

Mastitis Control in Dairy Herds

2nd Edition



Roger Blowey and Peter Edmondson

Mastitis Control in Dairy Herds, 2nd Edition

The authors would like to thank
Jane Upton for supplying the line drawings.

Mastitis Control in Dairy Herds, 2nd Edition

Roger Blowey

BSc, BVSc, FRCVS
*Wood Veterinary Group
The Animal Hospital
Gloucester
UK*

and

Peter Edmondson

MVB, Cert. CHP, Dip. ECBHM, FRCVS
*Shepton Veterinary Group
Shepton Mallet
Somerset
UK*

CABI is a trading name of CAB International

CABI Head Office
Nosworthy Way
Wallingford
Oxfordshire OX10 8DE
UK

CABI North American Office
875 Massachusetts Avenue
7th Floor
Cambridge, MA 02139
USA

Tel: +44 (0)1491 832111
Fax: +44 (0)1491 833508
E-mail: cabi@cabi.org
Website: www.cabi.org

Tel: +1 617 395 4056
Fax: +1 617 354 6875
E-mail: cabi-nao@cabi.org

© CAB International 2010. All rights reserved. No part of this publication may be reproduced in any form or by any means, electronically, mechanically, by photocopying, recording or otherwise, without the prior permission of the copyright owners.

First edition published in 1995 by Farming Press Ltd.
ISBN 978 085236 314 0

A catalogue record for this book is available from the British Library, London, UK.

Library of Congress Cataloging-in-Publication Data

Blowey, R.W. (Roger William)
Mastitis control in dairy herds/Roger Blowey and Peter Edmondson. -- 2nd ed.
p. cm.
Includes bibliographical references and index.
ISBN 978-1-84593-550-4 (alk. paper)
1. Mastitis--Prevention. I. Edmondson, Peter, 1958- II. Title.

SF976.M3B56 2010
630.2'142--dc22

2009022137

ISBN-13: 978 1 84593 550 4

Commissioning editor: Sarah Hulbert
Production editor: Fiona Harrison

Typeset by MRM Graphics, Ltd, Winslow, UK.
Printed and bound in the UK by Butler Tanner & Dennis.



Mixed Sources
Product group from well-managed
forests and other controlled sources
www.fsc.org Cert no. SGS-COC-005091
© 1996 Forest Stewardship Council

Contents

1	Introduction	1
2	Structure of Teats and Udder and Mechanisms of Milk Synthesis	5
3	Teat and Udder Defences Against Mastitis	20
4	The Mastitis Organisms	33
5	Milking Machines and Mastitis	60
6	The Milking Routine and its Effect on Mastitis	95
7	Teat Disinfection	116
8	The Environment and Mastitis	130
9	Somatic Cell Count	152
10	Bactoscan and Total Bacterial Count (TBC)	171
11	Targets and Monitoring	184
12	Treatment and Dry Cow Therapy	194
13	Summer Mastitis	215
14	Disorders of the Udder and Teats	220
15	Residue Avoidance in Milk	239
16	Best Practice Guides	248
	Appendix: Liner Life Charts	253
	Appendix: Parlour Audit	255
	References and Further Reading	256
	Index	259

This page intentionally left blank

1 Introduction

What is Mastitis?	1
Economics of Mastitis	2
What are Realistic Production Targets for the Future?	3

The aim of this book is to explain the many different factors which lead to mastitis and poor milk quality. If the farmer, vet or herdsman appreciates the way in which mastitis occurs, then he will be in a much better position to understand and implement the control measures required. Mastitis can never be eradicated. This is because environmental infections such as *Escherichia coli* (*E. coli*) will always be present.

It is also highly unlikely that a single all-embracing vaccine will ever be found to suppress the multiplicity of types of infection involved. Control must therefore be based on sound management, and this originates best from a thorough understanding of the principles of the disease involved.

The primary objective of this book is to achieve a thorough understanding of mastitis. If this results in a reduction in the incidence of infection, and in so doing benefits both the economics of dairy farming and the welfare of the cow, then the authors will be well pleased.

What is Mastitis?

Mastitis simply means 'inflammation of the udder'. Most farmers associate mastitis with an inflamed quarter together with a change in the appearance of the milk. These changes

are due to the effect of the cow's inflammatory response to infection. However, mastitis can also occur in the subclinical form. This means that although infection is present in the udder there are no visible external changes to indicate its presence.

Much of the information needed to reduce the incidence of mastitis has been available for the last 30 years. Research work carried out during the Mastitis Field Experiment (MFE) trials at the National Institute for Research into Dairying (NIRD) in the 1960s formed the basis of the important mastitis control measures used today, including the proven five-point plan, which recommended:

1. Treating and recording all clinical cases.
2. Dipping teats in disinfectant after every milking.
3. Dry cow therapy at the end of lactation.
4. Culling chronic mastitis cases.
5. Regular milking machine maintenance.

Over the past 40 years, great progress has been made in reducing cell counts, mainly due to the uptake of the five-point plan by dairy farmers. In the UK, the clinical incidence of mastitis has decreased from 121 cases per 100 cows per year in 1968, to between 40 to 50 in 2009. One case is one quarter affected once.

There are two basic types of mastitis: contagious and environmental. The greatest progress has been in reducing the incidence of contagious mastitis. Cell counts (also referred to as somatic cell counts, or SCCs) relate to the level of contagious infection and so the effect of this progress can be seen in the decrease in the national average cell count of milk in England and Wales from 571,000 (571,000/ml) to around 240,000 in 2009. This is shown in Fig. 1.1.

The aim now is to further reduce contagious mastitis and cell counts, and also to reduce environmental infections. The incidence of environmental mastitis has remained unchanged since 1960. This is largely due to an increase in herd size and higher milk yields. Milk yield is correlated to the speed of milking, and flow rates have doubled over the past 40 years. Over the same period, faster milking speeds have led to a 12-fold increase in mastitis susceptibility.

It is therefore a credit to farmers that they have improved the cow's environment and hygiene sufficiently to have prevented an increase in the clinical mastitis incidence over this period. As yields are likely to increase further in the future, the risk of new infections will continue to rise.

Mastitis leads to a reduction in the useful components of milk and increases the level of undesirable elements. This is, of course, exactly the opposite of what the dairy farmer is trying to achieve. Overall, mastitis results in a less acceptable product

and so the value of this milk is much reduced.

Table 1.1 shows the effect of subclinical mastitis (i.e. raised cell count) on various milk components. It indicates that the yield of lactose and casein is reduced substantially. While the total protein level remains little changed, the level of casein is decreased by up to 20%. This is of great significance to dairy manufacturers, especially cheese makers, as it reduces the manufacturing yield from milk. The changes in butterfat and lactose levels are of great economic significance to the farmer as they make up the basis of his milk price. Mastitis may cause a reduction in butterfat and protein, lowering the price of milk by up to 15%. This will have quite an effect on profit.

Mastitis also produces increased levels of the enzymes lipase and plasmin, which break down milk fat and casein respectively and therefore have a significant effect on manufacturing yield and keeping quality. These elements are of utmost concern to milk buyers and in the future it is possible that milk will be tested for plasmin and lipase and producers penalized for high levels of these enzymes.

Economics of Mastitis

Mastitis affects the farmer economically in two ways: through direct costs and indirect costs.

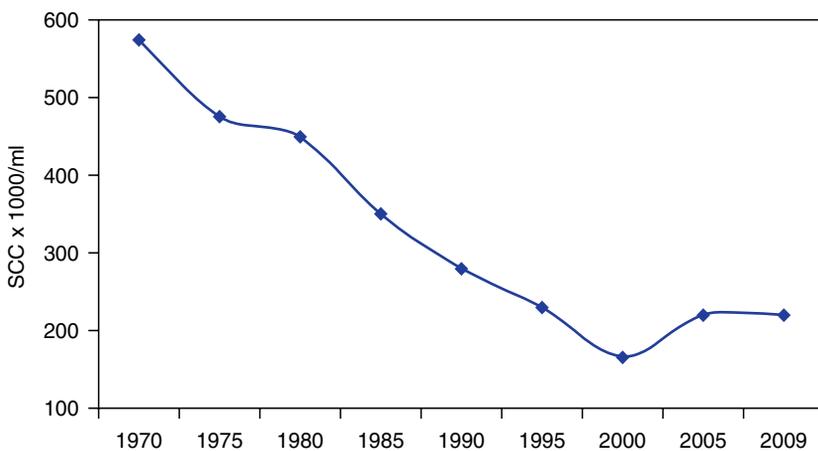


Fig. 1.1. Annual average cell count (level of contagious mastitis) for England and Wales, 1970–2009.

Table 1.1. The effect of mastitis on milk components. (From Philpot and Nickerson, 1991.)

	Components	Effect of subclinical mastitis
Desirable	Total proteins	Decreased slightly
	Casein	Decreased between 6 and 20%
	Lactose	Decreased between 5 and 20%
	Solids not fat (SNF)	Decreased by up to 8%
	Butterfat	Decreased between 4 and 12%
	Calcium	Decreased
	Phosphorus	Decreased
	Potassium	Decreased
	Stability and keeping quality	Decreased
	Taste	Deteriorates and becomes bitter
	Yogurt starter cultures	Inhibited
Undesirable	Plasmin (degrades casein)	Increased
	Lipase (breaks down fat)	Increased
	Immunoglobulins	Increased
	Sodium	Increased – hence the ‘bitter’ taste

Direct costs:

1. Discarded milk.
2. Drug and veterinary costs.

Indirect costs:

1. Penalties because of increased cell count.
2. Decreased milk yield during remainder of lactation due to udder damage and/or subclinical infection.
3. Extra labour requirements for treating and nursing.
4. Higher culling and replacement rates, leading to loss of genetic potential.
5. Deaths.

The costs of a clinical case of mastitis have been quantified: in 2009 it was estimated that the average cost of one case of mastitis was between £100 and £200. An average cost of £125 is a well-accepted figure for 2009.

This work assumed that there were three categories of mastitis: mild, severe and fatal. The most common form of mastitis is the mild case, which responds quickly to farmer treatment. The costs here include intramammary tubes, discarded milk and a reduced yield for the remainder of the lactation. A severe case of mastitis requires veterinary treatment, while not only does a fatal case of mastitis require veterinary treatment but also the cow never returns to the milking herd.

In addition to the cost of mastitis, there are extra risk factors that should be considered. These include high total bacterial counts (TBCs) or Bactoscans, and the risk of antibiotic residues entering the bulk milk supply. Both of these incur financial penalties.

The majority of the losses in high cell count herds are from subclinical infection resulting in depressed production and reduced yields of lactose, casein and butterfat. It is generally accepted that herds with a cell count of 200,000 or less will have no significant production losses due to subclinical infection. For every 100,000 increase in cell count above 200,000, there will be a reduction in yield of 2.5%. This reduction, together with financial penalties imposed for elevated cell counts, can be quite substantial.

The average incidence of clinical mastitis in the United Kingdom in 2009 was between 40 and 50 cases per 100 cows per year, ranging from some herds with levels as low as ten to others with up to 150 cases per 100 cows per year.

What are Realistic Production Targets for the Future?

The consumer and the dairy companies are requiring milk of increasing quality. In the future, dairy companies will continue to

want low Bactoscan and cell counts, above which producers will incur financial penalties. The importance of cell counts to the dairy companies can be seen from the large financial penalties that they are imposing. There is an escalating scale of penalties imposed for producers with high cell count milk, with most companies penalizing farmers with cell counts over 200,000. Some companies penalize farmers up to £300 per cow per year for cell counts over 300,000. Producers with a cell count over 400,000 are unable to sell their milk as it exceeds the EU thresholds for milk quality.

The farmer is therefore encouraged to keep reducing the herd cell count and

Bactoscan, and in so doing will ensure that he receives the premium price for his milk. This also benefits the consumer and the dairy industry, which will have a quality product with a good shelf life, suitable for manufacturing.

With good herd management it is possible to have an incidence of clinical mastitis below 30 cases per 100 cows per year, a herd cell count of under 150,000 and Bactoscans under 20,000/ml. For 'problem' herds this may take several years to achieve. Meeting these goals will improve profitability while ensuring a healthy future for both the dairy farmer and his cows.

2 Structure of Teats and Udder and Mechanisms of Milk Synthesis

Structure of the Udder	5
Development of the Udder	6
First isometric phase	6
First allometric phase	6
Second isometric phase	7
Second allometric phase	7
Suspension of the Udder	7
Rupture of the suspensory apparatus	8
Structure and Function of the Teats	9
Supernumerary teats	9
Functions of the teats	11
Teat size	11
The teat wall	11
Milk let-down	13
Poor milk let-down in heifers	14
Milk Synthesis and How it is Affected by Mastitis	15
Lactose	15
Protein	16
Milk fat	16
Minerals	17
Control of Milk Synthesis	17
Milking frequency	18
Environmental temperature	18
Length of dry period	18

This chapter examines the development and structure of the udder, the structure and function of the teats, and mechanisms of milk synthesis. The aspects of teat function that prevent new infections are discussed in Chapter 3.

Structure of the Udder

As shown in Figs 2.1 and 2.9, milk is produced by the cuboidal cells lining the mam-

mary alveoli deep within the mammary gland. Surrounding the alveoli are myoepithelial or muscle cells (Fig. 2.1). When the stimulus for milk let-down occurs, these cells contract, and this squeezes milk from the alveoli into the ducts. From there milk flows into the gland and teat cisterns where it is ready to be drawn from the udder. In higher-yielding cows particularly, there will, of course, be some milk stored in the ducts, cisterns and teats between milkings. The mechanisms of milk synthesis are described on page 15.

Development of the Udder

The udder (or mammary gland) is derived from a highly modified sweat gland. As such, the inside lining of the teats and ducts of the mammary gland is essentially modified skin.

The development of the udder from the birth of the calf to the start of its first lactation can be divided into four phases:

- First isometric phase.
- First allometric phase.
- Second isometric phase.
- Second allometric phase.

First isometric phase

In the young calf, the growth and development of the udder proceed at the same rate as the rest of the body, and hence the term 'isometric', i.e. growing at the same rate.

First allometric phase

There is then a sudden increase in the growth of the udder, which as a result begins to develop more rapidly than the rest of the body. This phase occurs at approximately 4 to 8 months old, i.e. around puberty, and is particularly associated with peaks of oestrogen occurring each time the heifer comes on heat. Development at

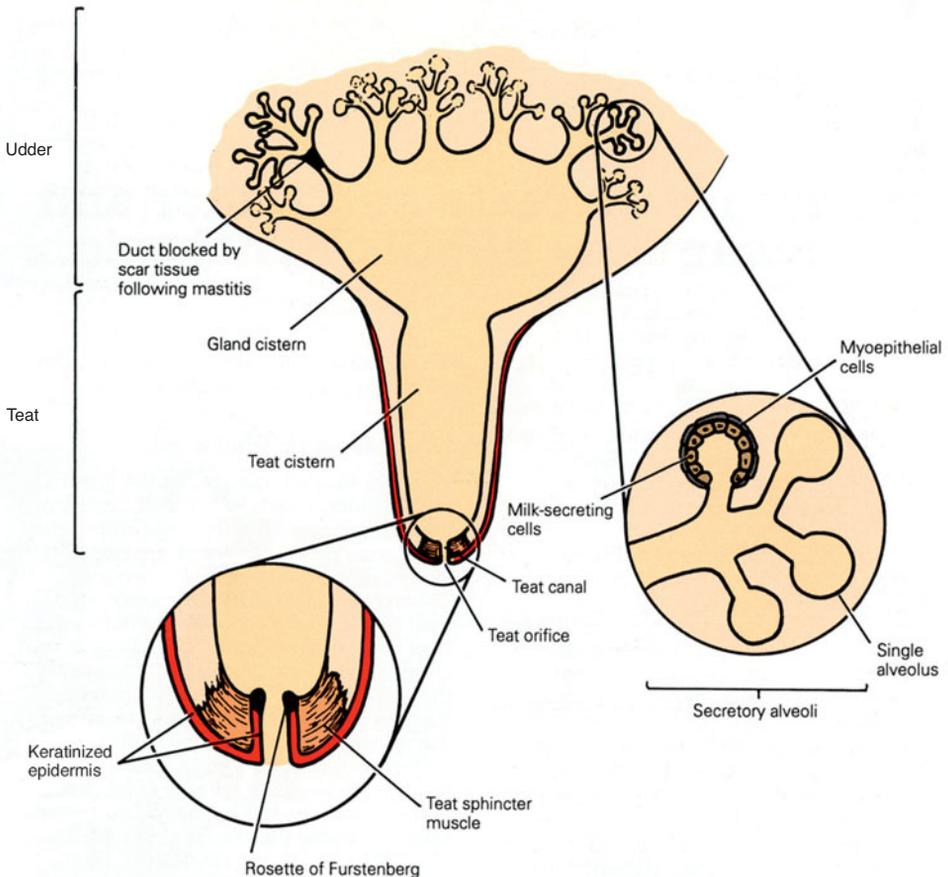


Fig. 2.1. The structure of the udder and teat.

this stage is primarily of the ducts, which lengthen and penetrate the pad of fat that occupies the site of the udder in the prepubertal calf. Overfeeding at this stage, and prior to it, leads to an excessive pad of fat being laid down at the site of the future udder. This can cause depressed yields later in life. For example, in one trial (Harrison *et al.*, 1983), two groups of heifers were reared to produce liveweight gains of 1.1 kg per day (high) and 0.74 kg per day (conventional). Not only did the mammary glands of the conventionally reared heifers weigh more (40% more) but they also contained much more secretory tissue (68% more). Gross overfeeding of young heifers is therefore to be avoided. It is thought that a diet high in forage during rearing stimulates greater rumen development and higher appetite capacity at maturity. Protein intakes should be high (for example, 18% crude protein) and of good quality to promote udder development, but excess intakes of starch should be avoided.

