unable to breastfeed for more than 3 hours, the infant needs to be fed in some other way.
- As part of the preparation for surgery, the mother can express milk from her unaffected breast, and the expressed milk can be fed to the infant from a cup while she is undergoing treatment.
- As soon as she has regained consciousness, (if she was given a general anaesthetic), or as soon as surgery is completed (if a local anaesthetic was used), she can breastfeed again on the unaffected side.
- As soon as the pain of the wound allows, she can resume breastfeeding from the affected side. This is usually possible within a few hours, unless surgery is close to the nipple. She should be given analgesics as needed to help to control the pain and permit earlier resumption of breastfeeding.
- She is likely to need skilled help to get the infant to attach to the affected breast again, and it may take several attempts before he suckles well. Encourage her to persist, to put the infant to the affected breast each time she feeds him, and help her to ensure good attachment.

- If the affected breast is still producing milk, it is important for the infant to suckle and remove it, to prevent continued milk stasis and relapse of the infection.
- If the infant is unwilling to attach or to suckle on the affected breast at first, it may be necessary to express the milk until he starts suckling again.
- If milk production in the affected breast has decreased, frequent suckling is the most effective way to stimulate an increase in production.
- Meantime, the infant can continue to feed from the unaffected breast. Usually an infant can get enough milk by feeding from one breast only, so he will be adequately nourished while the milk production from the affected breast recovers.

However, if an abscess is extensive, and treatment delayed, the breast may not return to full milk production. For further information about re-establishing lactation after an interruption, see (181).

10. Safety of continuing to breastfeed

Maintaining lactation when a woman has mastitis or breast abscess is important both for her own recovery, and for her infant's health.

Stopping breastfeeding during an attack of mastitis does not help the mother to recover (9; 32; 77; 117; 119); on the contrary, there is a risk that it can make her condition worse (11; 32; 84; 100; 117; 161; 170). Furthermore, if a woman stops breastfeeding before she is emotionally ready, she may suffer considerable emotional distress (54; 166).

Risk of infection to the infant
Many health workers are concerned about a possible risk of infection to the infant, especially if the milk appears to contain pus. They recommend manual expression, and discarding of the milk (103). However a number of studies have demonstrated that continuing to breastfeed is generally safe, even in the presence of *Staph. aureus* (Table 5)(32, 75; 100; 102; 119; 157). Only if a mother is HIV-positive is it necessary for her infant to stop feeding from the affected breast until it has recovered.

Table 5. Effect on infant of continued breastfeeding from an infected breast

Authors	Year	Cases of mastitis	Cases of breast abscesses	Infants who continued breastfeeding	Antibiotic therapy	Harmful effects
Taylor et al. (157)	1946	7	2	9	9/9	0
Jeffrey (75)	1947	48	2	50	45/50	0
Devereux (32)	1970	58	8	47	11/58	0
Marshall (100)	1975	41	0	41	41/41	0
Niebyl (119)	1978	20	0	20	20/20	0
Matheson (102)	1988	43	0	43	33/43	0

There have been occasional case reports of staphylococcal scalded skin syndrome in breastfed infants whose mothers had mastitis or breast abscess (80; 136). This syndrome is caused by an exotoxin which is produced by certain strains of staphylococci (104). However, it is not clear in those cases whether the infant or the mother was the original source of infection, or whether transmission was through breastfeeding or merely close contact. The authors suggest that when possible in such cases, both maternal milk and the infant's nasopharynx should be cultured prior to antibiotic therapy. When antibiotic therapy is indicated, both mother and infant can be treated with an isoxazole penicillin (oxacillin, cloxacillin or dicloxacillin) (80; 136).

There are a few documented cases of transmission of group B streptococcal infections to infants from mothers with a breast abscess (137). One case of Salmonella mastitis associated with a positive stool culture in the infant has been reported (51). However, both mother and infant recovered with antibiotic treatment and breastfeeding was not interrupted.

This small number of case reports and their usually benign outcome does not justify a general policy of discontinuation of breastfeeding, in view of the clear disadvantages to the infant of doing so. The infant should be observed for any signs of infection, and if mastitis is known to be due to staphylococcal or streptococcal infection, simultaneous antibiotic treatment of the infant may be considered (94).

11. Long term outcome

With timely, appropriate and adequate treatment of mastitis and breast abscess, recovery should be complete, and normal function of the breast can usually be expected with subsequent lactations.

However, delayed, inappropriate or inadequate treatment may result in relapse, more extensive lesions and even permanent tissue damage. Repeated episodes of mastitis may give rise to chronic inflammation (141) and irreversible distortion of the breast (134).

Breast abscess may compromise future lactation in about 10% of affected women (109). A large abscess may require extensive resection of breast tissue which can result in disfigurement and sometimes a functional mastectomy (112).

12. Mastitis in women who are HIV-positive

The management of mastitis described above is appropriate for women who are HIV negative, or whose HIV status is not known, and who are encouraged to breastfeed in the usual way. For women who are HIV-positive, there are other considerations, as mastitis has been found to increase the risk of mother-to-child transmission of HIV (76; 150).

WHO, UNICEF and UNAIDS policy (180) recommends that women who are HIV-positive should be counselled about a number of infant feeding options, and supported in their choice, whether they decide to breastfeed or to use one of the alternatives. Support for those women who choose to breastfeed must include help to prevent mastitis and related conditions, and appropriate management if any of them occur.

Prevention

HIV-positive women who choose to breastfeed, need skilled help from the beginning, to ensure that their breastfeeding technique is optimal, to minimise the risk of mastitis, sub-clinical mastitis, nipple fissure or abscess developing; and to encourage and enable them to breastfeed exclusively. The help that they need is the same as for HIV-negative women, though they may need more intensive care.

Management

If an HIV-positive woman develops mastitis, fissure or abscess, she should avoid breastfeeding from the affected side while the condition persists.

- She must express milk from the affected breast, by hand or pump, if necessary using the hot bottle technique, to ensure adequate removal of milk (see Annex 2). This is essential to prevent the condition becoming worse, to help the breast to recover, and to maintain milk production. The health worker should help her to ensure that she is able to express milk effectively.
- Antibiotic treatment will usually be indicated (section 9.2).
- If only one breast is affected, the infant can feed from the unaffected breast, feeding more often and for longer to increase milk production. Most infants can get enough milk from one breast. The infant can feed from the affected breast again when it has recovered.
- If both breasts are affected, she will not be able to feed from either side. She will need to express her milk from both breasts. She can resume breastfeeding when she has recovered.

- The health worker will need to discuss other feeding options for her to give meanwhile. She may decide to boil her expressed milk, or to give home prepared or commercial formula. She should feed the infant by cup.

Sometimes a woman may decide to discontinue breastfeeding at this time, if she is able to give another form of milk safely. She should continue to express enough milk to allow her breasts to recover, and to keep them healthy, until milk production ceases.

Further information about infant feeding options may be found in *HIV and Infant Feeding: A Training Course* (182).

13. Conclusion

Mastitis and breast abscess are common and largely preventable conditions, which occur in all populations and which put breastfeeding at risk. They are caused primarily by inefficient removal of breastmilk, but also by bacterial infection, which is probably secondary to milk stasis. Particularly virulent strains of bacteria may cause epidemics of puerperal mastitis in hospitals, when infants are kept in nurseries away from their mothers.

Improved breastfeeding practices, including early skin-to-skin contact between mother and infant, rooming-in, skilled help to ensure that an infant is well attached at the breast, and unrestricted and exclusive breastfeeding, are an efficient way to prevent both milk stasis and spread of infection. Health workers and the community as a whole need to understand this, in order to support women in the best practices.

Mothers need to know how to recognise early signs of mastitis, milk stasis and blocked ducts, what they can do at home to treat them and to prevent the condition from becoming worse. They need to know when to seek skilled help for the condition, and it is important that health workers or breastfeeding counsellors are available who understand the condition, and who can give appropriate and adequate help.

Whenever possible breastfeeding should continue, both to improve milk removal and to help the condition to resolve, and for the benefit of the infant. If present, bacterial or other infection should be treated with an appropriate anti-microbial agent, but this should be in addition to, and not an alternative to techniques which ensure efficient removal of milk.

Annex 1: Breastfeeding techniques to prevent and treat mastitis

The importance of good attachment for effective suckling

A probable cause of mastitis is failure to remove milk from the breast effectively; it is thus important to understand how an infant removes milk.

To remove milk efficiently and effectively, an infant needs to be *correctly attached to the breast,* and to be allowed to suckle *without any restriction of the time at the breast.* Milk removal should be *regulated by the infant's need to feed.* If the infant is *incorrectly attached* to the breast, or if his *time at the breast is restricted*, he will not be able to remove milk from the breast effectively or efficiently. Milk production is a continuous process (30), so if it is not removed as it is formed, then the volume of milk in the breast may exceed the capacity of the alveoli to store it comfortably. If this situation is not resolved it may cause *engorgement and mastitis* in the short term, and affect continued *milk production* in the long term.

Production of breastmilk

During pregnancy the breast is "primed" for milk production by the gradually rising serum prolactin levels in the maternal bloodstream. The action of prolactin is inhibited by the presence of high levels of the placental steroid hormones, progesterone and oestrogen, and milk production cannot begin until the levels of these hormones have dropped sufficiently, following the expulsion of the placenta.

Under the uninhibited action of prolactin, milk secretion begins at the base of the alveolar cells, where small droplets form which migrate to the apical cell membrane and are extruded into the alveolar ducts for storage (85).

Initially milk production is maintained by high prolactin levels. However, prolactin levels gradually decrease, and after a few weeks, milk removal becomes the principle driving force: "pull rather than push" (8). There is a change from hormonal control by prolactin in the bloodstream, to a local autocrine control. It is the continued removal of a suppressor peptide known as the *feedback inhibitor of lactation (FIL)* that allows the amount of milk produced to equal the milk removed from the breast (172).

As milk synthesis begins, a mother experiences considerable cardiovascular changes, including increased blood flow to the breasts, which may begin to feel warm and "full" even though the volume of milk produced is not yet very great (73). The length of time between placental delivery and copious milk production varies, but seems to be on average 48-96 hours, or 2-4 days post delivery.

Learning to breastfeed

For every mother, and with each infant she has, breastfeeding is a *learned skill*, only the production of the milk is "natural". Women who give birth in cultures in which the sight of an infant at the breast is commonplace, may well be better equipped in terms of the expertise available to them in the community, and their expectations of the process, but even for them breastfeeding is a skill that has to be acquired through practice.

During the first few days before copious milk is produced, the breasts are soft, and both mother and infant have time to start learning how to breastfeed. When the breasts become fuller, it is temporarily more difficult to attach the infant well if the mother has not already had some practice.

How an infant suckles and removes milk

In order to feed well, an infant needs to create a teat from the breast, which consists of about *one-third nipple and two-thirds breast tissue*. This ensures that the infant's tongue is in close proximity to the

lactiferous sinuses, which collect milk from all lobes of the breast and converge beneath the areola. The infant can then extract milk from the lactiferous sinuses using a wave-like movement of the tongue against the breast tissue. This process is aided by the *oxytocin reflex* which the infant stimulates as he begins to feed, and which helps the milk to flow along the ducts from the alveoli.

An infant who is incorrectly attached to the breast usually causes the mother pain. Pain in the context of breastfeeding, as in all aspects of human experience, is an indication that damage is being, or has been, done. The survival response is to avoid or remedy the cause. If a mother is experiencing nipple pain, the likeliest cause is that the nipple is being compressed between the infant's tongue and hard palate as he attempts to remove the milk from the lactiferous sinuses. If this continues, a nipple fissure is likely to result, which may become infected.

If poor attachment does not cause the mother pain or damage the nipple, then sub-optimal feeding continues until some other symptom appears. This may be in the infant, such as poor weight gain, weight loss, very frequent feeds, very long feeds, abdominal 'wind', explosive stools, vomiting or possetting after feeds; or in the mother, such as engorgement, mastitis or over production of milk.

The appropriate response is to enable the infant to take a bigger "mouthful" of breast, so that the nipple can be taken further back into the infant's mouth, as far as the junction of the hard and soft palate. The infant's tongue will then be in contact with the areola and underlying breast tissue, and not only the nipple. When this is achieved, the infant ceases to cause his mother pain, and is able to remove milk more efficiently.

Signs of good attachment

An infant who is well attached to the breast, feeds with a *wide mouth and an active tongue,* and this is evident from the rhythm of the feed. First the infant gives a few quick suckles, which stimulate the oxytocin reflex and the release of milk. Then he changes to slow *deep suckles* as the milk starts to flow, and can usually be seen or heard to swallow. He sometimes pauses, and pauses become more frequent as the feed progresses, and the flow slows. The infant's body *is relaxed* as he feeds and he has *no difficulty breathing.* The infant remains attached to the breast throughout the feed, until he is ready to stop, when he releases the breast *spontaneously,* either sleepily or calmly. An infant who comes off the breast with distress or restlessness may not have been well attached, and needs help to take more breast into his mouth. The exception to this is if the infant needs to stop after a minute or two of feeding when the milk flow is at its height, in order to bring up wind. The infant then returns to the same breast until he has finished with that breast. He indicates that he has finished by releasing the breast. The mother can then offer the second breast, which the infant takes or not, according to appetite.