Second isometric phase

From after the onset of puberty until the beginning of pregnancy, the udder again grows at the same rate as the other body organs.

Second allometric phase

Following conception, udder development once again becomes rapid, with the highest growth rate occurring from mid-pregnancy onwards. During this phase the cells of the alveoli especially become more developed and change into a tissue type that is able to secrete milk.

Suspension of the Udder

The udder consists of four separate mammary glands, each with its own distinct teat. There is no flow of milk from one quarter to another, neither is there any significant direct blood flow from one quarter to another. The blood supply to the udder is massive, with some 400 litres of blood flowing through the

udder to produce each litre of milk. Add to this the weight of the secretory tissue and the weight of milk stored and it is easy to see how total udder weights of 50 to 75 kg are obtained. The reason why all milk should be discarded when treating one quarter with antibiotics is that antibiotics may be absorbed from that quarter into the bloodstream, travel around the body and then be deposited back into one of the other untreated quarters. The amount of antibiotic involved is, of course, relatively small, but it may be enough to lead to a bulk tank failure.

The suspension of the udder is very important. It is shown in Fig. 2.2 and consists of the skin, the superficial lateral ligaments, the deep lateral ligaments and the median ligament.

- The skin. This plays only a very minor role.
- The superficial lateral ligaments. These originate from the bony floor of the pelvis and pass down the outside of the udder, especially at the front and the sides of the

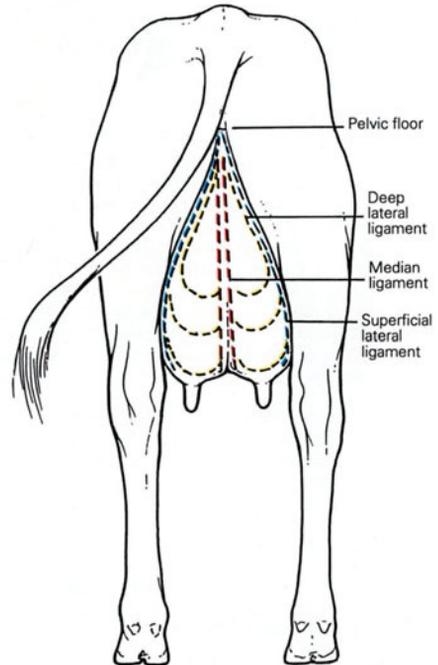


Fig. 2.2. The suspension of the udder.

- udder. They branch forwards attaching to the abdomen (in front) and in the upper leg area to the inner thighs.
- The deep lateral ligaments. These also originate from the floor of the pelvis. Passing down the outside of the udder (but inside the superficial ligaments), they send small ‘cups’ across within the mammary gland and these eventually connect to similar branches from the central median ligament. The largest branch sweeps under the base of the udder, just above the teats, to join the median ligament and provides the major suspensory apparatus for the udder.
 - The median ligaments. There are two median ligaments (Fig. 2.2). Both originate from the pelvic floor and associated abdominal wall. They pass down the centre of the udder and the base, where they separate and join the lateral ligaments at the left and right sides. Branches also connect to connective tissue that separates the fore and hind quarters. The median ligaments contain elastic fibres, which allow a degree of ‘give’, providing a shock-absorber effect and allowing the udder to expand as milk accumulates between milkings.

Rupture of the suspensory apparatus

Rupture of the ligaments may occur gradually or spontaneously. The ligaments that most commonly rupture are:

- the median ligaments.
- the deep lateral ligaments.
- the anterior ligaments (i.e. the front part of the superficial and deep ligaments).

On occasion, rupture of the anterior ligament can lead to a large accumulation of blood under the skin just in front of the udder. This is known as a haematoma. Some become infected and lead to a large, stinking abscess.

Rupture of the ligaments may be associated with a variety of factors, the most important of which are the following:

- Age: the elastic tissue in the median liga-

ments especially deteriorates with age.

- Over-engorgement and oedema of the udder (see pages 223–224 for the many causes of udder oedema). This is one good reason why heifers and cows should not be ‘steamed up’ (fed extra concentrates) excessively or be kept overfat before calving.
- Poor conformation: it is important to select for a ‘type’ that has good udder attachment and evenly placed front and rear teats.

Rupture of the median ligaments is probably the most common reason for poor udder suspension. It leads to loss of the ‘cleavage’ between quarters, causing the teats to splay outwards (see Fig. 2.3 and Plate 2.1), making it difficult to attach the milking units. It also often leads to air leakage during milking, especially when the unit is first applied, thus producing teat-end impacts (see pages 79–80) and increasing the mastitis risk. Rupture of the deep lateral ligaments is invariably associated with concurrent rupture of the superficial ligaments and leads to a total drop of the whole udder (see Fig. 2.4 and Plate 2.2). The teats drop to well below hock level and can easily become injured as the cow walks.

Rupture of the anterior ligaments (the front portions of the superficial and deep

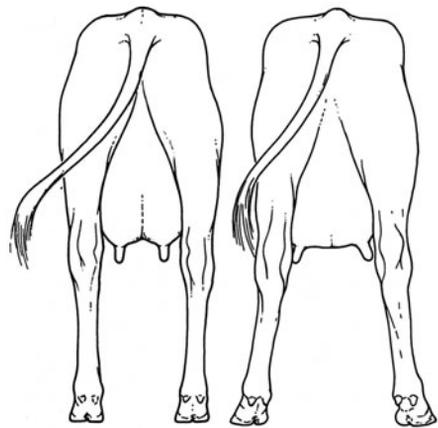


Fig. 2.3. Rupture of the median udder ligaments (right) leads to splaying of the teats and loss of normal udder cleavage (left).



Plate 2.1. Rupture of the median udder ligament, leading to splaying of the teats.

ligaments) occurs less frequently. It is seen as a gross enlargement at the front of the udder (see Fig. 2.5 and Plate 2.3), which often (but not always) leads to a dropping of the front teats. The characteristic feature is that the normal depression at the front of the udder, where the udder joins the abdominal wall, disappears and is replaced with a swelling. Conditions such as haematomas (which are large accumulations of blood under the skin) and rupture of the abdominal wall can sometimes be confused with rupture of the anterior udder ligament. Stretching of the udder suspension is one



Plate 2.2. Rupture of the median and lateral udder ligaments, leading to a total 'drop' of the udder.

reason why around 60% of cows have visually uneven quarters.

Structure and Function of the Teats

As described in the section above, the udder consists of a pad of fat containing many interconnecting tubes, all of which terminate at the same point, namely the teat and gland cisterns. The structure could be compared to a tree. The trunk is the teat and gland cistern, the branches are the lactiferous ducts, and the small leaves at the ends of twigs are the secretory alveoli, small sac-like structures deep within the mammary gland. This is shown in Fig. 2.1. Milk is produced by the cells lining the mammary alveoli, and much of the milk is stored here between milkings.

This section describes the structure and function of the teats, and discusses milk let-The cow has four main teats, with 60% of production coming from the two hind teats. There may be varying numbers of supernumerary teats (extra teats).

Supernumerary teats

Also known as accessory teats, supernumeraries are congenital, i.e. they are present at

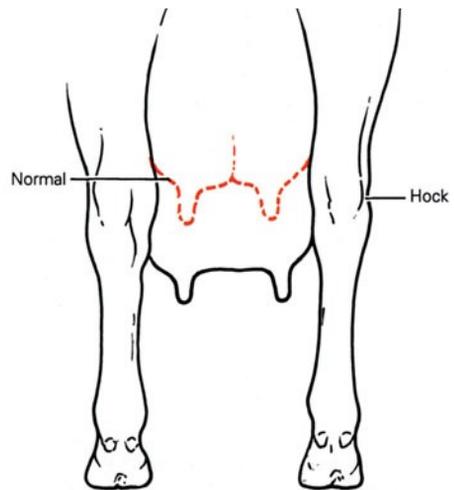


Fig. 2.4. Rupture of the deep lateral udder ligaments leads to the udder dropping to well below hock level.

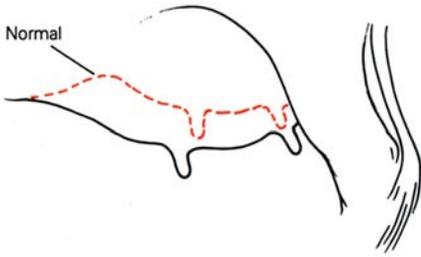


Fig. 2.5. Rupture of the anterior portion of the deep lateral udder ligament is seen less commonly than that of the other ligaments. It leads to a swelling in the front of the udder and the front teats drop.



Plate 2.3. Rupture of the anterior udder ligament – a large swelling appears at the front of the udder.

birth and often inherited. Hence it is advisable not to select heifers from cows with large numbers of supernumerary teats.

These teats are most commonly found at the rear of the udder, behind the two hind teats (Plate 2.4), although they may also be found between the front and rear teats (Plate 2.5), and occasionally attached to an existing teat (Plate 2.6). Supernumeraries attached to full teats need handling with care, as often they have a confluent teat sinus, and removal of the supernumerary can lead to milk leakage from the main teat. Supernumeraries should be removed at the same time as the calf is disbudded. The calf needs to be sitting upright in order to allow a thorough inspection of the udder. If sim-



Plate 2.4. Supernumerary teats are most commonly found behind the back teats.

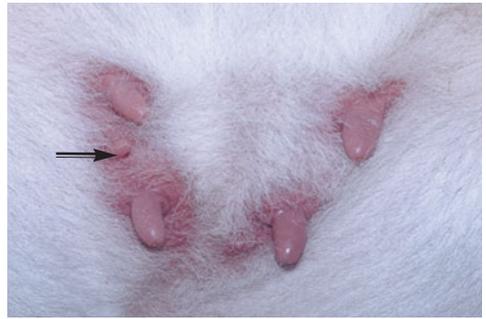


Plate 2.5. Supernumerary teats may also occur between front and back teats, as in this calf.



Plate 2.6. Occasionally a supernumerary teat is attached to a primary teat. The sinus of both teats may be conjoined at the base, making removal more difficult.

ply looked at from between the hind legs while standing, it is very easy to miss those accessory teats which are situated between the main teats. If in any doubt over which

are the supernumeraries, simply roll the teat between your finger and thumb. The supernumerary is much thicker and has no palpable teat cistern. It is most easily removed by lifting the skin under its base with your finger (Plate 2.7), and simply cutting it off, using curved scissors. No anaesthetic is required in animals less than 2 months old. Failure to remove accessory teats has several disadvantages: they are unsightly and affected animals are less saleable; the animals may develop mastitis (especially summer mastitis – see Chapter 13), and also an abscess on the udder; and if situated very near to or at the base of a true teat, supernumeraries may interfere with milking and lead to air leakage, with resulting teat-end impacts (see pages 79–80).



Plate 2.7. To remove a supernumerary teat, sit the calf upright, lift the teat with a finger under a fold of skin and then cut with sharp curved scissors.

Functions of the teats

The most important function of the teat is to convey milk to the young calf. Its use has, of course, been modified to allow hand and machine milking to produce food for man. The teat has an erectile venous plexus at its base, which assists milk flow, and, as described in Chapter 3, the teat, and especially the teat canal, have very important functions in preventing the entry of infection into the udder. Finally, the teat is richly innervated and hence can rapidly convey suckling stimuli to the brain, thus inducing good milk let-down. This rich innervation can occasionally make handling cows with

highly sensitive cut teats somewhat hazardous.

Teat size

As one would expect, this varies enormously, with lengths ranging from 3 to 14 cm. The diameter also varies, from 2 to 4 cm. Teat length increases from the first to the third lactation and then remains constant. On both small, short teats and long, wide teats it may be difficult to get good liner attachment and hence there is an increased risk of liner slippage and teat-end impacts. Teats may be cone-shaped and pointed or cylindrical with a flat tip (Fig. 2.6). Cylindrical teats are said to be less prone to mastitis and are certainly the most common.

Fig. 2.6. Teats may be cone-shaped (left) or cylindrical. Cylindrical teats are said to be less prone to mastitis.

During milking the teat lengthens by some 30 to 40% and also gets thinner. It is suggested that postmilking teat dip should be applied immediately after unit removal, while the teat is still stretched, as then the dip will penetrate the small cracks and folds in the teat before it reduces to its pre-milking length.

The teat wall

The teat wall consists of four layers, each having an important function in mastitis control and/or milk let-down. These layers, passing from the outside of the teat, are the epidermis, the dermis, the muscle and finally the endothelium lining the teat cistern. These structures are all shown in Fig. 2.7.

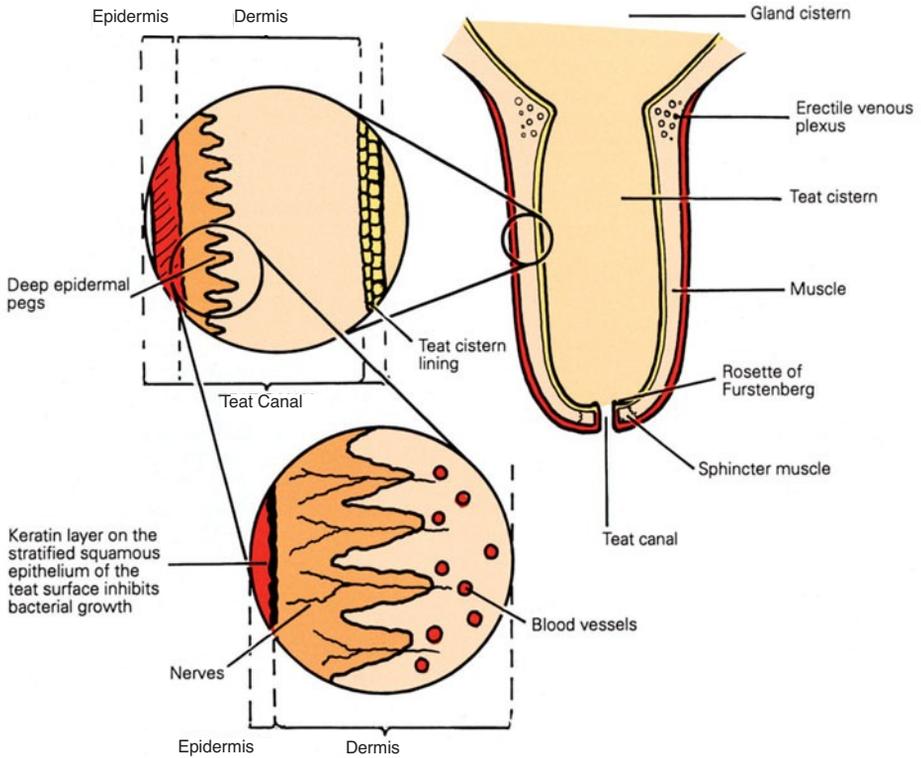


Fig. 2.7. Detailed structure of the teat.

The epidermis

This is the thick outer lining of the skin. Its surface consists of a layer of dead, keratinized cells (Fig. 2.7) which produce a hostile environment for bacterial growth. Keratin is a sulfur-containing protein that impregnates cells, thereby increasing their strength. It is also present in hair, horn and hoof.

All skin is lined with a keratinized epidermis, but teat skin has a particularly thick layer, some four or five times thicker than that of normal skin. It is also very firmly anchored to the underlying dermis (or second layer of skin) by deep epidermal pegs, or papillae. If the skin of the udder is pinched between the finger and thumb, it moves very freely over the underlying tissue. Try doing the same with teat skin: it is firmly

attached. The epidermis of the lips and muzzle of the cow has a similar structure. It is thought that the firm attachment of the epidermis protects the teat from the shear forces involved in both suckling and machine milking and also reduces the chances of injury due to physical trauma. Even so, it is surprising how frequently teats get damaged. Teat skin has no hair follicles, no sweat glands and no sebaceous glands. In practical terms this means that teat skin is particularly susceptible to drying and cracking, which is one reason why an emollient is necessary in teat dips (see also pages 101–102). It also means that there is little or no flow of sebum over teat skin and hence fly repellents should be applied directly on to the teats. Ear tags and pour-on preparations give a very poor flow of insecticide on to the teats.