A mother should not interrupt a breastfeed, or attempt to hurry it up by shaking the baby or the breast. She should let the baby continue undisturbed until he is ready to stop. A small baby may take a long time over feeds in the first few days, and may pause frequently during them.

Women have different sizes of areola; if an infant is well attached there may be no areola visible. If the areola is wide enough to see, and if the infant is well attached, then there will be *more areola visible above the infant's upper lip than below the lower lip.* If the infant's body is almost horizontal, mother and health worker can both see the areola above the upper lip. But if the infant is more upright, it is difficult for mother to see this. Health worker and mother can both see or feel that the infant's *chin is touching the breast*, but only the health worker can see if the infant's *lower lip is turned back*, and is well away from the base of the nipple. There is no reason to encourage a mother to look at the lower lip; if her infant' chin is as close as it should be, it is difficult for her to see the lower lip, and there are other, more reliable signs that she can use.

Both can see that the baby's *cheeks are rounded*, and not hollowed or sucked in; and that the *rhythmical jaw action* extends as far as the ears.

The feed should be quiet, with the baby in control. Noisy, gulping feeds suggest that an infant is not optimally attached.

Helping a mother to achieve correct attachment

The mother should be comfortable. If she is sitting, her back should be straight and her lap almost flat, with her knees slightly raised. The infant's head should be on the mother's forearm, not in the bend of her elbow, which is too far away from the breast and nipple. The infant needs to be *turned towards the mother* and yet be able to *come up to the breast from below*, so that the infant's upper eye can make eye contact with the mother. A pillow, some folded cloth, or some other means of supporting the weight of the infant, may be useful to help her achieve this. It may be helpful to wrap the infant while the mother is learning, to ensure that the infant's lower arm is at his side, and not between his body and the breast. Alternatively, the infant's arm can be tucked around the mother's body.

If the breast is large enough to need some support, the mother can place her fingers flat on her chest below her breast, where the breast meets the chest wall. Her thumb rests lightly on the top of the breast. Raising the breast slightly will make the breast tissue firmer. If a breast is very large, the mother may need to use her fingers to support the underside of the breast as well.

The infant's body and shoulders should be supported in such a way that *his head is free to extend slightly* as he is brought to the breast. The infant should be moved against the breast, so that his mouth touches the nipple, in order to *stimulate him to open his mouth wide and extend his tongue*. As he begins to do this, he should be moved *quickly and smoothly* to the breast so that his *chin and lower jaw reach the breast first*, while his mouth is still wide open. The aim is to get his *bottom lip and jaw as far away from the base of the nipple as possible.* This enables him to take in as much breast as possible with his tongue, and feed in a manner that is both efficient for removal of milk, and pain free for the mother. The infant should be brought to the breast – it is important not to push the breast towards the infant.

Fig. 1 An infant who is well attached at the breast

Fig.2 An infant who is poorly attached at the breast

Annex 2. Expression of breastmilk

How to express breastmilk by hand

Teach a mother to do this herself. Do not express her milk for her. Touch her only to show her what to do, and be gentle.

Teach her to:

- Wash her hands thoroughly.
- Sit or stand comfortably, and hold the container near her breast.
- Put her thumb on her breast ABOVE the nipple and areola, and her first finger on the breast BELOW the nipple and areola, opposite the thumb. She supports the breast with her other fingers (Figure 3).
- Press her thumb and first finger slightly inwards towards the chest wall. She should avoid pressing too far or she may block the milk ducts.
- Press her breast behind the nipple and areola between her finger and thumb. She must press on the lactiferous sinuses beneath the areola (Figure 4). Sometimes in a lactating breast it is possible to feel the sinuses. They are like pods, or peanuts. If she can feel them, she can press on them.
- Press and release, press and release.
  This should not hurt – if it hurts, the technique is wrong.
  At first no milk may come, but after pressing a few times, milk starts to drip out. It may flow in streams if the oxytocin reflex is active.
- Press the areola in the same way from the SIDES, to make sure that milk is expressed from all segments of the breast.
- Avoid rubbing or sliding her fingers along the skin. The movement of the fingers should be more like rolling.
- Avoid squeezing the nipple itself. Pressing or pulling the nipple cannot express the milk. It is the same as the baby sucking only the nipple.
- Express one breast for at least 3-5 minutes until the flow slows; then express the other side; and then repeat both sides. She can use either hand for either breast, and change when they tire.

Explain that to express breastmilk adequately may take 20-30 minutes. It is important not to try to express in a shorter time.

Fig. 3 **Fig. 4**

The warm bottle method for the expression of breastmilk

This is a useful technique to relieve severe engorgement, when a breast is very tender, and the nipple is tight, so that hand expression is difficult.

- You need a suitable bottle:
  - made of glass, not plastic;
  - 1-3 litres in size – not smaller than 700 ml;
  - with a wide neck – at least 2 cm diameter, if possible 4 cm – so that the nipple can fit into it.

- You also need:
  - a pan of hot water, to warm the bottle;
  - some cold water, to cool the neck of the bottle;
  - a thick cloth, to hold the hot bottle.

- Pour a little of the hot water into the bottle to start warming it up. Then almost fill the bottle with hot water. Do not fill it right up too quickly or the glass may crack.
- Let the bottle stand for a few minutes to warm the glass.
- Wrap the bottle in the cloth, and pour the hot water back into the pan.
- COOL THE NECK OF THE BOTTLE with cold water, inside and outside. (If you do not cool the neck of the bottle, you may burn the nipple skin.)
- Put the neck of the bottle over the nipple, touching the skin all round to make an airtight seal.
- Hold the bottle steady. After a few minutes the whole bottle cools, and makes gentle suction, which pulls the nipple into the neck of the bottle. Sometimes when a woman first feels the suction, she is surprised and pulls away. You may have to start again.
- The warmth helps the oxytocin reflex, and milk starts to flow, and collects in the bottle. Keep the bottle there as long as the milk flows.
- Pour out the breastmilk, and repeat if necessary, or do the same for the other breast. After some time, the acute pain in the breasts becomes less, and hand expression or suckling may become possible.

Fig. 5 a) Put hot water into the bottle.

Fig. 6 The mother holds the warm bottle over her nipple.

Fig. 6 b) Pour out the water.

Annex 3. Suppression of lactation

In the past, suppression of lactation was often included in the management of engorgement and mastitis. Now, maintenance of lactation is preferred. If is it necessary to discontinue breastfeeding, the current method of choice is physiological, and pharmacological methods of lactation suppression are no longer recommended.