The dermis (and erectile plexus)

This is the second layer of the teat wall and is the tissue that carries the blood vessels and nerves. However, the fine sensory nerve endings are in the epidermis, which is why exposure of an eroded epidermis (for example, a teat sore) can be so painful. At the base of the teat, adjacent to the udder, the dermis contains the venous erectile plexus. This is a mass of interconnecting blood vessels, which, under the stimulus of suckling or the milk let-down reflex, become engorged to produce a more rigid and turgid teat base. The stiff teat is extremely important in both suckling and machine milking. Suction on a balloon would lead to its collapse. If the base of the teat were to collapse, it would impede the flow of milk from the gland cistern into the teat cistern and hence slow down the milking process. Many herdsmen have probably seen how much blood the erectile venous plexus can hold: a cow with a cut at the tip of the teat bleeds very little, whereas a cut through the venous plexus (Plate 2.8) at the base bleeds profusely and can occasionally lead to serious, or even fatal, blood loss.



Plate 2.8. A cut into the venous erectile plexus at the base of the teat often results in profuse haemorrhage.

The muscles

There is a variety of muscles, which are set in transverse, oblique and longitudinal planes in the dermis of the teat wall. The most important muscle in terms of mastitis control is the circular sphincter muscle around the teat canal. During milking, when

the teat elongates, the canal opens but becomes shorter. After milking, sphincter muscle contraction leads to a shortening of the overall teat and closure of the teat sphincter, but a lengthening of the teat canal. The shortened teats are less prone to physical trauma, and the lengthened and closed canal reduces the risk of entry of bacteria. These changes are shown in Fig. 2.8. As the canal closes, interlocking folds in the lumen press tightly together to provide an improved teat-end seal.

Teat cistern lining

The teat cistern is lined with cuboidal epithelium, that is, a double layer of 'block' cells (Fig. 2.7). In the normal cow these are held tightly together; however, in response to bacterial invasion, they have the ability to move slightly apart, which allows the entry of infection-fighting white blood cells from the small blood vessels beneath (see page 32).

Milk let-down

As will be discussed in more detail in Chapter 6, achieving a good milk let-down prior to unit attachment is essential for rapid parlour throughput. The shorter the time the milking machine is on the cow the better, as this helps to avoid teat-end damage and hence to reduce mastitis levels. The following section describes the mechanics of milk let-down. Chapter 6 describes its practical importance.

There are three phases of milk let-down.

1. Contraction of the myoepithelial or small muscle cells that line the outside of the alveoli (Fig. 2.1). These effectively surround the milk-secreting cells, like a tyre around the rubber inner tube of a car wheel. Contraction of the myoepithelial cells forces milk from the mammary alveoli into the ducts, and hence into the teat and gland cisterns. The herdsman sees this as an enlargement of the udder and engorging of the teats.



Fig. 2.8. Teat changes during milking. After milking, the teat shortens, the canal lengthens and the folds interdigitate to form a tight lipid seal.

2. Engorgement of erectile tissue. Figure 2.7 shows that there is an erectile venous plexus at the base of the teat. When this becomes engorged, it prevents the base of the teat from collapsing during milk flow. If the teat had the structure of a limp, elongated balloon, then one suck from a calf or a milking machine liner and the teat would collapse, leading to a cessation of milk flow. The engorged erectile tissue holds the teat 'open' between the teat cistern and gland cistern, and this allows milk to flow.
3. Relaxation of the teat canal. Between milkings, the circular sphincter muscle surrounding the teat canal pulls it closed and this helps to prevent leakage of milk and entry of infection. The third phase of milk let-down is relaxation of this muscle, to allow milk to flow. Studies have shown that it takes around 15 kPa of pressure to force milk through a closed teat, but only 4–6 kPa when the canal is relaxed for milk let-down. Milk can then flow without causing teat-end damage.

Poor milk let-down in heifers

Poor milk let-down in heifers can be a major problem in some herds, and herdsmen may find that they need to use quite large quantities of oxytocin by injection. This should not be necessary. The following section outlines some of the factors that may be involved.

It is important to make milking a pleasant experience, and not a process associated with fear or pain. The heifers need to know what to expect. If fear is involved, adrenalin will be produced and the let-down mechanisms will be inhibited. For example, it may be a good idea to bring heifers through the parlour before calving so that they know the routine. Applying a good teat dip at this stage will also get them used to being handled, as well as reducing the incidence of dry period infections and subsequent clinical mastitis in early lactation. Do not chase them around the collecting yard to get them into the parlour. They are often last in, when the milker's patience may be waning, so extra care is needed. Make sure that the parlour stall work is the correct size, i.e. that the heifer is not squashed in to the parlour between large cows, making her become uncomfortable.

Take care with the backing gate. The heifers are often at the back of the collecting yard, so if they are being pushed by the backing gate, or, even worse, if it is electrified, then this will inhibit milk let-down when they enter the parlour. Some farms use a separate heifer group, where there will then be less stress from mixing with other animals. Heifers that have a hereditary nervous predisposition may be worse affected. It may be that leaving the calf suckling for too long may make the heifer fret. However, the converse may also be true for some animals, namely that leaving the calf for long enough gets the heifer used to milk let-down and to being milked, even if it is only by the calf.

Excess udder oedema can be a problem, as this is painful and will reduce milk let-down. Overfeeding and insufficient exercise precalving are predisposing factors. Some consider that feeding the heifer will help to take her mind off the milking machine, but many farms no longer do this. It is, of course, vital to ensure that udder preparation has been maximized and that the heifer is well stimulated before unit application. This means going through the full procedure of predip, foremilk, wipe and dry, described in Chapter 6, before the unit is applied. Some farms claim that an initial udder massage with a warm cloth helps, and others that extra comfort from rubber flooring in the parlour may help. One machine manufacturer has an initial rapid 'stimulation pulsation' phase, run at lower vacuum, to try to stimulate milk let-down before unit application.

It is difficult to know how long to leave the unit on a freshly calved heifer if she is not letting her milk down. A suggested routine for the first few milkings is:

1. Heifer taken gently into parlour, apply full udder prep routine, and then unit on. If no milk, take off after 1–2 min max.
2. Repeat for next two milkings, doing your best to optimize the let-down response, perhaps by manual massage of the udder.
3. If there is still no milk let-down, at the fourth milking inject oxytocin as soon as she enters the parlour, so that she associates milk let-down with udder prep, and not with unit on.
4. Many farms try 2.0 ml (depending on its strength) oxytocin for four milkings, then

1.0 ml for the next two milkings, then 0.5 ml for two (provided this low dose still works), then try without.

Poor let-down in heifers is a very variable condition, at least partly associated with the temperament of the heifer herself, and, although the above protocol may be adhered to quite carefully, there will always be one or two animals that do not seem to respond. There will be no 'one size fits all' effect, and it may be necessary to try a range of approaches before one works with a particular batch of heifers.

Milk Synthesis and How it is Affected by Mastitis

Milk is synthesized in cells lining the alveoli, the small sacs at the very end of the ducts deep within the udder (see Figs 2.1 and 2.9). The average composition of milk is shown in Table 2.1.

Colostrum is much more concentrated than milk, having twice the level of total solids (25%) and a very much higher level of protein (15%) due to the high level of antibody present. This is why heating colostrum leads to its coagulation and why Dairy Regulations state that milk should be discarded for the first 4 days after calving.

Table 2.1. Approximate composition of milk from Friesian/Holstein cows.

Component	Amount
Total solids	12.5%
Protein	3.3%
Casein	2.9%
Lactose	4.8%
Ash	0.7%
Calcium	0.12%
Phosphorus	0.09%
Immunoglobulins	1.0%
Vitamin A ($\mu\text{g/g}$ fat)	8
Vitamin D ($\mu\text{g/g}$ fat)	15
Vitamin E ($\mu\text{g/g}$ fat)	20
Water	87.5%

Lactose

Glucose is produced in the liver, primarily from propionate, a product of rumen fer-

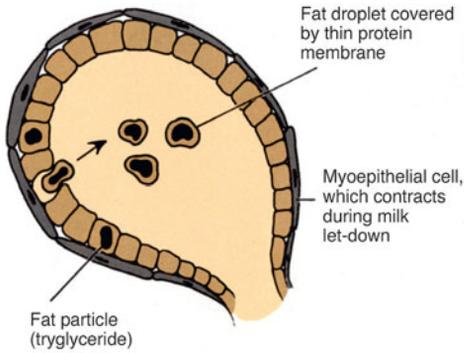


Fig. 2.9. The synthesis of milk fat droplets in the alveolus.

mentation. After it is transferred to the udder, part of the glucose is converted into another simple sugar, galactose. Next, one molecule of glucose combines with one of galactose to produce lactose. Lactose is known as a disaccharide (i.e. two monosaccharide sugars conjoined). In summary:

- Liver: propionate to glucose.
- Udder: glucose to galactose.
- Udder: glucose + galactose = lactose.

Lactose is the main osmotic determinant of milk (the factor governing the concentration of its components in solution). To maintain milk at the same concentration as blood, lactose increases and decreases as the concentration of the other milk components varies. However, the pH of milk is slightly lower than that of blood (i.e. more acidic):

- pH of blood = 7.4
- pH of milk = 6.7

This difference may be used to attract drugs such as erythromycin, trimethoprim, tylosin and penethamate into the mammary gland, as lower pH solutions are drawn to those with higher pH. If lactose concentrations in the udder fall (as occurs with mastitis), then sodium and chloride levels increase to maintain the osmotic pressure of the milk. This is one of the causes of the bitter and slightly salty taste of mastitic milk. (Some farmers occasionally taste the milk of cows they are intending to purchase, in an attempt to identify the slightly salty flavour of mastitis.)

These changes can also be used to help assess mastitis status by electrical conductivity measurements, since sodium and chloride are much better conductors of electricity than lactose.

Protein

The majority of protein in milk is in the form of casein. Amino acids are transported to the udder via the bloodstream and transformed into casein by the mammary alveolar cells. Once formed, casein is extruded from these cells in a mechanism similar to the fat droplets shown in Fig. 2.9.

Surprisingly, it is the energy content of the diet that has the major effect on the casein content of milk. Dietary protein has relatively little influence on milk protein content. Other types of protein present in milk in small quantities are albumin and globulins. These are transferred directly from the blood into milk. Mastitic milk has a reduced casein content but increased levels of albumin and globulin. The total protein content of the milk may remain constant therefore, but the milk is of much poorer quality, particularly for manufacture. This is because the coagulation of casein is very important as part of the starting process for cheese and yogurt production. In addition, mastitic milk contains increased levels of the enzyme plasmin, which decomposes casein in stored milk. Unfortunately, plasmin is not destroyed by pasteurization and it remains active even at 4°C (the storage temperature in supermarkets). Mastitic milk will therefore continue to be degraded even following pasteurization and storage at 4°C; this explains why manufacturers are prepared to pay a premium for low cell count milk.

Milk fat

Milk fat is formed in the udder secretory cells when fatty acids are combined with glycerol and converted into a neutral form of fat called triglyceride.



Fatty acids are derived from three main sources:

- Body fat (50% of total fatty acids). Hence body condition score is an important determinant of milk fat levels, especially in early lactation.
- Dietary fat, especially long-chain fatty acids (those which are solid at room temperature and are components of butter and lard). The use of protected fat (i.e. fat that has been treated so that it can pass through the rumen unchanged) can therefore increase the butterfat content of milk. Conversely, short-chain and polyunsaturated fatty acids in the diet can lead to a decrease in milk fat content.
- Finally, fatty acids are synthesized in the udder from acetate, which is absorbed as a product of rumen fermentation. High-fibre diets, which promote increased levels of acetate in the rumen, will therefore lead to an increase in milk fat production.

Small particles of milk fat (triglyceride) are extruded from the secretory cells in the alveoli and are covered by a thin protein membrane before passing into the milk (Fig. 2.9).

Besides the enzyme plasmin, mastitic milk also has an increased level of the enzyme lipase. This leads to degradation of the milk fat into its fatty acid components and thus imparts a rancid flavour to the milk. Increased levels of fatty acids can inhibit starter cultures used in cheese and yoghurt manufacture, and can also impart a rancid flavour to these products.

In summary, mastitic and high cell count milk is of poorer quality because:

1. Its casein content is lower, and hence cheese manufacture yield per 1000 kg of milk is reduced.
2. Plasmin (which degrades casein) levels are higher, and plasmin remains active after pasteurization.
3. Lipase levels increase, inhibiting yogurt starter cultures, and may impart an adverse flavour.

Minerals

The minerals in milk are derived directly from the blood. Calcium is actively secreted in association with casein.

Control of Milk Synthesis

The rate of milk synthesis, and hence the level of yield, is controlled by a number of factors. These include: diet and factors that influence feed intake; hormones, such as prolactin and BST (bovine somatotrophin); and frequency of removal of milk from the udder, i.e. milking frequency. Diet and management factors affecting feed intake clearly determine the rate at which nutrients arrive at the udder to be used for milk synthesis, and are major determinants of milk production. A discussion of these factors is outside the scope of this book.

In most mammals, initiation of lactation and continued milk production are controlled by the hormone prolactin. In the cow, however, continued milk secretion is influenced by a complex interaction of steroids, thyroid hormone and growth hormone, the latter being more commonly known as bovine somatotrophin (BST). BST is a natural hormone synthesized by the pituitary gland, a small organ at the base of the brain. Higher-yielding cows have more BST circulating in their blood than lower-yielding cows, and cows at peak yield more than late lactation animals. BST can now be produced synthetically and, at the dose rate currently being suggested, increases yields by 10–20%, i.e. 4–6 litres per day. BST alters the cow's metabolism so that a greater proportion of her food is used for milk production, thus making her more efficient. Some 4–6 weeks after starting dosing and after an initial increase in yield, there is an increase in food intake and appetite. In many countries, the use of BST has been prohibited as a result of consumer pressure, or on the grounds of food safety.

Milking frequency

Increased frequency of milking also increases yield. Changing from twice to three times daily will increase production by around 10–15% in cows and 15–20% in heifers. Because of the flatter lactation curve it produces, three times a day milking has to be continued to the end of lactation to obtain its full beneficial effect. Reducing milking frequency decreases yield. For example, if cows are only milked once a day, yields may fall by up to 40%. The majority of farms milk at intervals of 10 hours and 14 hours. Trials have suggested that this does not produce significantly lower production than precise 12-hourly milking in anything but the highest-yielding cows. The influence of milking frequency on milk yield appears to be controlled by local mechanisms acting within the udder. This is thought to be true because if two quarters are milked twice daily and the other two are milked four times daily, only the four times daily quarters show an increase in yield. Initially it was thought that back-pressure of milk within the alveoli was responsible. However, if the milk withdrawn from the four times daily quarters is replaced by an equal volume of saline (i.e. to restore the pressure within the alveoli), yields still increase. It has now been shown that milk naturally contains an inhibitor protein and it is the presence of this inhibitor, acting directly on the secretory cells within the alveoli, which influences yield. More frequent milking leads to more frequent removal of the inhibitor protein, and hence more milk is produced. Not only does frequent removal of the inhibitor protein stimulate increased activity of secretory tissue (and hence increased yields), it also slowly increases the amount of secretory tissue present, producing a longer-term effect. Finally, and after 2–3 months of three times daily milking, the number of secretory cells increases. This gives a longer-term response, which will persist when milking returns to twice daily. The extent of these effects depends partly on the internal anatomy of the udder. An udder with large teat and gland

cisterns and large ducts will store less milk in the alveoli between milkings. There is then less contact between milk inhibitor protein and the secretory tissue, and hence the cow, will be a higher-yielding animal. In the average cow, approximately 60% of the total milk is stored in the alveoli and small ducts, and 40% in the cisterns and large ducts.

Although not yet feasible, vaccination of cows against their own inhibitor protein raises interesting possibilities, as this could be a further way of increasing yields.

Environmental temperature

Under very cold conditions, water consumption and therefore milk yield fall. When the weather is very hot, food, and especially forage, intakes fall and this can depress both milk yield and milk fat levels. High environmental humidity exacerbates the effects of both hot and cold weather.

Length of dry period

Towards the end of lactation, the number of active alveolar secretory cells slowly declines, reaching a minimum during the early dry period. The alveolar cells do not die, but simply collapse, so that the space within the alveolus disappears and the udder consists of a greater proportion of connective tissue. New secretory tissue is laid down when the cow starts to 'freshen' ready for the next calving, and hence the total amount of secretory tissue (and therefore yield) increases from one lactation to the next. A dry period of between 4 and 8 weeks is ideal. If the cow is not dried off at all, the next lactation yield may be as much as 25–30% lower. This may occur, for example, following an abortion, or if a bull is running with the herd and no pregnancy diagnosis (PD) is carried out. Cows with excessively long dry periods often get overfat and metabolically inactive. This produces metabolic disorders around calving, and increases the

risk of mastitis. Conversely, very short dry periods have, under some situations, been associated with increased cell counts, but this effect is not large, and generally cows are more affected by prolonged than by excessively short dry periods.