Physiological methods

These include breast support, with a comfortable brassiere or binder, and expression of enough milk to keep the breasts comfortable and healthy, while natural involution takes place. The woman may experience pain, and analgesics should be given.

Pharmacological methods

The following drugs were formerly used, but no longer considered treatment of choice.

- *Stilboestrol.*
  Side effects include recurrence of engorgement when it is discontinued, withdrawal bleeding, thromoboembolism, and foetal abnormalities if the woman is pregnant.

- *Oestrogen,* alone or in combination with testosterone.
  This is not very effective, and is associated with an increased incidence of thromboembolic disease (85; 173). On discontinuation of the drug, pain and engorgement may recur.

- *Bromocriptine.*
  Bromocriptine inhibits prolactin secretion, and can be effective if given early in lactation, while prolactin levels are high. However, it may be less effective after some weeks when prolactin levels are low. It has been withdrawn from use for this indication in several countries because of the risk of myocardial infarction, hypertension, seizures and strokes. Other side effects reported include nausea, dizziness, hypotension, and severe headache (57; 85).

- *Cabergoline*.
  Cabergoline is similar to bromocriptine, and also inhibits prolactin secretion. It is longer acting. It may have some of the same minor side effects including headache, dizziness, hypotension, and nose bleeds, but is considered safer (57).

References

1. Editorial: Puerperal mastitis. *British Medical Journal,* 1976, **1**(6015):920-921.

2. *Successful Breastfeeding,* 2nd ed. Edinburgh, Churchill Livingstone, 1991.

3. White spots (Corpora amylacea). *Australian Lactation Consultants Association News,* 1992, **3**(3):8-9.

4. Ajusi JD, Onyango FE, Mutanda LN et al. Bacteriology of unheated expressed breast milk stored at room temperature. *East African Medical Journal,* 1989, **66**(6):381-387.

5. Amir L. Mastitis. *Australian Lactation Consultants Association News,* 1993, **4**(3):117.

6. Amir LH. Candida and the lactating breast: predisposing factors. *Journal of Human Lactation,* 1991, **7**(4):177-181.

7. Amir LH, Harris H, Adriske L. An audit of mastitis in the emergency department. *Journal of Human Lactation,* 1999, **15**:221-224.

8. Applebaum RM. The modern management of successful breast feeding. *Pediatric Clinics of North America,* 1970, **17**(1):203-225.

9. Banapurmath CR, Banapurmath SC, Mallikarjuna HB et al. Successful management of breast abscess with ongoing breastfeeding. *Indian Pediatrics,* 1995, **32**(4):4-9.

10. Benson EA, Goodman MA. Incision with primary suture in the treatment of acute puerperal breast abscess. *British Journal of Surgery,* 1970, **57**(1):55-58.

11. Bertrand H, Rosenblood LK. Stripping out pus in lactational mastitis: a means of preventing breast abscess. *Canadian Medical Association Journal,* 1991, **145**(4):299-306.

12. Beuningh H. A study of the value of a spray containing chlorhexidine for the prevention of puerperal mastitis. *Nederlands Tijdschrift Voor Geneeskunde,* 1972, **116**:1763-1764.

13. Bjorksten B, Gothefors L, Sidenvall R. The effect of human colostrum on neutrophil function. *Pediatric Research,* 1979, **13**(6):737-741.

14. Blaikeley J, Clarke S, MacKeith R et al. Breastfeeding: factors affecting success. *Journal of Obstetrics and Gynaecology of the British Empire,* 1953, **60**:657-669.

15. Breen K. Surviving mastitis. *La Leche League GB News,* 1992, **72**:3-4.

16. Brooker BE. The origin, structure and occurrence of corpora amylacea in the bovine mammary gland and in milk. *Cell and Tissue Research,* 1978, **191**(3):525-538.

17. Brown L, Fisher C. Blocked ducts. *New Generation,* 1982, **1**(1):16-17.

18. Brown WE, Hagler J, Morgan FE. The inhibition of breast engorgement with endocrine substances and its possible role in the prevention of puerperal breast abscess. *Southern Medical Journal,* 1960, **53**:548.

19. Cairns, J. *Breast abscesses drained*. 1996 (unpublished report from St Francis Hospital, Katete, Zambia).

20. Cantile H. Treatment of acute puerpural mastitis and breast abscess. *Canadian Family Physician,* 1988, **34**:2221-2227.

21. Centuori S, Burmaz T, Ronfani L et al. Nipple care, sore nipples and breastfeeding: a randomized trial. *Journal of Human Lactation,* 1999, **15**(2):127-132.

22. Chmelik V, Stribrny J, Papez L. Rotersept spray v prevenci mastidy a ragad bradavek [Rotersept spray in the prevention of mastitis and cracked nipples]. *Ceskoslovenska Gynekologie (Praha),* 1973, **38**:689-690.

23. Cigolini MC. Mastitis - the big picture. 1995, Sydney, Nursing Mothers Association of Australia. (Paper presented at NMAA Conference: *Breastfeeding - getting it right*. Sydney, Australia, March 1995).

24. Colbeck JC. An extensive outbreak of staphyloccal infections in maternity units. *Canadian Medical Association Journal,* 1949, **61**:557.

25. Conner AE. Elevated levels of sodium and chloride in milk from mastitic breast. *Pediatrics,* 1979, **63**(6):910-911.

26. Cook K. Mastitis and breastfeeding [letter; comment]. *Australian Family Physician,* 1994, **23**(3):497-499.

27. Cowie AT, Forsyth IA, Hart IC. *Hormonal control of lactation.* Berlin, Springer Verlag, 1980 (Monographs on Endocrinology, No. 15).

28. Craven DE, Reed C, Kollisch N et al. A large outbreak of infections caused by a strain of Staphylococcus aureus resistant of oxacillin and aminoglycosides. *American Journal of Medicine,* 1981, **71**(1):53-58.

29. Creasy RK, Resnik R. Maternal and fetal pathophysiology. *Maternal-Fetal Medicine: principles and practice*. Philadelphia, W.B. Saunders, 1984:628.

30. Daly SE, Owens RA, Hartmann PE. The short-term synthesis and infant-regulated removal of milk in lactating women. *Experimental Physiology,* 1993, **78**(2):209-220.

31. Dever J. Mastitis: positive interventions. *Midwifery Today,* 1992, **22**:22-25.

32. Devereux WP. Acute puerperal mastitis. Evaluation of its management. *American Journal of Obstetrics and Gynecology,* 1970, **108**(1):78-81.

33. Dewdney JM. The effects of antibacterial antibiotics on immune reactions and host resistance to infection. In: Lessof M, ed. *Immunological and clinical aspects of allergy* MTP Press, 1999:407-427.

34. Dinarello CA. Interleukin-1. *Reviews of Infectious Diseases,* 1984, **6**(1):51-95.

35. Dixon JM. Repeated aspiration of breast abscesses in lactating women. *British Medical Journal,* 1988, **297**(6662):1517-1518.

36. Dixon JM, Mansel RE. ABC of breast diseases. Symptoms, assessment and guidelines for referral. *British Medical Journal,* 1994, **309**(6956):722-726.

37. Duckworth GF. Methicillin-resistant Staphyloccus aureus. *Recent advances in Infection*1993:189-207.

38. Duncan JT, Walker J. Staphylococcus aureus in the milk of nursing mothers and the alimentary canal of their infants: A report to the Medical Research Council. *Journal of Hygiene,* 1942, **42**:474.

39. Eder M. Mamma: Entzündungen. In: Eder M, Gedigk P, eds. *Lehrbuch der allgemeinen Pathologie und der pathologischen Anatomie*, 32nd ed. Berlin, Springer-Verlag, 1986:758.

40. Edmond MB, Wenzel RP, Pasculle AW. Vancomycin-resistant Staphylococcus aureus: perspectives on measures needed for control. *Annals of Internal Medicine,* 1996, **124**(3):329-334.

41. Eschenbach DA. Acute postpartum infections. *Emergency Medical Clinics of North America,* 1985, **3**(1):87-115.

42. Escott R. Positioning, attachment and milk transfer. *Breastfeeding Reviews,* 1989, **14**:31-36.

43. Evans M, Head J. Mastitis; Incidence, prevalence and cost. *Breastfeeding Reviews,* 1995, **3**(2):65-72.

44. Fetherston C. Risk factors for lactation mastitis. *Journal of Human Lactation,* 1998, **14**(2):101-109.

45. Fildes V. Putting Mum in the picture. *Nursing Mirror,* 1979, **149**(3):22-24.

46. Filteau SM, Lietz G, Mulokozi G et al. Milk cytokines and subclinical breast inflammation in Tanzanian women: effects of dietary red palm oil or sunflower oil supplementation. *Immunology,* 1999, **97**:595-600.

47. Florey ME, MacVine JS, Bigby MAM. Treatment of breast abscesses with penicillin. *British Medical Journal,* 1946, **ii**:846-848.

48. Foxman B, Schwartz K, Looman SJ. Breastfeeding practices and lactation mastitis. *Social Science and Medicine,* 1994, **38**(5):755-761.

49. Fulton AA. Incidence of puerperal and lactational mastitis in an industrial town of some 43,000 inhabitants. *British Medical Journal,* 1945, **I**:693-696.

50. Fulton B, Moore LL. Antiinfectives in breastmilk. Part I: Penicillins and cephalosporins. *Journal of Human Lactation,* 1992, **8**(3):157-158.

51. Gibb AP, Welsby PD. Infantile salmonella gastroenteritis in association with maternal mastitis. *Journal of Infection,* 1983, **6**(2):193-194.

52. Gibberd GF. Sporadic and epidemic puerpural breast infections: a contrast in morbid anatomy and clinical signs. *American Journal of Obstetrics and Gynecology,* 1953, **65**(5):1038-1041.

53. Gibson RA, Rassias G. Infant nutrition and human milk. *Omega-6 Essential Fatty Acids: Pathophysiology and roles in clinical medicine* Alan R. Liss, Inc., 1999:283-293.

54. Groome A. From despair to happiness. *New Generation,* 1989, **8**(3):34-35.

55. Gunther M. Discussion on the breast in pregnancy and lactation. *Proceedings of the Royal Society of Medicine,* 1958, **51**(Section of General Practice):305-309.

56. Gupta R, Gupta AS, Duggal N. Tubercular mastitis. *International Surgery,* 1982, **67**(4 Suppl):422-44¡.

57. Hale TW. Bromocriptine mesylate. In: Hale TW, eds. *Medications and mother's milk*, 1998 ed. Amarillo, Texas, Pharmasoft Medical Publishing, 1998:75-76.

58. Hartmann PE, Kulski JK. Changes in the composition of the mammary secretion of women after abrupt termination of breast feeding. *Journal of Physiology (Cambridge),* 1978, **275**:1-11.

59. Hayes R, Michell M, Nunnerley HB. Acute inflammation of the breast--the role of breast ultrasound in diagnosis and management. *Clinical Radiology,* 1991, **44**(4):253-256.

60. Heinig MJ, Francis J, Pappagianis D. Mammary candidosis in lactating women. *Journal of Human Lactation,* 1999, **15**:281-288.

61. Herd B, Feeney JG. Two aerosol sprays in nipple trauma. *Practitioner,* 1986, **230** (1411):31-38.

62. Hesseltine HC, Freundlich CG, Hite KE. Acute puerpural mastitis: Clinical and bacteriological studies studies in relation to penicillin therapy. *American Journal of Obstetrics and Gynecology,* 1948, **55**:778-788.

63. Hewitt JH, Coe AW, Parker MT. The detection of methicillin resistance in Staphylococcus aureus. *Journal of Medical Microbiology,* 1969, **2**(4):443-456.

64. Hogan C. Mastitis and breastfeeding [letter]. *Australian Family Physician,* 1994, **23**(1):77-77.

65. Hoyle D, Gottlieb E. Blocked ducts. *New Generation,* 1982, **1**(1):16-17.

66. Huai-Chin H. Puerperal mastitis treated with Fructus gleditsiae (a report of 43 cases). *Chinese Medical Journal,* 1973, **11**:152.

67. Hughes LE, Mansel RE, Webster DJT. Infection of the breast. *Benign disorders and diseases of the breast*. London, Baillière Tindal, 1989:143-149.

68. Huysmans-Evers AGM, Rottinghuis H, Mudde HAL et al. Control of puerperal mastitis in a maternity ward. *Nederlands Tijdschrift Voor Geneeskunde,* 1964, **108**:1903-1905.

69. Illingworth RS, Stone DG. Self-demand feeding in a maternity Unit. *Lancet,* 1952, **I**:683-687.

70. Inch S, Fisher C. Antiseptic sprays and nipple trauma [letter]. *Practitioner,* 1986, **230**(1422):1037-1038.

71. Inch S, Fisher C. Mastitis: infection or inflammation? *Practitioner,* 1995, **239**(1553):472-476.

72. Inch S, Holloway, St Barts. *Incidence of mastitis among breastfeeding women from selected Oxfordshire general practitioners practices 1993-1994*. 1996 (Unpublished data).