3 Teat and Udder Defences Against Mastitis

Teat Defences	21
The teat skin	21
The teat canal	22
The keratin flush	22
The keratin plug	22
Teat closure	22
Teat canal dimensions and speed of milking	23
Mastitis and milking frequency	24
Teat-end damage and mastitis	25
Defences within the Udder	25
Intrinsic defence mechanisms	25
Inducible defence mechanisms	27
Poor response in the freshly calved cow	30
Individual cow variation	31
Can cell counts get too low?	31
Effect of low selenium and/or vitamin E	31
Reduced PMN activity in milk	32

Apart from very few exceptions, for example tuberculosis and leptospirosis, infections causing mastitis enter through the teat canal. The cow has very effective ways of reducing the risk of entry of infection through the teat, and even those infections that do succeed in penetrating the teat canal defences are commonly overcome by defences within the udder. Considering how often the teats, and especially the teat ends, become contaminated with bacteria, the overall incidence of mastitis in most herds remains relatively low, although no doubt most farms would prefer it to be even lower. This chapter studies the many ways in which the cow repels

infection. It will then be easier to understand the reason for carrying out some of the in-parlour control measures discussed in later chapters.

Defence mechanisms involve both the teat and udder and can be summarized as follows:

Teat defences act by preventing entry of infection into the udder.

- Intact skin provides a hostile environment for bacterial multiplication.
- Teat canal closure mechanisms reduce the risk of entry between milkings.

- Bacteria adherent to keratin in the teat canal are flushed out at the next milking.

Udder defences act by removing infections that have been able to pass through the teat canal. They are:

- Intrinsic, i.e. mechanisms that are always present.
- Induced, i.e. those mechanisms that come into operation in response to bacterial invasion.



Plate 3.1. The very dry skin on this teat would not only act as a potential reservoir for staphylococci and other mastitis organisms, but also reduce milk flow rates and hence speed of milking.

Teat Defences

The teat skin

Teat skin has a thick covering of stratified squamous epithelium (Fig. 2.7), the surface of which consists of dead cells filled with keratin. When intact, this provides a hostile environment for bacteria, thus preventing their growth. In addition, there are fatty acids present on skin that are bacteriostatic, that is, they prevent bacterial growth. However, these bacteriostatic properties can be removed by continual washing, especially using detergents, and this is why the premilking teat sanitizer should be chosen carefully.

The normally intact surface of the skin may also become compromised by cuts, cracks, chaps, bruising, warts, pox lesions, etc. Bacteria can then multiply on the sur-

face of the skin and become a reservoir for mastitis infections. This is particularly the case for organisms such as *Streptococcus dysgalactiae* and *Staphylococcus aureus*. An example is shown in Plate 3.1. Not only would this teat act as a reservoir of mastitis organisms, but it would also reduce milking speed. Trials have shown that cows with badly dry and cracked teat skin are much slower milkers (Fig. 3.1). They may have double the 'unit on' time to achieve the same level of yield, and, of course, this increased time can lead to teat-end damage.

Maintaining an intact and healthy teat skin is one of the important functions of the emollient present in postmilking teat dips.

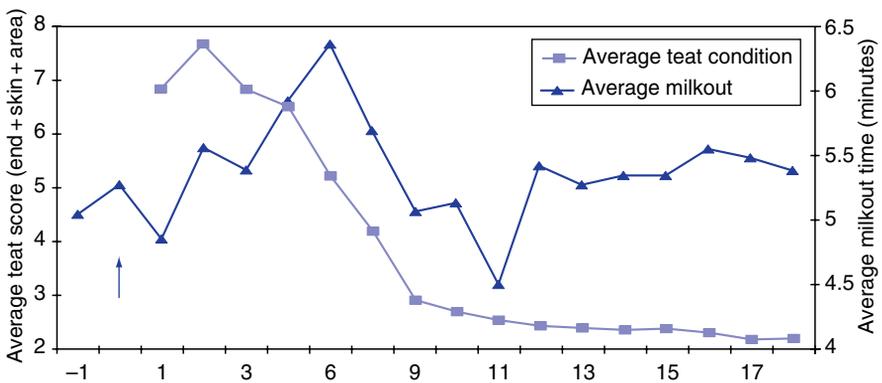


Fig. 3.1. Teat condition and milkout time. In this experiment, cows' teats were artificially damaged at time zero, leading to an increase in teat score. Note how this is followed by milkout time increasing from 4 minutes to 6.5 minutes by day 6.

The teat canal

The teat canal is 9 mm long (range 5–13 mm) and is lined with folds of keratinized skin epidermis, covered by a thin film of lipid. This has similar antibacterial properties to teat skin (Figs 2.1 and 2.7). These properties are most effective when contraction of the sphincter muscle leads to canal closure. At teat closure, the sphincter muscle contracts, the folds interdigitate to form a tight seal and the hydrophobic lipid lining ensures that no residual continuous column of milk persists, which could otherwise act as a ‘wick’ for bacterial entry. A few droplets remain, sometimes referred to as ‘milk lakes’. These often contain bacteria, which must be flushed out at the next milking.

Damage to the canal lining and lipid seal could result in a persistent residual column of milk, while fissures and serum ooze from a cracked epidermis would predispose to bacterial proliferation.

The rosette of Furstenberg (Fig. 2.1), on the inner side of the teat canal, is a ring of lymphocyte cells that detect invading bacteria and stimulate an immune response.

It takes at least 20–30 minutes for the teat end to become fully closed and hence, in order to protect teats from bacterial contamination, advice is often given that animals should not be allowed to lie down until at least 30 minutes after milking. Cows should not be left just standing doing nothing, however, as the extra standing times might increase the incidence of lameness. In addition, if they are left standing in an overcrowded, dirty or draughty passageway, the resulting increased teat skin damage and/or teat contamination might actually increase the risk of mastitis. The majority of farms would now simply encourage cows to walk back along clean passageways, past fresh food and into clean cubicles, and those cows that fail to stop to eat are probably so bad on their feet that they are best allowed to lie down to rest. Foot baths are commonly situated a short distance from the parlour exit. These should not be too deep, i.e. 70 mm maximum, to avoid splashing of the open teat ends, and the bath solution should be changed on a regular basis.

The keratin flush

Many bacteria entering the teat between milkings become trapped by the layer of keratin and lipid lining the teat canal. They are then flushed out at the start of the next milking by the first flow of milk, as this removes the superficial layers of keratin lining the teat canal. This is known as ‘the keratin flush’. It is very important to ensure that udder preparation and unit attachment are such that milk flows out of the teat when the cluster is applied, and that there are no reverse flow mechanisms that might lead to milk and infection being propelled back up into the udder. Foremilking will help in the removal of these trapped organisms.

The keratin plug

During the dry period a mixture of wax and keratin accumulates in the teat canal to form a physical plug. This mechanism is extremely important in preventing new infections, although as discussed in the section on dry period infections in Chapter 4, it is by no means always effective. This is especially the case for cows with ‘open’ teat ends that are fast milkers.

Teat closure

Figures 3.2a and b show the importance of teat sphincter closure in relation to *E. coli* mastitis. Teats were dipped in a broth culture of *E. coli* at varying times after milking. Of the teats dipped and exposed to *E. coli* in the first 10 min after milking, 35% developed mastitis. However, if the teats were not dipped into the *E. coli* broth until a few hours before the next milking, then only 5% developed mastitis.

It is particularly important to prevent liner slippage and resultant teat-end impacts at the end of milking (see Chapter 5). This is because: (i) the canal is more ‘open’ at the end of milking; and (ii) there may be no milk remaining in the quarter to flush out the organisms that have penetrated the teat canal by reverse flow.

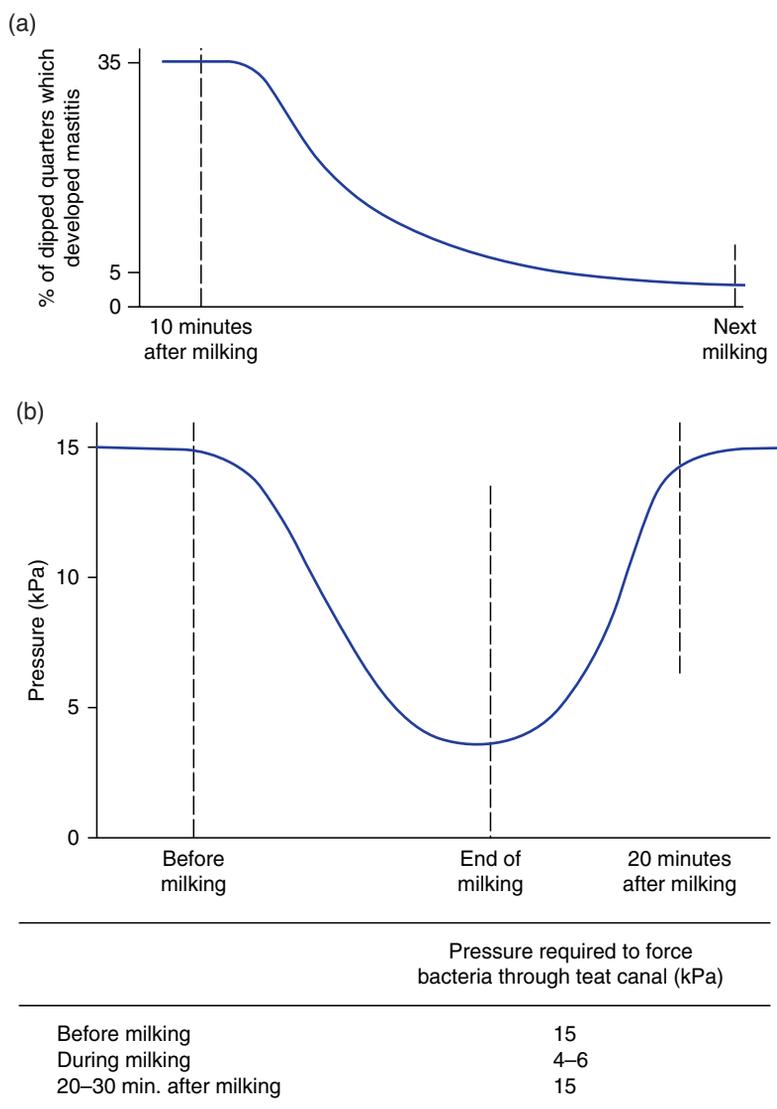


Fig.3.2. (a) The importance of teat sphincter closure in relation to *E. coli* mastitis: if teats were dipped in a broth culture of *E. coli* 0–10 min after milking, 35% of quarters developed mastitis. This reduced to 5% if teats were dipped in *E. coli* broth immediately prior to the next milking. (From Bramley *et al.*, 1981.)

(b) Pressure required to force fluid through the teat canal before, during and after milking. (From Bramley *et al.*, 1981.)

The degree of closure of the teat canal can be quantified in terms of the pressure required to force fluid back up through the teat canal, and is shown graphically in Fig. 3.2b.

Teat canal dimensions and speed of milking

Cows with short teat canals (i.e. short vertical length) and those with a wide cross-section diameter are more susceptible to mastitis. Cows with 'open' teat canals also milk faster. As this is likely to be an

inherited feature, there will be a genetic susceptibility to mastitis. Conversely, provided that teat-end lesions do not develop, 'hard' milkers, with slow milk flow rates, will have a lower infection rate than fast milkers.

However, speed of milking is correlated with yield (the greater the yield the greater the milk flow rate) and hence increasing selection for yield has led to an overall increase in milk flow rates. Table 3.1 shows that the average milk flow rate for a fast milker doubled between 1950 and 1990.

Table 3.1. Average milk flow rates of fast milkers (kg/min). (From Grindal *et al.*, 1991.)

	Per quarter	Per cow
1950	0.8	3.2
1990	1.6	6.4

This has led to a 12-fold increase in susceptibility to mastitis over the same period. Yields have undoubtedly increased further since 1990, and hence we can expect to see a corresponding increase in mastitis susceptibility. It will be a challenge to us all to provide optimum conditions of housing, machine function and management to control these infections.

Table 3.2 shows the numerical relationship between flow rate and mastitis incidence when teats were experimentally exposed to a high bacterial challenge. Three different machine conditions and varying flow rates were used. Initial results were obtained with a well-functioning machine where the liners were fitted with teat shields

(see Fig. 5.7). If there was no pulsation or, even worse, if teat-end impacts were a problem, then the mastitis risk (expressed as the percentage of quarters becoming infected) became greater, reaching 100% in cows with very high milk flow rates. (Milking machine function is discussed in Chapter 5.)

Cows with high flow rates are also much more susceptible to contracting new infections during the dry period.

Mastitis and milking frequency

The flushing action of milking removes the superficial layers of keratin lining the teat canal and in so doing removes bacteria that are adherent to the keratin. This is sometimes referred to as 'the keratin flush' (see page 22), and it is particularly important for the removal of *Streptococcus agalactiae* and *Staphylococcus aureus*, which invade by slow growth through the teat canal. Hence, cows milked three times daily are generally less susceptible to mastitis than cows milked twice daily, and, provided there is no adverse effect of machine milking, they tend to have lower cell counts.

Increased frequency of milking also decreases the volume and pressure of milk within the udder, and hence reduces the risk of milk leakage on to cubicle beds, which further decreases mastitis risk. Both factors further decrease the susceptibility to mastitis organisms invading the udder.

This all assumes optimum functioning of the milking equipment. If machine function is poor, with defective pulsation and/or teat-end impacts, then increased frequency

Table 3.2. The influence of milk flow rate from the teat end on the percentage of quarters becoming infected following experimental challenge. A poorly functioning machine dramatically increases the infection rate. (From Grindal *et al.*, 1991.)

Milking conditions	Quarter flow rate (kg/min)			
	<0.8	0.8–1.2	1.2–1.6	>1.6
	Percentage infection			
Good pulsation + shields	3	4	7	36
No pulsation	15	20	38	92
Pulsation + impacts	36	37	55	100

of milking would lead to an increased risk of mastitis.

Teat-end damage and mastitis

The teat canal is obviously of vital importance in the prevention of new cases of mastitis and clearly it follows that any damage to the teat end will compromise the defence mechanisms. Examples of teat-end damage are described in detail in Chapter 14 and include:

- Hyperkeratosis (a protrusion of keratinized skin at the teat sphincter) and sphincter eversion, both of which are caused primarily by adverse effects of machine milking.
- Physical trauma: cuts, crushing or bruising.
- 'Black spot': a lesion, probably traumatic in origin, with secondary infection caused by the bacterium *Fusobacterium necrophorum*.
- Milking machine damage: teat-end oedema, haemorrhage, sphincter eversion, etc.
- Excessive dilatation of the canal, for example, when administering intramammary antibiotics or when inserting a teat cannula. This can produce cracks in the keratin and lipid lining, thereby providing an opportunity for bacterial multiplication.

The way that teat cannulae are used is particularly critical, since it is often 1–2 days after withdrawal of the cannula (especially after it has been *in situ* for several days) that mastitis occurs. This is presumably because the tightly fitting cannula prevents bacterial entry while it is in position, but after removal the stretched canal has lost both its ability to close and its bacterial defences, allowing easy entry of infection. For this reason many recommend infusing a small quantity of antibiotic after each milking for the first 3–4 days following removal of the cannula.

Defences within the Udder

Even when bacteria have managed to overcome the defence mechanisms of the teat canal and have either grown through it or been forced through by the milking machine, clinical or subclinical udder infections are by no means a certainty. There are several highly efficient systems within the udder that assist in the removal of bacteria and often prevent infections becoming established. These can be categorized as intrinsic defence mechanisms, which are systems continually present in the udder, and inducible systems, which come into operation in response to bacterial invasion.

Intrinsic defence mechanisms

Lactoferrin

Iron is required for bacterial growth, and especially for the growth of *E. coli*. In the dry, non-lactating udder, lactoferrin removes the iron from udder secretions and in so doing minimizes bacterial multiplication. Although the risk of new *E. coli* infections during the dry period is four times greater than in lactation, the presence of lactoferrin ensures that clinical disease (i.e. clinical *E. coli* mastitis from these infections) is rare until the next lactation (see Table 3.3).

Table 3.3. Experimental *E. coli* infection in lactating and dry cows. (From Hill, 1981.)

	No. of quarters challenged	No. of quarters developing clinical mastitis
Lactating cows	16	12 ¹
Dry cows	12	2 ²

¹ Of the four quarters that did not show clinical mastitis, two had a high cell count and two had subclinical mastitis.

² Both cases were in cows challenged only a few days prior to calving, when the lactoferrin in milk had already fallen to a low level.

The bacteriostatic effects of lactoferrin are lost during lactation because:

- Lactoferrin is present in only low concentrations.
- High citrate levels in milk compete with lactoferrin for iron, producing iron citrate. This can be utilized by the bacteria during their growth processes.

Lactoperoxidase

All milk contains the enzyme lactoperoxidase (LP). In the presence of thiocyanate (SCN) and hydrogen peroxide (H_2O_2), lactoperoxidase can inhibit the growth of some bacteria (Gram-positive organisms, see page 57) and kill others (Gram-negatives). The level of thiocyanate in milk varies with the diet, being particularly high when brassicas and legumes are fed. Hydrogen peroxide can be produced by bacteria themselves. Gram-negative bacteria produce very little H_2O_2 , and so the lactoperoxidase system is probably not important in their control. There is some evidence that Gram-positive bacteria such as *Streptococcus uberis* may produce sufficient H_2O_2 for the lactoperoxidase system to be partially effective in their control.