73. Ingelman-Sundberg A. Early puerperal breast engorgement. *Acta Paediatrica Scandinavica,* 1958, **32**:399-402.

74. Isbister C. Acute mastitis: a study of 28 cases. *Medical Journal of Australia,* 1952, 801-808.

75. Jeffrey JS. Treatment of acute puerpural mastitis. *Edinburgh Medical Journal,* 1947, **54**:442-446.

76. John G, Nduati R, et.al. Correlates of perinatal HIV-1 transmission in the Kenyan breastfeeding study. 1999. (Abstract 13ET5-1. XIth International Conference on AIDS and STDs in Africa, September 1999, Lusaka, Zambia.).

77. Johnsén C. Inflammatory lesion of the breast. In: Strömbeck JO, Rosato FE, eds. *Surgery of the breast*. Stuttgart, Georg Thieme Verlag, 1986:48-52.

78. Jonsson S, Pulkkinen MO. Mastitis today: incidence, prevention and treatment. *Annales Chirurgiae Et Gynaecologiae.Supplementum,* 1994, **208**:84-87.

79. Karstrup S, Solvig J, Nolsoe CP et al. Acute Puerperal breast abscess: US guided drainage. *Radiology,* 1993, **188**:807-809.

80. Katzman DK, Wald ER. Staphylococcal scalded skin syndrome in a breast-fed infant. *Pediatric Infectious Disease Journal,* 1987, **6**(3):295-296.

81. Kaufmann R, Foxman B. Mastitis among lactating women: Occurrence and risk factors. *Social Science and Medicine,* 1991, **33**(6):701-705.

82. Kenny JF. Recurrent group B streptococcal disease in an infant associated with the ingestion of infected mother's milk [letter]. *Journal of Pediatrics,* 1977, **91**(1):158-159.

83. Kinlay JR, O'Connell DL, Kinlay S. Incidence of mastitis in breastfeeding women during the six months after delivery: a prospective cohort study. *Medical Journal of Australia,* 1998, **169**(6):310-312.

84. Knight ICS, Nolan B. Breast abscess. *British Medical Journal,* 1959, **1**:1224-1226.

85. Kochenour NK. Lactation suppression. *Clinical Obstetrics and Gynecology,* 1980, **23**(4):1045-1059.

86. La Leche League International. *The Womanly Art of Breastfeeding,* 6th ed. P.O. Box 4079, Schaumburg, IIllinois 60168-4079, USA, 1998.

87. Lacey RW, Mitchell AA. Gentamicin-resistant Staphylococcus aureus. *Lancet,* 1969, **2**(7635):1425-1426.

88. Larson E. A causal link between handwashing and risk of infection? Examination of the evidence. *Infection Control,* 1988, **9**(1):28-36.

89. Larson E. Handwashing: it's essential--even when you use gloves. *American Journal of Nursing,* 1989, **89**(7):934-939.

90. Larson E, Zuill R, Zier V et al. Storage of human breast milk. *Infection Control,* 1984, **5**(3):127-30¡.

91. Lauwers J, Shinskie D. *Counselling the nursing mother: a lactation consultant's guide,* 3rd ed. Boston, Jones and Bartlett, 2000.

92. Lawlor-Smith C. Treating mastitis [letter; comment]. *Australian Family Physician,* 1994, **23**(5):978-979.

93. Lawrence RA. The puerperium, breastfeeding, and breast milk. *Current Opinion in Obstetrics and Gynecology,* 1990, **2**(1):23-30.

94. Lawrence RA. *Breastfeeding - a guide for the medical profession,* 5th ed. St. Louis, CV Mosby, 1999 (pages 273-283).

95. Leary WGJr. Acute puerperal mastitis - a review. *California Medicine,* 1948, **68**:147-151.

96. Light IJ, Walton RL, Sutherland JM et al. Use of bacterial interference to control a staphylococcal nursery outbreak. Deliberate colonization of all infants with the 502A strain of Staphylococcus aureus. *American Journal of Diseases of Children,* 1967, **113**(3):291-300.

97. Livingstone VH, Stringer LJ. The treatment of Staphylococcus Aureus infected sore nipples: a randomized comparative study. *Journal of Human Lactation,* 1999, **15**:241-246.

98. Lodge J, Taylor P, Kennedy P et al. Blocked ducts - revisited. *New Generation,* 1982, **1**(4):16-19.

99. Marmet C, Shell E, Marmet R. Neonatal frenotomy may be necessary to correct breastfeeding problems. *Journal of Human Lactation,* 1990, **6**(3):117-121.

100. Marshall BR, Hepper JK, Zirbel CC. Sporadic puerperal mastitis. An infection that need not interrupt lactation. *Journal of the American Medical Association,* 1975, **233**(13):1377-1379.

101. Masaitis NS, Kaempf JW. Developing a frenotomy policy at one medical center: a case study approach. *Journal of Human Lactation,* 1996, **12**(3):229-32¡.

102. Matheson I, Aursnes I, Horgen M et al. Bacteriological findings and clinical symptoms in relation to clinical outcome in puerperal mastitis. *Acta Obstetrica Et Gynecologica Scandinavica,* 1988, **67**(8):723-726.

103. Melish ME, Campbell KA. Coagulase-positive staphylococcal infections: Breast abcesses. In: Feigin RD, Cherry JD, eds. *Textbook of pediatric infectious diseases*, 4th ed. Philadelphia, W.B. Saunders Co., 1998:1039-1066.

104. Melish ME, Campbell KA. Coagulase-positive staphylococcal infections: Staphylococcal scalded skin syndrome. In: Feigin RD, Cherry JD, eds. *Textbook of infectious diseases*, 4th ed. Philadelphia, W.B. Saunders Co., 1998:1055-1057.

105. Minchin MK. *Breastfeeding Matters*, 4th ed. Australia, Alma Publications, 1998 (pages 151-165).

106. Miranda R, Saravia NG, Ackerman R et al. Effect of maternal nutritional status on immunological substances in human colostrum and milk. *American Journal of Clinical Nutrition,* 1983, **37**(4):632-640.

107. Mohrbacher M, Stock J. *The breastfeeding answer book* (revised ed. 1997) La Leche League International, 1400 N. Meachem Road, Schaumburg, IL 60168-4079, USA., 1997.

108. Montgomery TL, Wise RI, Lang WR et al. A study of staphylococcal colonization of postpartum mothers and newborn infants. Comparison of central care and rooming-in. *American Journal of Obstetrics and Gynecology,* 1959, **78**:1227-1233.

109. Moon AA, Gilbert B. A study of acute mastitis of the puerperium. *Journal of Obstetrics and Gynaecology of the British Commonwealth,* 1935, **42**:268-282.

110. Moon JL, Humenick SS. Breast engorgement: contributing variables and variables amenable to nursing intervention. *Journal of Obstetric, Gynecologic, and Neonatal Nursing,* 1989, **18**(4):309-315.

111. Morton JA. The clinical usefulness of breastmilk sodium in the assessment of lactogenesis. *Pediatrics,* 1994, **93**:802.

112. Murphy DP, SenGupta SK, Muthaiah AC. Benign breast disease in Papua New Guinea. *Papua New Guinea Medical Journal,* 1992, **35**(2):101-105.

113. Naish C. *Breastfeeding*. London, Lloyd-Luke, 1956.

114. Naish FC. *Breast feeding. A guide to the natural feeding of infants*. Oxford, Oxford Medical Publications, 1948.

115. Nelson J. *An essay on Goverment of Children under three general heads; viz, health, manners and education*. London, 1753.

116. Neville MC, Allen JC, Archer PC et al. Studies in human lactation: milk volume and nutrient composition during weaning and lactogenesis. *American Journal of Clinical Nutrition,* 1991, **54**:81-92.

117. Newton M, Newton N. Breast abscess: A result of lactation failure. *Surgery, Gynecology and Obstetrics,* 1950, **91**:651-655.

118. Nicholson W, Yuen HP. A study of breastfeeding rates at a large Australian obstetric hospital. *Aust NZ J ObstetGynaecol,* 1995, **35**:393-397.

119. Niebyl JR, Spence MR, Parmley TH. Sporadic (nonepidemic) puerperal mastitis. *Journal of Reproductive Medicine,* 1978, **20**(2):97-100.

120. Novy MJ. Disorders of lactation. In: Benson RC, eds. *Obstetric and gynecologic diagnosis and treatment*. Los Altos, Lange Medical Publications, 1984:864-867.

121. Nwankwo MU, Offor E, Okolo AA et al. Bacterial growth in expressed breast-milk. *Annals of Tropical Paediatrics,* 1988, **8**(2):92-95.

122. Ogle KS, Davis S. Mastitis in lactating women [clinical conference]. *Journal of Family Practice,* 1988, **26**(2):139-144.

123. Opri F. Mammary mycoses. *Chemotherapy,* 1982, **28**(Suppl 1):61-65.

124. Oseas R, Yang HH, Baehner RL et al. Lactoferrin: a promoter of polymorphonuclear leukocyte adhesiveness. *Blood,* 1981, **57**(5):939-45¡.

125. Paape MJ, Wergin WP, Guidry AJ et al. Leukocytes--second line of defense against invading mastitis pathogens. *Journal of Dairy Science,* 1979, **62**(1):135-153.

126. Peaker M. Ion and water transport in the mammary gland. In: Larson BL, eds. *Lactation: A comprehensive treatise*. New York, Academic Press, 1978:437.

127. Peters F, Flick-Fillies D, Ebel S. [Hand disinfection as the central factor in prevention of puerperal mastitis. Clinical study and results of a survey]. *Geburtshilfe Und Frauenheilkunde,* 1992, **52**(2):117-120.

128. Pittard WB, Anderson DM, Cerutti ER et al. Bacteriostatic qualities of human milk. *Journal of Pediatrics,* 1985, **107**(2):240-243.

129. Porthouse A, Brown DF, Smith RG et al. Gentamicin resistance in Staphylococcus aureus. *Lancet,* 1976, **1**(7949):20-21.

130. Prentice A, Prentice AM. Unilateral breast dysfunction in lactating Gambian women. *Annals of Tropical Paediatrics,* 1984, **4**(1):19-23.

131. Prentice A, Prentice AM, Lamb WH. Mastitis in rural Gambian mothers and the protection of the breast by milk antimicrobial factors. *Transactions of the Royal Society of Tropical Medicine and Hygiene,* 1985, **79**(1):90-95.

132. Prosser CG, Hartmann PE. Comparison of mammary gland function during the ovulatory menstrual cycle and acute breast inflammation in women. *Australian Journal of Experimental Biology and Medical Science,* 1983, **61**(Pt 3):277-286.

133. Purves C, Browne L. Blocked ducts - revisited. *New Generation,* 1982, **1**(4):16-19.

134. Qureshi F. The acute breast abscess. Practical procedures. *Australian Family Physician,* 1982, **11**(3):213-214.

135. Ravenbolt RT, Wright P, Mulhern M. Epidemiology and prevention of nursery derived staphylococcal disease. *New England Journal of Medicine,* 1957, **257**:789-795.

136. Raymond J, Bingen E, Brahimi N et al. Staphylococcal scalded skin syndrome in a neonate. *European Journal of Clinical Microbiology and Infectious Diseases,* 1997, **16**(6):453-454.

137. Rench MA, Baker CJ. Group B streptococcal breast abscess in a mother and mastitis in her infant. *Obstetrics and Gynecology,* 1989, **73**(5 Pt 2):875-877.

138. Righard L, Alade MO. Sucking technique and its effect on success of breastfeeding. *Birth,* 1992, **19**(4):185-189.

139. Riordan J, Auerbach KG. *Breastfeeding and Human lactation,* 2nd ed. Sudbury, Jones & Bartlett, 1999.

140. Riordan JM, Nichols FH. A descriptive study of lactation mastitis in long-term breastfeeding women. *Journal of Human Lactation,* 1990, **6**(2):53-58.

141. Robbins MJ. Breast-feeding in the face of adversity. *MCN; American Journal of Maternal Child Nursing,* 1992, **17**(5):242-245.

142. Roberts K. A comparison of chilled and room temperature cabbage leaves in treating breast engorgement. *Journal of Human Lactation,* 1995, **11**(3):191-194.

143. Roberts K. A comparison of chilled cabbage leaves and chilled gelpaks in reducing breast engorgement. *Journal of Human Lactation,* 1995, **11**(1):17-20.

144. Scherer P. Bestimmung des Säure- und Keimgehaltes bei roher und sterilisierter Frauenmilch unter Berücksichtigung verschiedener Aufbewahrung [Determination of the acid and germ content of raw and sterilised breastmilk under different storage conditions]. *Zeitschrift Für Hygiene Und Infektionskrankheiten,* 1951, **132**:217-222.

145. Scholefield JH, Duncan JL, Rogers K. Review of a hospital experience of breast abscesses. *British Journal of Surgery,* 1987, **74**(6):469-470.

146. Schonheyder H. [Salmonella mastitis]. *Ugeskrift for Laeger,* 1980, **142**(18):1156-1159.

147. Schreiner RL, Coates T, Shackelford PG. Possible breast milk transmission of group B streptococcal infection [letter]. *Journal of Pediatrics,* 1977, **91**(1):159-159.