Complement

Complement is the general term for a series of proteins which, when acting together, produce a cascade effect that results in the killing of certain strains of Gram-negative bacteria, such as *E. coli*. *E. coli* is one of a number of coliforms that can be grouped into serum-sensitive strains (killed by complement) and serum-resistant strains (not

killed). It has been shown that only the latter are likely to produce mastitis. If a serum-sensitive strain of *E. coli* is isolated from a milk sample therefore, it is likely to be a contaminant only and not a cause of mastitis.

Immunoglobulins (antibodies)

Antibodies are unlikely to have a primary role in mastitis control since it is well known that colostrum contains very high levels of antibodies, and yet freshly calved cows can develop peracute mastitis and frequently do get severe mastitis several days after calving. The role of specific antibodies against mastitic bacteria is unclear. Probably their main function is in the opsonization of bacteria before they are engulfed by white blood cells and macrophages. Opsonization is a process whereby the bacteria become coated with antibody. A portion of an antibody molecule (the Fab arm) attaches to the bacteria, leaving a second arm (the Fc fragment) exposed. White blood cells (PMNs) are activated by the exposed Fc arm and attach to it. Phagocytosis (engulfing) of the bacteria can then proceed much more rapidly.

Cellular response

There is a variety of different types of cells in normal milk, but by no means all of them can kill bacteria. The total number of cells can be counted and is expressed as the somatic cell count (SCC). Approximate percentages are given in Table 3.4, although there is still some dispute concerning which cell types are present. The proportions will vary with factors such as level of yield, stage of lactation and, of course, the presence of infection.

Table 3.4. Percentage of cell types in milk and colostrum. (From Lee *et al.*, 1980.)

	Mid-lactation	Colostrum
PMNs ^a (neutrophils)	3	61
Vacuolated macrophages	65	8
Non-vacuolated macrophages	14	25
Lymphocytes	16	3
Duct cells	2	3

^a PMNs = polymorphonuclear leucocytes, bacteria-killing cells, mainly neutrophils.

The main function of macrophages and lymphocytes is to recognize bacteria and then trigger alarm systems that induce a more vigorous host response, eventually leading to huge numbers of PMNs (polymorphonuclear leucocytes, mainly neutrophils) entering the milk. These alarm systems are the inducible defence mechanisms described in the next section.

PMNs are important bacteria-killing cells that originate from blood. However, in normal milk they are present in such low numbers as to be ineffective against a heavy bacterial challenge.

Inducible defence mechanisms

When all else has failed and bacteria have penetrated the teat canal and overcome the intrinsic defence mechanisms, alarm signals are sent out to the body of the cow requesting 'help'. The response to the alarm is the induced system of mammary defences. It is both highly effective and fascinating in its mechanisms. The various stages will be described in some detail.

The chemotaxin alarm

The macrophages and PMNs (see Table 3.4) already present in the milk recognise and engulf fragments of dead bacteria and their toxins in a process known as phagocytosis (Fig. 3.3). Phagocytosis in turn leads to the

release of various chemical mediators, known collectively as chemotaxins. Specific chemotaxins include chemicals such as interleukin 8 and tumour necrosis factor (TNF). It is these chemicals, plus the toxins produced directly from bacteria multiplying within the udder, which act as the alarm system.

The inflammatory response

The principal response to chemotaxins is a massive inflow of PMNs from the capillaries in the teat wall and udder into the cisterns and ducts. This is achieved in a variety of stages (Fig. 3.4):

- Increased blood flow: blood vessels in the teat wall dilate, thus increasing the blood flow and the supply of PMNs to the affected quarter. Thus a quarter with an acute mastitis infection becomes palpably swollen, hot and painful.
- Margination: small carbohydrate projections (selectins) appear on the inner surface of the cells lining the capillary wall. These attract PMNs towards the sides of the capillaries and help to force them between the capillary cells and out through the wall.
- Loosening of endothelial cell junctions: under the influence of specific chemotaxins, the endothelial cells lining both the capillaries and the teat and udder cisterns literally move apart to facilitate a more

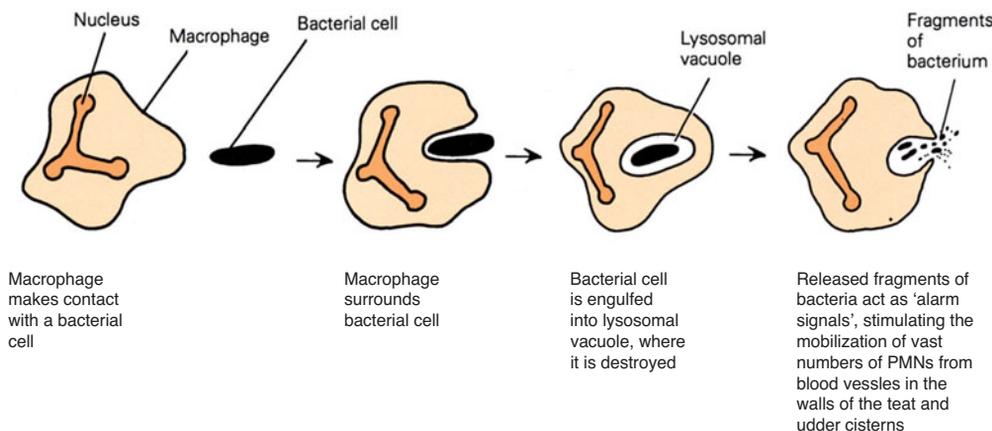
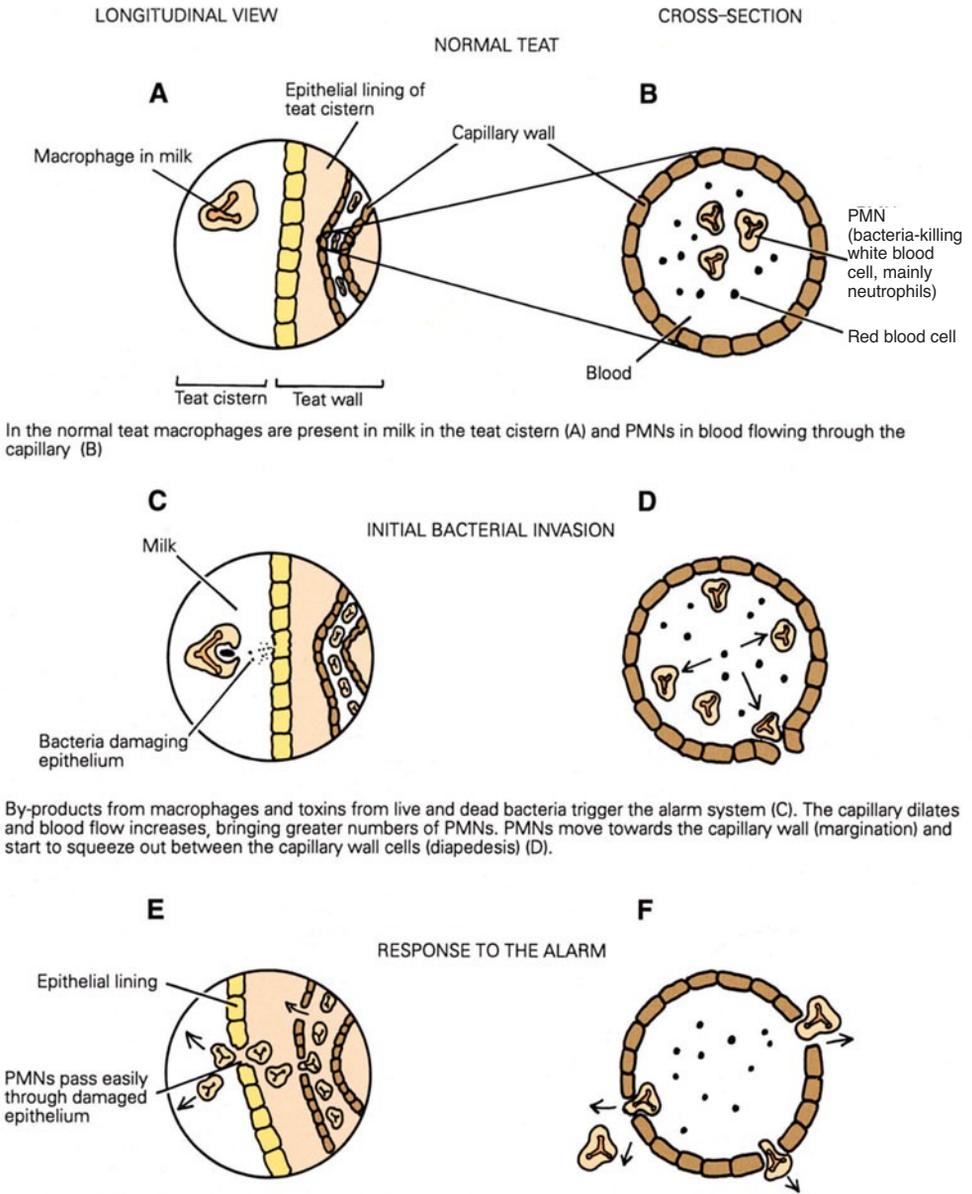


Fig. 3.3. The process of phagocytosis, in which a macrophage engulfs and destroys a bacterial cell.



In the normal teat macrophages are present in milk in the teat cistern (A) and PMNs in blood flowing through the capillary (B)

By-products from macrophages and toxins from live and dead bacteria trigger the alarm system (C). The capillary dilates and blood flow increases, bringing greater numbers of PMNs. PMNs move towards the capillary wall (margination) and start to squeeze out between the capillary wall cells (diapedesis) (D).

(E) and (F) Huge numbers of PMNs pass into the milk in the teat and udder cisterns, to produce a massive increase in cell count. They start engulfing and killing bacteria, releasing more by-products, which further activates the alarm system.

Fig. 3.4. The response to the alarm signals of bacterial invasion.

rapid passage of PMNs into the infected milk. They close again when the PMNs have passed through.

- **Diapedesis:** PMNs squeeze through the walls of the capillaries, across the tissue of the teat wall and udder, through the endothelial lining and into the milk, where they are able to engulf the bacteria.
- **Damage to epithelial cells:** some of the cells lining the teat duct and lactiferous sinuses can be totally destroyed by the toxins produced by *E. coli* infections, and this allows further access of PMNs (and serum) into the area of multiplying bacteria. Plate 4.8 shows the inside of a normal teat, which can be compared with the severely inflamed mastitic teat in Plate 4.9. (See Chapter 4.)
- **Serum ooze from blood vessels:** because the junctions between endothelial cells in the capillary walls have opened to allow the passage of PMNs, serum can also flow into the tissues. This produces an uncomfortable swelling of the affected quarter, as tissues stretched and dilated by fluid are painful. In acute *E. coli* infections particularly, the leakage of serum is so pronounced that it flows directly into the milk and produces the yellow, watery secretion that is so typical of an acute coliform mastitis. Occasionally serum ooze may even be seen on the skin surface, as in Plate 4.10.

- **Phagocytosis.** Once they have passed into the milk, the PMNs released in response to the chemotaxin alarm start to engulf whole bacteria (Fig. 3.3) and the major part of the bacteria-killing process, known as phagocytosis, begins. Inside the PMN the bacteria are destroyed by a system involving hydrogen peroxide. The first PMNs to arrive are highly active. They release lysosomal granules from their cytoplasm, and this further amplifies the inflammatory response.

The severity of the inflammation is often such that it persists well after the bacteria have been destroyed. This explains the common finding of a hard, hot and painful quarter with a watery secretion, from which bacteria cannot be cultured. This is almost certainly caused by an acute *E. coli* infection that has been rapidly counteracted by the cow's defence mechanisms.

The increase in the number of cells in milk due to the inflammatory response can be enormous. From a base level of only 100,000 (10^5) per ml, i.e. a cell count of 100, it may increase to as many as 100,000,000 (10^8) per ml (a cell count of 100,000) in just a few hours, and many quarters rapidly reach a cell count of 10 billion (10^9). Bacteria are then rapidly eliminated, as shown in Fig. 3.5a, and so many PMNs may have entered the udder that the white cell count of the blood falls to almost zero.

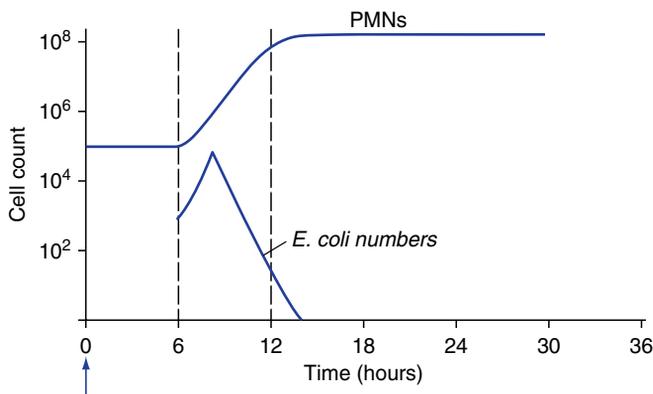


Fig. 3.5a. Good PMN (white cell) response in a mid-lactation cow can lead to rapid elimination of *E. coli*. Infection at time zero. (From Hill, 1981.)

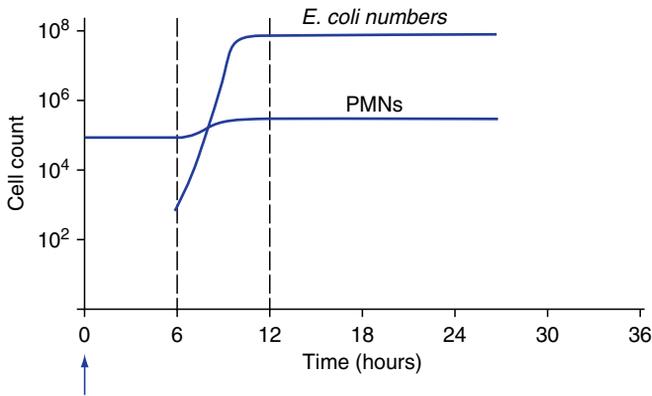


Fig. 3.5b. A poor cellular response seen especially in some freshly calved cows allows *E. coli* to multiply to very high numbers in the udder (compare this with the good response shown in Fig. 3.5a). Provided the cow survives, bacterial numbers may remain high for several days. Infection at time zero. (From Hill, 1981.)

Poor response in the freshly calved cow

The description given above applies to healthy cows which are able to mount a dramatic inflammatory response, producing a hard, hot, swollen quarter. Some of these cows may be sick, others less so. There is, of course, an alternative reaction. For a variety of reasons, freshly calved cows seem unable to mount an effective PMN response, and when *E. coli* invades the udder it can continue to multiply almost unchecked. In the instance shown in Fig. 3.5b, as few as ten organisms (a minute number) may have infected the quarter 12–18 hours earlier, but because the cow was less able to mount an immune response, the bacteria continued to multiply, with bacterial levels reaching 10^8 (100,000,000) per ml.

Because there is a very limited inflammatory response in these cases, the mastitis may be difficult to detect. The udder may well remain soft and the changes in the milk could be minimal, making it almost indistinguishable from colostrum. However, the cow herself will be very ill, due to the systemic effects of large quantities of endotoxin, which have been produced by the multiplying *E. coli* bacteria. (Not all bacteria produce endotoxins.) Severely affected cows may be recumbent, scouring, dull and not eating. They may or may not have a temperature

(cows with a good inflammatory response invariably have an elevated temperature) but will probably be shivering with a foul-smelling greenish, mucoid diarrhoea.

Cows that do not die may remain seriously ill for some considerable time. The lipopolysaccharide endotoxin produced by *E. coli* has a generalized effect on all body organs, which may leave the cow in poor condition, dull and with a poor appetite, for several weeks. There is little that can be done for such cows, since the damage to the udder tissue has already occurred, and it is simply time, nursing and tissue regeneration that will effect a recovery. Associated damage to the teat lining is shown in Fig. 3.6.

When phagocytic cells eventually appear, they are often monocytes, cells that are much less effective than PMNs, and therefore coliforms may continue to be excreted in the milk for 1–2 weeks post infection. This strongly justifies the use of antibiotic for the treatment of early lactation coliform mastitis cases. When healing eventually occurs, it is often with alveolar keratinization and milk production in that quarter is then lost, although most recover in the next lactation.

The pronounced immunosuppression in the periparturient cow (which leads to an increase in many diseases around calving) is probably an innate mechanism protecting

Fig. 3.6. Damage to teat canal lining following an acute *E. coli* infection.

the dam against an overreaction to potential release of fetal (and therefore paternal) antigen into the maternal circulation during parturition and against antigens released from uterine trauma. Feeding and management also play a part. This is discussed in more detail in Chapter 4.

Individual cow variation

There is a considerable variation between individual cows in their response to an *E. coli* challenge, even in cows at the same stage of lactation. For example, when *E. coli* bacteria were experimentally infused into two different cows:

- 98% were killed within 6 hours in one cow compared with
- only 80% killed within 6 hours in the second cow.

Part of this variation is undoubtedly due to an inherent difference in the rate at which PMNs can kill bacteria. Using test-tube experiments, it can be shown that PMNs taken from the blood of different cows will kill (or eliminate) *E. coli* at different rates. However, the main difference between cows is the rate at which cells can be mobi-

lized from blood into the teat and udder sinuses.

Can cell counts get too low?