148. Semba RD, Kumwenda N, Hoover DR et al. Human Immunodeficiency Virus Load in Breast Milk, Mastitis, and Mother-to-Child Transmission of Human Immunodeficiency Virus Type 1. *Journal of Infectious Diseases,* 1999, **180**:000.

149. Semba RD, Kumwenda N, Taha ET. Mastitis and immunological factors in breast milk of human immunodeficiency virus-infected women. *Journal of Human Lactation,* 1999, **15**:301-306.

150. Semba RD, Neville MC. Breast-feeding, mastitis and HIV transmission: nutritional implications. *Nutrition Reviews,* 1999, **57**(5):146-153.

151. Shinefield HR, Ribble JC, Boris M. Bacterial interference: its effect on nursery aquired infection with Staphylococcus aureus. *American Journal of Diseases of Children,* 1963, **105**:646-654.

152. Sinclair CM. *Fats in Human Milk.*Topics in Breastfeeding, No. Set IV, 1992. Lactation Resource Centre, Nursing Mothers' Association of Australia Library, P.O. Box 4000, Glen Iris, Victoria 3146, Australia.

153. Soder O. Isolation of interleukin-1 from human milk. *International Archives of Allergy and Applied Immunology,* 1987, **83**(1):19-23.

154. Soltau DHK, Hatcher GW. Some observations on the aetiology of breast abscess in the puerperium. *British Medical Journal,* 1960, **1**:1603.

155. Speller DC, Raghunath D, Stephens M et al. Epidemic infection by a gentamicin-resistant Staphylococcus aureus in three hospitals. *Lancet,* 1976, **1**(7957):464-466.

156. Stanway P. Coping with mastitis. *New Generation,* 1988, **7**(2):36.

157. Taylor MD, Way S. Penicillin treatment of acute puerperal mastitis. *British Medical Journal,* 1946, 731-732.

158. Thomas V, Shelokov A, Forland M. Antibody-coated bacteria in the urine and the site of urinary-tract infection. *New England Journal of Medicine,* 1974, **290**(11):588-590.

159. Thomassen P, Johansson V, Wassberg C et al. Breastfeeding, pain and infection. *Gynaecological and Obstetric Investigation,* 1998, **46**:73-74.

160. Thomsen AC. Infectious mastitis and occurrence of antibody-coated bacteria in milk. *American Journal of Obstetrics and Gynecology,* 1982, **144**(3):350-351.

161. Thomsen AC, Espersen T, Maigaard S. Course and treatment of milk stasis, noninfectious inflammation of the breast, and infectious mastitis in nursing women. *American Journal of Obstetrics and Gynecology,* 1984, **149**(5):492-495.

162. Thomsen AC, Hansen KB, Moller BR. Leukocyte counts and microbiologic cultivation in the diagnosis of puerperal mastitis. *American Journal of Obstetrics and Gynecology,* 1983, **146**(8):938-941.

163. Turnbridge J. What to use instead of Flucloxacillin (editorial). *Australian Prescriber,* 1995, **18**(3):54-55.

164. Victora CG, Behague DP, Barros FC et al. Pacifier use and short breastfeeding duration: cause, consequence, or coincidence? *Pediatrics,* 1997, **99**(3):445-453.

165. Walia HS, Abraham TK, Shaikh H. Fungal mastitis. Case report. *Acta Chirurgica Scandinavica,* 1987, **153**(2):133-135.

166. Walker G. When breastfeeding had to stop. *Nursing Times,* 1988, **84**(39):42-3¡.

167. Walker M. *Mastitis* Lactation Consultant Series 2, No. 298-2. La Leche League International, 1400 N. Meachem Road, Schaumburg, IL 60168-4079, USA, 1999.

168. Waller H. The early failure of breastfeeding. *Archives of Disease in Childhood,* 1946, **21**:1-12.

169. Waller HK. Clinical studies in lactation. 1938…, 115-135. London, Heinemann Publ.

170. Walsh A. Acute mastitis. *Lancet,* 1949, **2**:635-639.

171. Whitehead RG, Rowland MG, Hutton M et al. Factors influencing lactation performance in rural Gambian mothers. *Lancet,* 1978, **2**(8082):178-181.

172. Wilde CJ, Prentice A, Peaker M. Breast-feeding: matching supply with demand in human lactation. *Proceedings of the Nutrition Society,* 1995, **54**(2):401-406.

173. Williams CL, Stancel GM. Estrogens and progesterins. In: Limbird L, eds. *Goodman & Gilman's The Pharmacological Basis of Therapeutics*, 9th ed. New York, McGraw-Hill, 1995:1411-1440.

174. Williams RC, Gibbons RJ. Inhibition of bacterial adherence by secretory immunoglobulin A: a mechanism of antigen disposal. *Science,* 1972, **177**(50):697-699.

175. Willumsen JF, Filteau SM, Coutsoudis A et al. Subclinical mastitis as a risk factor for mother-infant HIV transmission. 1999. Kotelzo et al (eds). Short and long term effects of breastfeeding, Plenum Press, Germany, 2000.

176. Woolridge M. The 'anatomy' of infant sucking. *Midwifery,* 1986, **2**:164-171.

177. Woolridge MW. The aetiology of sore nipples. *Midwifery,* 1986, **2**:172-176.

178. World Health Organization. *Breastfeeding counselling: A training course*. Geneva, WHO/UNICEF, 1993 (documents WHO/CDR/93.3, 4, 5, and 6; World Health Organization, 1211 Geneva 27, Switzerland).

179. World Health Organization. *Violence against women*. Geneva, WHO, 1997 (document WHO/FRH/WHD/97.8; World Health Organization, 1211 Geneva 27, Switzerland).

180. World Health Organization. *HIV and infant feeding: guidelines for decision makers*. 1998 (document WHO/FRH/NUT/CHD/98.1; World Health Organization, 1211 Geneva 27, Switzerland).

181. World Health Organization. *Relactation: a review of experience and recommendation for practice*. Geneva, WHO, 1998 (document WHO/CHS/CAH/98.14; World Health Organization, 1211 Geneva 27, Switzerland).

182. World Health Organization. *HIV and infant feeding counselling: a training course*. Geneva, WHO, 2000 (document WHO/FCH/CAH/00.13; World Health Organization, 1211 Geneva 27, Switzerland).

183. Wright J. Bacteriology of the collection and preservation of human milk. *Lancet,* 1947, **2**:121-124.

184. Wyatt RG, Mata LJ. Bacteria in colostrum and milk of Guatemalan Indian women. *Journal of Tropical Pediatrics,* 1969, **15**(4):159-162.

185. Wysham DN, Mulhern ME, Navarre GC. Staphyloccocal infections in an obstetric unit, II. Epidemic studies of puerperal mastitis. *New England Journal of Medicine,* 1957, **257**:304-308.