There is a body of opinion which suggests that if somatic cell counts are too low, then cows are more prone to developing the peracute and fatal form of *E. coli* and other types of mastitis. Initial survey work (Green *et al.*, 1996) showed that herds with lower cell counts had a higher incidence of toxic mastitis than herds with higher cell counts. This was then followed by more detailed work (Peeler *et al.*, 2002) on individual quarters, showing that quarters with a cell count of less than 20,000 had an increased risk of developing clinical mastitis. However, the same study showed that quarters with a cell count of above 100,000 had an increased risk of clinical disease. There are many other studies that have shown that herds with raised cell counts have an increased risk of clinical mastitis, and that bulls producing daughters with raised cell counts also have an increased risk of mastitis.

The difference between an initial cell count of 50,000 or 150,000 cells per ml is almost insignificant when, with clinical mastitis, cell counts could rise to 100,000,000 per ml within a few hours. It appears to be the speed at which cells can be mobilized into the udder, rather than the number present initially, which is the critical factor.

Effect of low selenium and/or vitamin E

Macrophages and PMNs engulf bacteria and destroy them. One of the methods of destruction is the release of lysozymes (destructive enzymes) within the PMN vacuole, with the resultant production of hydrogen peroxide. A vacuole is simply a compartment within a cell. The hydrogen peroxide thus produced needs to be destroyed immediately, and this is done by the action of glutathione peroxidase, (GSH-PX), a selenium-dependent enzyme. Failure to destroy the hydrogen peroxide can quite

rapidly result in the death of the phagocytosing cell itself.

Vitamin E reduces the rate of hydrogen peroxide formation within the PMN and stabilizes its cell membranes against its attack, while selenium increases the activity of GSH-PX. Workers in North America have demonstrated a correlation between dietary levels of selenium and vitamin E and mastitis, and recommend supplementation of 1000 IU vitamin E per cow per day during the dry period and 400–600 IU per cow per day during lactation. British diets containing a higher proportion of grass silage are less likely to be vitamin E deficient, but all-maize diets and diets containing more gluten or high fat, especially polyunsaturated fatty acids (PUFAs), require supplementation. One British survey showed that in low mastitis incidence herds there was a correlation between increased cell count and low GSH-PX levels: those herds low in vitamin E/selenium had higher counts.

Reduced PMN activity in milk

Unfortunately PMNs are less active in milk than in blood and this is a further reason why peracute mastitis and endotoxic shock

may occur. The reduced PMN activity is thought to be associated with a variety of factors, including the following:

- They may become coated with casein, which reduces their activity.
- PMNs are unable to distinguish fat and casein globules from bacteria. The globules may be continually engulfed, thereby exhausting PMNs.
- Oxygen levels are naturally lower in milk than in blood, and are reduced even further by bacterial multiplication in mastitic secretions. This limits the ability of the PMN to destroy the phagocytosed bacteria.

When PMNs leave the capillaries, they effectively need to take their food stores (glycogen) with them. This is sometimes referred to as 'taking their packed lunch'. Once the food has been exhausted, the PMNs become relatively inactive.

Although the above factors limit the activity of PMNs, the system is still highly effective, probably because of the very large numbers of PMNs present. In fact, a cow with acute mastitis may pour so many white cells into the mammary gland that blood levels may fall almost to zero.

4 The Mastitis Organisms

Mastitis Definitions	34
Development of a New Infection	34
Arrival of a reservoir of infection	34
Transfer of the infection from the reservoir to the teat end	34
Penetration of the teat canal	34
Host response	35
Dry period versus lactation infections	36
Strategy for Mastitis Control	36
Contagious and Environmental Organisms	36
Epidemiology of contagious organisms	37
Epidemiology of environmental organisms	37
Specific Organisms Causing Mastitis	37
<i>Staphylococcus aureus</i>	38
Coagulase-negative staphylococci (CNS)	42
<i>Streptococcus agalactiae</i>	42
<i>Streptococcus dysgalactiae</i>	43
<i>Mycoplasma</i> species	44
Environmental Organisms	44
Coliforms, including <i>Escherichia coli</i>	44
Other coliforms	46
<i>Streptococcus uberis</i>	47
Sources of infection	47
Spread within the herd	49
Dry Period Infections	50
Phases of the dry period	50
The host immune response	53
The influence of dry matter intake	53
Short dry periods	54
Less Common Causes of Mastitis	54
<i>Bacillus</i> species	54
Yeasts, fungi and moulds	55
Relatively rare causes of mastitis	55
Culturing Milk for Bacteria	56
Taking a milk sample	56
Laboratory plating and incubation	56
Antibiotic sensitivity testing	57
Interpretation of results	58
Total Bacterial Count, Laboratory Pasteurized Count and Coliforms	58
Methodology	59

This chapter examines mastitis in general terms, discusses the organisms involved and provides an overview of control measures.

Some diseases, for example, foot-and-mouth, can be totally eliminated by a test and cull policy and strict biosecurity. Other diseases, for example, the bacterial infection blackleg, can be totally controlled by vaccination. Mastitis is different. It will never be eradicated, because there are too many different bacteria involved, and many of these are always present in the environment. Antibiotic treatment has varying degrees of effectiveness and, for a variety of reasons, vaccination can only ever produce a partial reduction in incidence. The approach to mastitis must therefore be one of control and, with increased milk flow rates producing ever higher mastitis susceptibility (see page 24), control will become increasingly important in the future.

Mastitis Definitions

Mastitis is commonly referred to under the following categories:

- Clinical mastitis: an udder infection that can be seen, e.g. by clots in the milk, hardness, swelling, etc.
- Subclinical mastitis: an udder infection that shows no external changes.

Clinical mastitis can be:

- Acute mastitis: sudden in onset and shows severe signs.
- Chronic mastitis: persists for a long time, but is not severe.

Development of a New Infection

To be able to appreciate the importance of the various control measures discussed later, it is first necessary to understand how and when a new case of mastitis occurs. This will be dealt with under the following headings: (i) arrival of a reservoir of infection; (ii) transfer of infection from the reservoir to the

teat end; (iii) penetration of the teat canal; (iv) host response; and (v) dry period versus lactation infections. The ‘cow factors’, i.e. the mechanics of teat and udder defence mechanisms, were described in the previous chapter.

Arrival of a reservoir of infection

Some of the bacteria that cause mastitis are always present in the environment and are therefore called ‘environmental organisms’. For these, ‘arrival of a reservoir’ simply means a change in environmental conditions, leading to an increased challenge of infection at the teat end. Many studies have shown that teats that are soiled with mastitic bacteria are more liable to develop environmental mastitis.

Other infections (e.g. *Streptococcus agalactiae*) are normally only present in the udder of infected cows and ‘arrival of a reservoir’ indicates either the purchase of an infected cow or perhaps an infected cow calving down into a herd. In this instance, the infection is ‘contagious’ because it passes from cow to cow.

Transfer of infection from the reservoir to the teat end

This will generally occur *between* milkings for environmental organisms, since the first stage in the establishment of a new infection is the transfer of bacteria from the environment to the teat end. However, for contagious organisms, transfer occurs *during* the milking process and a vector is needed to carry the bacteria from the infected to the non-infected cow (or infected to non-infected quarter). Examples of vectors include the milker’s hands, udder cloths (if the same cloth is used on more than one cow) and the milking machine liner.

Penetration of the teat canal

There appear to be two ways in which bacteria commonly penetrate the teat canal:

- First, growth through the canal. After transfer to the teat end, contagious organisms, especially *Staphylococcus aureus* and *Streptococcus agalactiae*, have strong adhesive factors and begin their 'attack' on the udder by first establishing a colony at the teat end, i.e. they multiply. After colonising the teat end, bacteria literally grow up through the teat canal and move into the teat sinus.
- Second, propulsion through the canal. Pathogens, particularly environmental bacteria such as *E. coli*, do not have adhesive properties and hence are often forced through the canal, usually with a reverse flow of milk, such as occurs with teat-end impacts (see pages 79–80). The exception to this is infections that develop during the dry period.

This difference between the two groups of organisms is shown in Table 4.1. A culture of either *Staphylococcus aureus* or of *E. coli* was applied to the teat ends. Swabs were then taken on a daily basis and the percentage of swabs positive for the organism each day was monitored.

Although there was a high recovery rate for *E. coli* on days 1 and 2, the organism had been eliminated by day 4. Because the staphylococci form a colony, the number of organisms multiplies and gradually increases with time. In this experiment, teats were not disinfected after milking. If postdip had been applied, then the infection rate with staphylococci would have been much lower.

One of the other reasons for differences in the way organisms penetrate the teat canal is the variation in their inherent ability to adhere to epithelial surfaces. Contagious organisms such as *Staphylococcus aureus*

and *Streptococcus agalactiae* have strong adhesive properties. They therefore stick to surfaces and become established as chronic conditions. The environmental organism *E. coli* has virtually no adhesive properties. Hence, during lactation, transfer into the udder is most commonly associated with reverse flow of milk through the teat canal, often by teat-end impacts (pages 79–80). However, new infections during the dry period are an extremely important part of the pathogenesis for both *E. coli* and *Streptococcus uberis*, and these new infections are clearly not propelled through the teat canal via a reverse flow of milk. Dry period infections are dealt with in detail later in this chapter.

Although it has been stated that contagious organisms grow up through the teat canal and environmental organisms are propelled, it should be appreciated that the distinction is by no means as precise as this. Clearly a reverse flow of milk will assist movement of contagious pathogens through the canal, while there are occasions (e.g. exposure to high teat-end challenge immediately after milking) when *E. coli* seems to penetrate without reverse flow of milk. *Streptococcus uberis*, which has both environmental and contagious properties, can enter by both routes.

Host response

Even when bacteria have penetrated the teat canal and entered the udder, establishment of infection is by no means a certainty. There are a variety of ways in which the udder can overcome infection, and the effectiveness of these mechanisms can vary enormously between cows. This was discussed in

Table 4.1. A comparison of the pathogenesis of contagious and environmental organisms following experimental infection. *Staphylococcus aureus* (contagious) establishes a colony at the teat end and hence represents a continuous risk. *E. coli* is present for a much shorter period of time.

% teat swabs positive each day	Day 1	Day 2	Day 5	Day 10	% infected quarters
<i>S. aureus</i>	50	68	75	73	4
<i>E. coli</i>	93	34	10	0	0

Chapter 3. There is also variation in host response to the different organisms, especially between the dry period and lactation.

Dry period versus lactation infections

The previous paragraphs refer primarily to those new infections that occur during lactation. It is now known that many new infections occur during the dry period. These infections often remain dormant in the udder, and do not appear as clinical mastitis until the first 3 to 4 months of lactation. Their precise mechanism of entry through the teat canal is unknown, but it must be by slow growth. This is discussed later in this chapter.

Strategy for Mastitis Control

Having seen how a new case of mastitis is established, it is now possible to define a strategy for control. This can be subdivided into three parts:

1. Reduce the reservoirs of infection. This means keeping the environment as clean as possible and reducing the number of cows carrying contagious organisms, e.g. by dry cow therapy, postmilking teat disinfection and culling.
2. Control spread by vectors. This is particularly important for contagious organisms and is discussed in detail in Chapter 6.
3. Optimize host defences. The host defence mechanisms were described in Chapter 3. Keeping teats and teat ends in good condition is obviously a vital component of mastitis control and is, once again, influenced by milking machine function, which is described in detail in Chapter 5.

Contagious and Environmental Organisms

It is not the purpose of this book to go into precise details of every organism that could cause mastitis: over 200 different organisms

have been recorded in scientific literature as being causes of bovine mastitis. They can be grouped as follows – organisms in bold type cause the majority of mastitis cases.

- **Contagious**

Staphylococcus aureus

Streptococcus agalactiae

Coagulase-negative staphylococci

Streptococcus dysgalactiae

Corynebacterium bovis

Mycoplasma

- **Environmental**

Streptococcus uberis

Coliforms:

E. coli

Citrobacter

Enterobacter

Klebsiella

Pseudomonas aeruginosa

Bacillus cereus

Bacillus licheniformis

Pasteurella

Streptococcus faecalis

Fungi

Yeasts

There are a number of other, less common causes of mastitis that are more difficult to categorize into contagious or environmental. These are listed on page 55. Although it is possible for this wide number of organisms to be involved, the majority of mastitis cases are caused by a few common bacteria. Table 4.2 shows the incidence of mastitis infection by different types of organism in 1968 compared with that in 1995 and 2007. Note the enormous decrease in the percentage of *Staphylococcus aureus* cases, and the proportional rise in the percentage of environmental cases (*E. coli* and *Streptococcus uberis*). The overall incidence of clinical mastitis has in fact declined dramatically, from 121 cases per 100 cows a year in 1968 to only 50 cases per 100 cows a year in 1995 and 47 in 2007. This decrease was largely due to the dramatic effects of control measures, such as postmilking teat disinfection, dry cow therapy and culling, on contagious mastitis.

Table 4.2. The results of one survey showing the decline in the incidence of contagious mastitis between 1968, 1995, and 2007 and the proportional rise in importance of the environmental infections *E. coli* and *Streptococcus uberis*. (Adapted from Hill, 1990; Booth, 1993; Bradley *et al.*, 2007.)

Type	Cases of clinical mastitis %		
	1968	1995	2007
Coliforms	5.4	26	19.8
<i>Streptococcus agalactiae</i>	3	0	0
<i>Staphylococcus aureus</i>	37.5	15.4	3.3
<i>Streptococcus dysgalactiae</i>	20.1	10.8	1.5
<i>Streptococcus uberis</i>	17.7	32	23.5
Others	16.3	15.8	0
No growth	0	0	26.5
No. of cases per cow per year	121	50	47

Although the percentage of environmental cases has increased therefore, from approximately 23% in 1968 to 43% in 1995, this is due to a decline in the contagious infections (*Staphylococcus aureus*, *Streptococcus agalactiae* and *Streptococcus dysgalactiae*), rather than a rise in the number of environmental cases.

The major change that has occurred over the past few years in the UK is the big increase in *Streptococcus uberis* infections. There are many species of *Streptococcus*, and combined, they show both environmental and contagious properties, in that the first case may arise from an environmental infection, especially during the dry period, but because of a poor response to treatment, further cases occur as a result of cow-to-cow spread during the milking routine.

The following section shows that contagious and environmental organisms have marked differences in their epidemiology.

Epidemiology of contagious organisms

- The mammary gland and/or teat skin are reservoirs of infection.
- Organisms are transmitted from the carrier cow or quarter to the teats of non-infected cows/quarters during the milking process.

- Colonies become established at the teat end and slowly grow through the canal over 1–3 days.
- Dry cow therapy (see Chapter 12) and postmilking teat disinfection (see Chapter 7) are important means of control.
- The dry period is not an important time for new infections.
- Herds with a high incidence of contagious infections often have high cell counts but a normal TBC/Bactoscan (see Chapter 10).
- Herds that only have a problem with contagious infections typically have a high cell count but often a low incidence of clinical mastitis.

Epidemiology of environmental organisms

- The environment is the reservoir of infection.
- Organisms are transferred from the reservoir to the teats between milkings.
- Penetration of the teat canal occurs by propulsion on a reverse flow of milk.
- Dry cow therapy, to eliminate existing coliform infections, is of limited value as environmental infections do not persist subclinically and are not carried from one lactation to the next.
- Many new infections occur during the dry period, and here dry cow therapy and internal teat seals are important preventive measures.
- Premilking teat disinfection is important in control, postmilking disinfection less so.
- Herds with a high incidence of environmental infections may have an acceptable cell count but a high level of clinical cases and a raised TBC/Bactoscan.

The differences in the epidemiology of contagious and environmental organisms are summarised in Table 4.3.

Specific Organisms Causing Mastitis

This section gives a short description of some of the major organisms causing

Table 4.3. A summary of the major differences between contagious and environmental organisms. The distinction between the two groups is not always precise.

	Contagious	Environmental
Source of infection	Teats and udder	Contaminated environment
Transfer of infection into the udder	During milking	Between milkings and during the dry period
Clinical mastitis	Most cases are subclinical	A higher proportion are clinical (<i>Streptococcus uberis</i> can be subclinical)
Control by	Postmilking teat dipping Dry cow therapy Milking hygiene Culling	Environmental hygiene Predipping Dry period teat sealants

mastitis, their appearance in culture and the type of mastitis they produce. It is certainly not in any way intended to be a comprehensive guide to the bacteriology of mastitis.

It should also be noted that it is not consistently possible to determine the organism producing mastitis from clinical signs alone. While there may be a few classic guidelines – for example, the serum-coloured watery secretion produced by an acute *E. coli* infection – these are by no means consistent. *E. coli* can also cause a very mild mastitis, with a few clots seen at one milking, which will have totally disappeared to give a normal udder at the next milking, or even occasionally a recurrent mastitis with a high cell count. The organisms described in some detail in the following sections are the contagious organisms *Staphylococcus aureus*, other staphylococci, *Streptococcus agalactiae*, *Streptococcus dysgalactiae* and *Mycoplasma*, and the environmental organisms *E. coli* and *Streptococcus uberis*.

Staphylococcus aureus

Staphylococcus aureus organisms are haemolytic gram-positive cocci, seen as creamy yellow/white colonies on blood agar (Plate 4.1).

They are coagulase-positive (i.e. they agglutinate with rabbit serum) and are sometimes referred to as coagulase-positive staphylococci, although not all *S. aureus* colonies are coagulase-positive. (More detail

of culture techniques is given later in this chapter.)

The primary reservoir for *S. aureus* is within the mammary gland. Staphylococci are notoriously difficult to treat and, once infection has become established, it is extremely hard to eliminate. Table 4.4 shows that treatment of clinical cases of staphylococcal mastitis with cloxacillin gives only a 25% cure rate and in subclinical cases only 40%. Treatment of primary infections in heifers should result in much better response rates, however, and conversely



Plate 4.1. Staphylococci growing on a blood agar plate. Each small creamy-white dot represents a colony of staphylococci containing literally millions of bacteria. The slightly lighter ring around the outside of the colony is the ring of haemolysis (broken-down blood).

Table 4.4. Bacteriological cure rates (as percentages) for Gram-positive intramammary infections using the antibiotic cloxacillin. (From Tyler and Baggot, 1992.)

Organism	Lactation		
	Clinical infection	Subclinical infection	At drying off
<i>Staphylococcus aureus</i>	25	40	65
<i>Streptococcus agalactiae</i>	85	>90	>95
<i>Streptococcus dysgalactiae</i>	90	>90	>95
<i>Streptococcus uberis</i>	70	85	85

chronically infected older cows may have bacteriological cure rates as low as 10%. This is discussed in more detail in the treatment section of Chapter 12.

The reasons for this poor response to treatment are as follows:

- Once established within the udder, staphylococci often become 'walled off' by fibrous tissue, allowing only poor penetration by antibiotics. The cow in Plate 4.2 is obviously affected, with large fibrous lumps protruding from the rear of her udder. She had a cell count of over 3 million cells/ml and suffered from recurrent bouts of mastitis.
- *S. aureus* is able to live within macrophages, PMNs (see page 26) and epithelial cells, out of the reach of antibiotics. Antibiotics can circulate within body fluids but are largely unable to penetrate cells. Other reasons for poor response are given in Chapter 12.

**Plate 4.2.** Chronic staphylococcal mastitis. Note the lumps in the udder.

These two factors also partly explain the very variable cell count and bacterial excretion rates of cows chronically infected with *S. aureus*, as shown in Table 4.5. These results show very clearly that it would be most unwise to take action (e.g. culling) against a cow on the basis of a single cell count or milk culture result. A negative culture result does not necessarily mean that the cow is free of *S. aureus*; it just means that on that day no organisms were isolated. Conversely, due to intermittent excretion and to excretion in low numbers, only around one-third of milk samples from infected cows are culture-positive. The poor response to treatment also emphasises the importance of ensuring that cows do not become infected with *S. aureus*, and hence the importance of strict hygiene in the milking parlour. Dry cow therapy is vital, and although response to treatment is disappointing (see Table 4.4), at least its use lowers the level of infection in a herd, and it is one further method of reducing the level of challenge to uninfected cows.

Table 4.5. Variation in cell count and bacterial excretion rate of a mammary gland infected with *Staphylococcus aureus*. (From Bramley, 1992.)

Day sampled	Bacteria/ml	Cell count ($\times 1000$ /ml)
1	2,800	880
2	6,000	144
4	7,000	104
5	10,000	896
13	>10,000	152
14	1,200	1,000
15	>10,000	168

Table 4.6. Stages in the transfer of *Staphylococcus aureus*, following the milking of an infected cow, to the teats of uninfected cows, using different hygiene routines. (From Bramley, 1981a.)

Hygiene applied	% swabs positive for <i>S. aureus</i> from			
	Teats before foremilk	Teats after foremilk	Teats after udder wash	Teats after milking
Water	0	29	63	97
Disinfectant, paper towel and gloves	0	16	39	79

Table 4.6 shows the effects on the next cows to be milked of milking a cow known to be infected with *S. aureus* in her udder. The control cows had been tested previously and shown to be free of *S. aureus* on their teat skin. Note how the level of contamination increases each time the teats are handled, and, even when strict hygiene is practised (gloves, disinfectant in the wash water and paper towel wipes), contamination still occurs.

Trials have shown that a cow shedding *S. aureus* in her milk may contaminate the teats of the next six to eight cows to be milked. The possible level of contamination is therefore enormous and will depend on factors such as the quality of the liners (avoid rough rubber, etc.), the initial amount of infection shed and the efficiency of the milking machine. We are, of course, talking about the degree of contamination of the teat skin and not about of actual udder infection. Most infections are killed by postdipping.

It is clear that, if *S. aureus* is present in a herd:

- Postmilking teat disinfection is vital. Even with optimal milking hygiene, it will be impossible to totally prevent the transfer of infection from cow to cow, but postdipping should destroy much of that infection on the teats before it can penetrate the teat canal.
- Ideally, infected cows should be milked last and in a separate group. Where this is impractical, milking known infected cows through a separate cluster, which can then be disinfected between uses, will considerably reduce the risk of spread from cow to cow.
- Consideration could be given to disinfect-

ing clusters between all cows. This is discussed in Chapter 6.

- Teat skin should be maintained in optimum condition. *S. aureus* is quite a resistant organism. It can live outside the mammary gland in sites such as udder cloths, the milker's hands and teat skin. Infections of the teat skin are particularly common if the skin is cracked or chapped, or if it has been damaged by pox virus infections, malfunction of the machine, warts, etc. This is another important reason for using a postmilking dip containing an emollient.

Acute gangrenous staphylococcal mastitis

Under certain circumstances, *S. aureus* can cause an acute gangrenous mastitis. This occurs following the production of large amounts of toxin. As shown in Table 4.7, the condition can be replicated experimentally by removing all immunity from the mammary gland, e.g. by infusing anti-bovine leucocyte antiserum, which eliminates all the white cells. A cow that may have been carrying a chronic *S. aureus* infection for months, or even years, can develop Staphylococcal gangrenous mastitis over just a few days. Gangrenous mastitis is not caused by a specific acute strain of *Staphylococcus* therefore, but rather by a change in the immune status of the udder.

The clinical appearance of gangrenous mastitis is shown in Plates 4.3 to 4.6. The skin of the teat and of lower parts of the udder, adjacent to the teat, develops a blue/black discoloration. It will probably be clammy and cold to the touch and may have a slightly sticky feel, due to a surface dis-

Table 4.7. Gangrenous *Staphylococcus* mastitis results from a reduced immune response and not a specific organism. Note how removing defensive white blood cells (PMNs, by infusing antiserum) leads to a huge rise in bacterial numbers and the rapid death of the cow.

Time (days)	PMN count (million/ml)	<i>S. aureus</i> count
1	3.5	10
2	5.0	22
Infuse antiserum		
3	9.0	37
4	0	14,000
5	(Died)	170,000



Plate 4.3. Acute gangrenous staphylococcal mastitis – note the blue/black discoloration. Other organisms such as *Bacillus cereus* and occasionally *E. coli* can produce similar changes.



Plate 4.5. The reddish brown, watery secretion, often mixed with gas, which is characteristic of gangrenous mastitis.



Plate 4.4. Blistered teat skin associated with gangrenous mastitis.



Plate 4.6. Severe gangrenous mastitis, leading to an udder slough. This cow should be culled.

charge. In some cases the surface of the skin forms small blisters, as in Plate 4.4.

Stripping the teat produces a dark, port-wine-coloured secretion (Plate 4.5), often mixed with gas. If the cow is very sick, then the prognosis is hopeless. Even in cows that are not seriously ill, there is a risk that at a later date the udder may slough and discharge the affected quarter (as in Plate 4.6). Badly affected cows are therefore best culled, although, if only a smaller part of the udder is necrotic, the necrotic tissue will eventually discharge and complete healing can be achieved.

However, one word of warning. Cows can also develop a bruised udder (Plate 4.7), causing blood to accumulate under the skin, resulting in a blue/black discoloration. These cows will be healthy in themselves, their milk will be normal, the quarter warm, not cold, and they will recover without any treatment. They should certainly not be treated or culled as a case of gangrenous mastitis.

Coagulase-negative staphylococci (CNS)

Coagulase-negative staphylococci (CNS organisms) have been identified as a cause



Plate 4.7. Udder bruising. This must not be confused with gangrenous mastitis.

of clinical and subclinical mastitis and rising cell counts. They are Gram-positive cocci that do not form clots with the tube coagulase test. They may be haemolytic or non-haemolytic, but this does not appear to affect their pathogenicity. Examples of coagulase-negative staphylococci are: *S. xylosus*, *S. intermedius*, *S. hyicus* and *S. epidermis*. They commonly colonize the teat skin, teat end and teat canal; hence it is difficult to be sure whether they are a cause of clinical or subclinical mastitis, or simply a teat-end contaminant. However, if milk culture from a cow with a raised cell count produces a pure growth of CNS, then these organisms are likely to be the cause of an udder infection producing the cell count response. It is very important to discard the first four to six squirts of milk before taking a sample for bacteriology. If this is not done, any CNS isolated may have originated from the teat canal only.

Increased bulk tank levels of CNS may result from poor postmilking teat disinfection or from poor teat skin condition. Such organisms are known to be present in maiden and pregnant heifers, and some authors have shown that this can lead to reduced yields post-partum. There are even data to show that intramammary treatment of heifers 6 months pre-partum will increase yields, but there are great dangers in doing this, namely the removal of the natural keratin plug and the introduction of new infections, e.g. coliforms, yeasts or moulds, when tubing.

Streptococcus agalactiae

Streptococcus agalactiae is a Gram-positive, alpha-haemolytic coccus. Its very small colonies have a bluish appearance on Edwards medium.

Streptococcus agalactiae is a highly contagious cause of mastitis and is easily transmitted from cow to cow during the milking process. Its primary reservoir of infection is in the udder, although it may occasionally colonize the teat canal and even the teat skin, especially if these surfaces are cracked. Its response to antibiotic

therapy is very good (see Table 4.4) and hence it should be possible to eliminate infection from a herd, provided that careful attention is also paid to the following six control points:

- Hygiene during milking.
- Postmilking teat disinfection.
- Dry cow therapy.
- Culling of chronic recurrent cases.
- Optimum milking machine function.
- Careful selection of replacement animals

Hence, if *S. agalactiae* is isolated from a herd, it is a good indication that there has been a breakdown in the basic hygiene of the milking routine.

Milk from infected quarters may contain massive numbers of bacteria, in some cases up to 100,000,000 (10^8) per ml. This can lead to an elevated and fluctuating TBC/Bactoscan in badly infected herds. In such herds, a dramatic response can be obtained by a system of blitz therapy. This involves treating all cows by infusing antibiotic into each quarter at three consecutive milkings. Almost all antibiotics are effective against *S. agalactiae*, allowing short withdrawal periods, if commercially available, to be used. Because response to therapy is good and because milk was only discarded for 24 hours after treatment, this used to be an economic procedure in badly infected herds. However, it must be carried out with strict attention to hygiene. Blitz therapy is discussed in more detail in Chapter 12 under treatment.

The level of *S. agalactiae* infection in an individual cow is much more closely associated with cell count than is *Staphylococcus aureus* infection. For this reason, it is possible to carry out partial blitz therapy, where only cows above a certain cell count are treated. This has also produced good results.

Streptococcus agalactiae is thought to penetrate the teat canal by slowly growing through it. Undermilking, which reduces the amount of flushing ('keratin stripping') of the canal, is hence thought to promote new infections. Although mainly an udder pathogen, *S. agalactiae* can also survive in

the environment. For example, it has been shown to persist on milkers' hands, particularly when the hands are badly cracked (as in Plate 6.1) and in this way it can be spread from farm to farm.

Subclinical infections are common, and these often lead to raised cell counts. Clinical signs may also be transient. For example, in one experiment, infusion of large numbers of *S. agalactiae* into a quarter caused severe clinical signs within 8 hours, but by 24 hours all clinical signs were gone and infection had become subclinical (Mackie *et al.*, 1983). Such cows will then be culture-negative but antibody positive and clearly represent a potential source of infection to other cows (Logan *et al.*, 1982). In contrast to *Staphylococcus aureus*, most infections lead to high levels of bacterial shedding, and culture is therefore a good diagnostic tool.

Streptococcus dysgalactiae

Streptococcus dysgalactiae is a Gram-positive haemolytic coccus that produces very small colonies and a green discoloration of Edwards medium.

Streptococcus dysgalactiae is the fourth major cause of contagious mastitis. As such it shares many of the properties and control methods applicable to *Staphylococcus aureus* and *Streptococcus agalactiae*. However, there are a few specific differences.

Streptococcus dysgalactiae survives well in the environment and has been considered by some to be halfway between contagious and environmental organisms. It is commonly found on teat skin, particularly when the surface integrity is compromised by chaps, cuts, machine damage, pox virus lesions, etc., and as such its presence in bulk milk samples is sometimes used as an indicator of teat skin damage. Mammary gland carriers are less important. *Streptococcus dysgalactiae* is also present on the tonsils and hence licking could transmit infection to teats. This could explain why *S. dysgalactiae* is a common cause of mastitis in heifers, including heifer calves, and dry cows. Teat irritation associated with flies or

chapping due to cold weather, might encourage an animal to lick its teats and hence transfer infection, which gradually colonizes the teat canal, until clinical mastitis occurs.

Finally, *S. dysgalactiae* is commonly found as part of the summer mastitis complex (see Chapter 13) and can be isolated from the carrier fly, the sheep-head fly *Hydrotea irritans*.

***Mycoplasma* species**

Mycoplasma colonies are slow-growing (10 days) and are said to have a typical 'poached egg' shape when grown on blood agar. *Mycoplasma* needs special culturing facilities and cannot be grown using the techniques described on pages 56–57.

There are two common species of *Mycoplasma* that cause mastitis: *Mycoplasma bovis* and *Mycoplasma californicum*. They are highly contagious and can rapidly spread in an infected herd. Response to antibiotics is poor and, once identified, infected cows should be milked last, in a separate group, and monitored until self-cure has occurred. However, most cows have to be culled. Although infected cows may not be clinically ill, infection can lead to a pronounced drop in yield, often referred to as agalactia (meaning 'no milk'). Affected quarters may be swollen and produce only a scant 'gritty' or sandy, watery secretion.

As this is a highly contagious organism, strict attention must be paid to hygiene at milking. This should include flushing or even pasteurization of the clusters between cows (see Chapter 6). Both clinically infected and subclinical carriers shed large numbers of the organism.

Mycoplasma bovis can also be a cause of joint infections and of pneumonia in calves.

Environmental Organisms

This section describes the coliforms, including *E. coli*, and, of course, *Streptococcus uberis*. As the dry period plays such an

important part in the epidemiology of environmental organisms, a description of the organisms is followed by a section on the importance of dry period infections.

Coliforms including *Escherichia coli*

Escherichia coli is a Gram-negative bacillus that produces grey mucoid colonies on blood agar. There are haemolytic and non-haemolytic strains.

After *S. uberis*, *E. coli* is the most prevalent environmental organism causing mastitis. It is present in large numbers in faeces and hence infection occurs primarily in housed cows, when conditions are wet and humid and when hygiene is poor. Environmental factors are discussed in more detail in Chapter 8. During lactation, *E. coli* is thought to penetrate the teat canal by propulsion and hence increased mastitis is seen with dirty teats, suboptimal machine function or techniques that lead to teat-end impacts. Penetration of the open teat canal immediately postmilking may also be a factor.

E. coli penetration of the teat canal by no means always causes clinical mastitis; in fact, quite high numbers (80–90%) of infections undergo self-cure. In some cows, the only detectable change is a rise in cell count and in bacterial numbers. In others, very slight damage to the endothelial lining of the teat wall produces just a few white flaky clots, which have disappeared by the next milking. The symptoms of typical *E. coli* mastitis are a hard, hot swollen quarter, with a watery discharge. A proportion of cows develop a severe shock reaction and can die within hours. This variation in the response of the cow to invading *E. coli* and the reasons for the wide differences in clinical signs are discussed in Chapter 3.

Unlike *Staphylococcus aureus* and *Streptococcus agalactiae*, *E. coli* does not adhere to the endothelial lining of the teat and udder cisterns. This is probably one reason why chronic carrier cows with recurrent bouts of *E. coli* mastitis are rare.

Escherichia coli toxins

The toxic effects of *E. coli* mastitis are due to the release of an endotoxin that is a lipopolysaccharide (LPS) derived from the bacterial cell wall the LPS is removed by phagocytosing PMNs (see page 27), which in turn release lysosomal granules, further exacerbating the shock reaction.

Plate 4.8 shows a normal teat in cross-section, with a creamy-pink-coloured teat cistern wall. Contrast this with the intense haemorrhage of the endothelial lining of the teat wall in Plate 4.9, which was taken from a cow that died as a result of *E. coli* mastitis. If *E. coli* reaches the smaller ducts and lactiferous sinuses of the main gland, then a massive multiplication of bacteria occurs, and this leads to a severe response in the cow. Sometimes damage to blood vessels is so great that serum ooze is seen on the surface of the udder and teat, as in Plate 4.10.



Plate 4.8. A normal teat in cross-section.

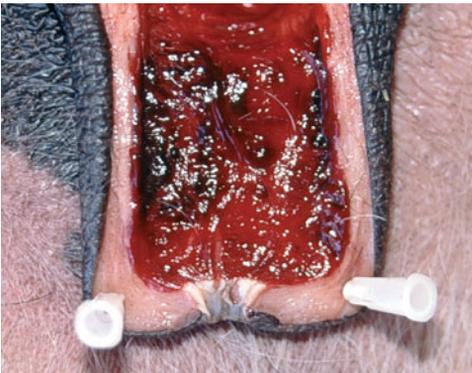


Plate 4.9. A teat infected with *E. coli* mastitis. Note the intense haemorrhage on the teat wall.

Such cases can lead to extensive gangrene and resultant sloughing of udder tissue, as seen in Plate 4.6.

Dry period coliform infections

Although lactoferrin prevents clinical dry cow coliform mastitis, many new subclinical infections are contracted during the dry period, especially in the first and last 2 weeks. These infections remain dormant in the udder and commonly produce clinical mastitis during the first 100 days of lactation. Dry period infections are common for both *E. coli* and *S. uberis*, and are dealt with in detail later in this chapter.

Variation in strains of Escherichia coli

When a herd outbreak of severe *E. coli* mastitis occurs, it is unlikely to be caused by the same strain of *E. coli* in every cow, even though clinically this may appear to be the case. There are usually a number of strains involved in a severe challenge. For example, in one survey of 290 isolates from cases of acute *E. coli* mastitis:

- 82% were typed as 63 different strains of *E. coli*.
- 18% could not be typed.

Cow-to-cow transmission, as occurs with contagious mastitis, is therefore unlikely to be important. A severe outbreak of coliform



Plate 4.10. *E. coli* mastitis. Damage to blood vessels may be so extensive that serum oozes through the surface of the udder skin, as well as into the mammary gland.

mastitis is likely to be caused either by a heavy environmental challenge, e.g. increased exposure from the environment, dry period and/or milking machine function producing high teat-end contamination, or a decreased immune response.

Vaccination against *Escherichia coli*

The toxic effects of *E. coli* are produced by an endotoxin that chemically is an LPS derived from the cell wall. Each time one *E. coli* divides into two (and this happens every 20 minutes under the ideal conditions of warm milk within the mammary gland), a certain amount of LPS is released. In addition, when the *E. coli* die, further quantities of LPS are released. Although the LPSs produced by the various strains of *E. coli* are all different, there is one strain known as a 'rough mutant', that produces LPS which has a fragment that is common to all LPSs produced by all other strains. This forms the basis of what is known as the J5 vaccine. The manufacturers claim 80% protection following three subcutaneous injections of an oil adjuvant vaccine given at drying off, 28 days later and within 2 weeks after calving. The results of one field trial, in which half of each herd was vaccinated and the other half was left as a control, are shown in Table 4.8. The vaccine does not result in any fewer new infections. There is a reduction in clinical cases, and a marked reduction in acute toxic cases, which become particularly rare.

Table 4.8. Response to J5 *E. coli* vaccine. (From A.W. Hill, personal communication, 1922.)

	No. of cows	Cases of conform mastitis	% infected
Vaccinated	233	6	2.6
Non-vaccinated	227	29	12.8

However, it is interesting that the vaccine does not protect against experimental challenge by *E. coli*, and a good deal of work has attempted to explain this difference. A logical explanation would be that the vaccine in some way alters the method by

which *E. coli* infections penetrate the teat canal or become established in the udder. This has still to be clarified.

Chronic recurrent coliforms

In the majority of herds, response to *E. coli* infection is very prompt and the organisms are rapidly eliminated through the natural response of the cows, e.g. within 12–36 hours. These cows have a hard swollen quarter with a brown watery secretion, but often there are no residual bacteria present when the quarter is sampled. Cows that mount a poor inflammatory response (see pages 27–29) become very sick and *E. coli* may persist within the udder for 10–14 days, despite the use of antibiotics. This persistent presence of *E. coli* can act as a chronic irritant, leading to hyperplasia (abnormal cell growth) and keratinization of the gland, and the quarter then dries up. Many such quarters become productive again in the next lactation.

In a Dutch study, 5% of all clinical coliform infections resulted in chronic recurrent mastitis, and this figure may be greater with high initial *E. coli* numbers and when antibiotics are not used. In a UK study (Bradley and Green, 1998), 35% of coliform cases (20 cases in 13 quarters) recurred if infections originated during the dry period, and 17% (18 cases in 15 quarters) recurred if they originated during lactation. It would therefore appear that chronic infections are more likely to arise from the dry period.

Other coliforms

There are a range of other organisms, in addition to *E. coli*, which fall into the general category of coliform mastitis and may be isolated from time to time. These include the following:

- *Enterobacter aerogenes*.
- *Citrobacter*.
- *Klebsiella pneumoniae*. This organism may be found in damp, stored sawdust from freshly felled wood. It can produce a

severe, toxic mastitis if the sawdust or wood product is used for cubicle (free-stall) bedding.

- *Pseudomonas aeruginosa*. Typically, *Pseudomonas* originates from contaminated water, found, for example, in udder wash header tanks that are maintained at a low, warm heat and that do not have lids over them or sanitizer added to them. *Pseudomonas* may also be found in water from boreholes. The clinical signs vary enormously, from acute toxic mastitis to chronic recurrent cases. Response to treatment is poor, probably because the organism can live inside cells where it is not accessible to antibiotics. Hence, chronically infected cows with high cell counts may have to be culled, since they represent a source of infection to other cows (though it is perhaps not too serious, as only low numbers of organisms are shed).
- Non-lactose-fermenting coliforms, often referred to as NLFs, have become an increasingly common cause of clinical mastitis. Many of these are also environmental, i.e. non-enteric *Pseudomonas* species.

Streptococcus uberis

Streptococcus uberis grows as a non-haemolytic, Gram-positive coccus, producing brown colonies on an Edwards plate, due to splitting of aesculin. Some workers categorize all aesculin-splitting streptococci as *S. uberis*. However, this is incorrect as there are many other examples, including *Streptococcus faecium* and *Streptococcus bovis*.

A typical case of *S. uberis* mastitis is often sudden in onset, and produces a hard, swollen quarter, with large white clots in the milk, and sometimes with a high, or very high, body temperature.

Variation in strains of Streptococcus uberis

DNA fingerprinting studies have shown that *S. uberis* is not a single organism, but a range of organisms. Some strains of *S. uberis* are much more resistant to opsonization (coat-

ing of the bacteria with antibody: see Chapter 3), and hence phagocytosis (engulfing) and destruction of these strains of bacteria by white cells is also poor. This is particularly so in the presence of the milk protein casein (see Fig. 4.1). Although *S. uberis* was initially considered to be an environmental organism, it is now also known that some strains can be a cause of chronic, recurrent and subclinical mastitis, with a poor response to antibiotics.

Prompt antibiotic therapy is therefore important, although for reasons given later, response to treatment is often poor. This results in subclinically infected cases, which then spread further infection from cow to cow during the milking process.

Sources of infection

Streptococcus uberis is currently the most common environmental organism causing mastitis in the UK. It is particularly associated with straw yards, where a very high level of infection may occur. Up to 1,000,000 (10^6) organisms/g of straw bedding have been reported. Fig. 4.2 shows the correlation between the level of *S. uberis*/g of straw bedding and incidence of *S. uberis* mastitis in the herds surveyed. Note that the majority of problem herds were associated with high levels of *S. uberis* in the bedding. This has led to a move away from straw and especially straw yards, and into sand-bedded cubicles. Clean sand supports only a low level of *S. uberis* and *E. coli*, and, if the pH of the bedding can be kept high, for example using lime or ash from power station waste, this further reduces bacterial growth. Contaminated sand, e.g. with milk, urine or faeces, will, of course, support bacterial growth.

In addition to being in the environment, *S. uberis* can also be found on a wide range of sites on an animal, such as the mouth, vulva, groin and axilla, as shown in Table 4.9.

Although present in faeces, levels are not particularly high, and in this respect *S. uberis* differs from *E. coli*.

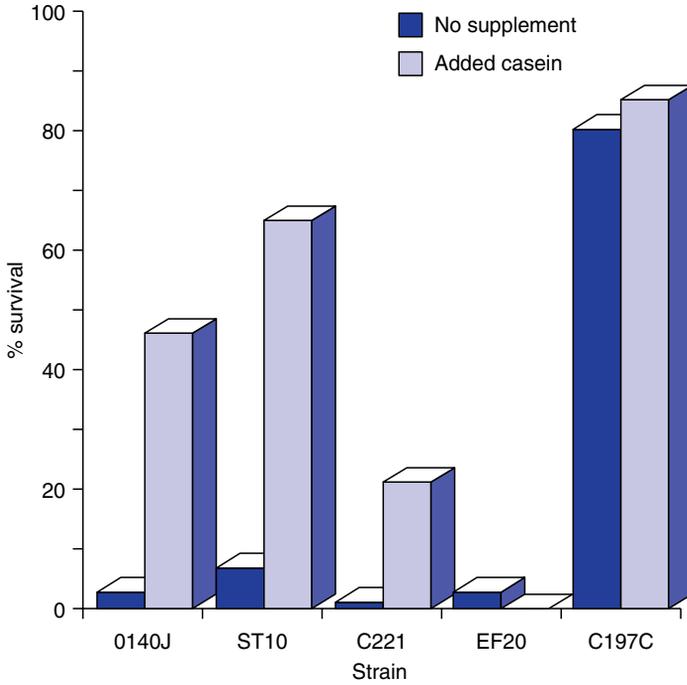


Fig. 4.1. Phagocytic resistance of *Streptococcus uberis* grown with and without casein. Most strains appear to be protected from phagocytosis in the presence of casein. (From Hill, 1992b.)

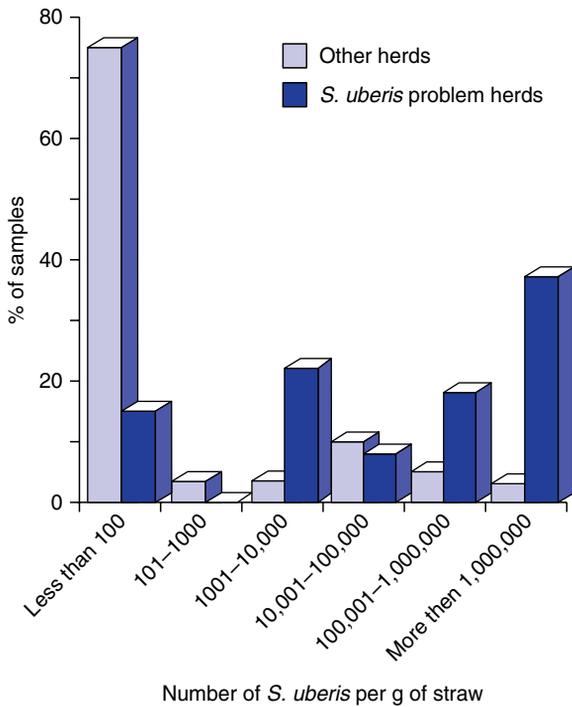


Fig. 4.2. Levels of *Streptococcus uberis* in straw bedding in *S. uberis* problem herds compared with other herds. (From Hill, 1992a.)

Table 4.9. Isolation of *Streptococcus uberis* from 53 in-calf heifers kept at pasture under dry sunny conditions. (From Bramley, 1981b.)

Site examined	No. positive	% positive
Escutcheon	48	90
Legs	46	86
Hind teats	49	92
Lips	12	22
Total	155	73

Spread within the herd

The initial infection may be from the environment or from the purchase of an infected cow, but *S. uberis* can rapidly spread within the herd. Following infection, *S. uberis* is known to remain refractory in the udder for quite a long period. In one study, this was an average of 1½ months after infection and treatment. This poor treatment response rate and the long refractory period within the udder are thought to be due to a number of factors, for example:

- Its resistance to phagocytosis, caused by poor opsonization.
- It can exist inside cells, where it is protected from the action of many antibiotics.
- It can enter the mammary lymph node, from where it maintains a reservoir of infection.

In one experimental infection, *S. uberis* had reached the lymph node in as little as 6 days post inoculation. Hence, in herds where *S. uberis* is a problem, treatment of clinical cases needs to be long and aggressive, to try to avoid the carrier state.

Some cows experimentally infected with *S. uberis* recover, but only after prolonged treatment with antibiotics, for example, 5–7 days of combined parenteral (by injection) and intramammary therapy. These are presumably the ‘chronic mastitic’ strains, which are related more closely to contagious than to environmental pathogens.

As further evidence of the contagious nature of *S. uberis*, it has been shown that the prevalence of infected quarters in a herd is a good predictor for the incidence of new

infections. Hence, if the level of *S. uberis* infected quarters is low, then the future clinical incidence will remain low. However, as the level of infected quarters rises (possibly initially from an environmental or dry period source), so will the future number of clinical cases. This is commonly seen in practice. There will be an initial outbreak and the source of the outbreak may be identified and corrected, but clinical cases continue for several months due to recycling of infection within the herd. Often culling of these ‘reservoir’ cases is the only control option. As the organism shows both contagious and environmental properties, then both predip and postdip are relevant for control.

Although most herds already have many different strains of *S. uberis* present, the introduction of another new strain can still cause a severe outbreak, all of the same new strain. This suggests that strain cross-protection is limited, and shows the importance of biosecurity. Bulk tank levels of *S. uberis* give a reasonable assessment of herd status, because strains in the udder are generally the same as those found in the bulk tank, i.e. they come from the udder and not from the environment.

Streptococcus uberis in the dry period

Streptococcus uberis is the most common new infection of dry cows, especially during the first and last 2 weeks of the dry period. These infections lie dormant in the udder to produce clinical disease in early lactation. Dry cow therapy and environmental hygiene are therefore very important in control. One study (Williamson *et al.*, 1995) showed that 12.3% of control cows were infected with *S. uberis* at calving, compared with only 1.2% of quarters given antibiotic dry cow therapy. Dry period infections are dealt with in detail in the next section.

Outbreaks at pasture

Streptococcus uberis mastitis outbreaks sometimes occur in cows at pasture, especially in late summer. The most probable cause is that, during the summer especially,

cows often tend to lie on the same area at night, which could then develop a build-up of infection. It is advisable to move cows every 2 weeks, to avoid this build-up of infection, and not let them back to the same paddock for at least 2 and preferably 4 weeks. Other factors leading to outbreaks at pasture include the contagious transmission of 'chronic' strains, or perhaps cows transmitting oral infections by licking teats irritated by flies.

Vaccination against Streptococcus uberis

Because there is such a wide range of strains of *S. uberis*, it is important to find a single antigen that is present in all strains. Leigh (2000) found that most strains produce the enzyme PauA, which activates plasminogen thus releasing casein and other nutrients from milk to allow the organism to grow. An experimental vaccine against PauA has been found to be multi-strain effective, without leading to increase in mammary PMNs, although to date there are no commercial products available.

Dry Period Infections

Dry period infections are an extremely important part of the epidemiology of environmental pathogens such as *E. coli* and *S. uberis*. These infections often remain dormant, i.e. subclinical, throughout the dry period, but are then an important cause of clinical mastitis in the first few months of the next lactation. To fully understand this process, it is necessary to examine what happens in the dry period in some detail.

The following section examines the changes that take place during the dry period, when infections occur, environmental and other factors leading to an increased level of new infections, and finally the immune response, i.e. the way in which the cow modulates the outcome of these new infections.

Phases of the dry period

There are three phases of the dry period:

1. The first 2 weeks. The teat canal slowly closes, and a plug of keratin and lipid is excreted into the lumen of the canal to form a teat seal. Mammary alveoli (milk-secreting tissue) slowly regress.
2. The rest phase during the mid dry period. The secretory tissue is dormant at this stage, and there is a build-up of natural inhibitory substances, such as lactoferrin, neutrophils, NAGase (*N*-acetylglucosaminidase) and, of course, immunoglobulins, especially towards the end of the rest phase.
3. The last 2 weeks before calving. New secretory tissue forms, i.e. new mammary alveoli, and the keratin plug slowly dissolves ready for the start of the next lactation.

It is during the first and last 2 weeks of the dry period, i.e. when the teat canal keratin plug is forming and then dissolving, that the cow is especially susceptible to new infections. This is shown in Fig. 4.3. This graph shows that cows in late lactation (a) only develop a low level of new infections, but immediately after drying off there is a big increase in new infections, namely at (b). These infections do not develop into clinical cases at this stage however. During the central dry period (c), the level of new infections is very low and many residual infections from the previous lactation are eliminated. The maximum level of new infections occurs at (d), just before and just after calving, which is when the teat canal keratin plug is dissolving and milk is starting to accumulate in the udder. The period just before and just after calving is therefore the time of major risk for new dry period infections, and this is when management of the cow should be at its highest.

As stated above, these new infections do not appear as disease, i.e. as clinical mastitis, during the dry period. The majority remain dormant in the udder, and do not appear as clinical disease until early lactation. In Fig. 4.4 it can be seen that most new cases of

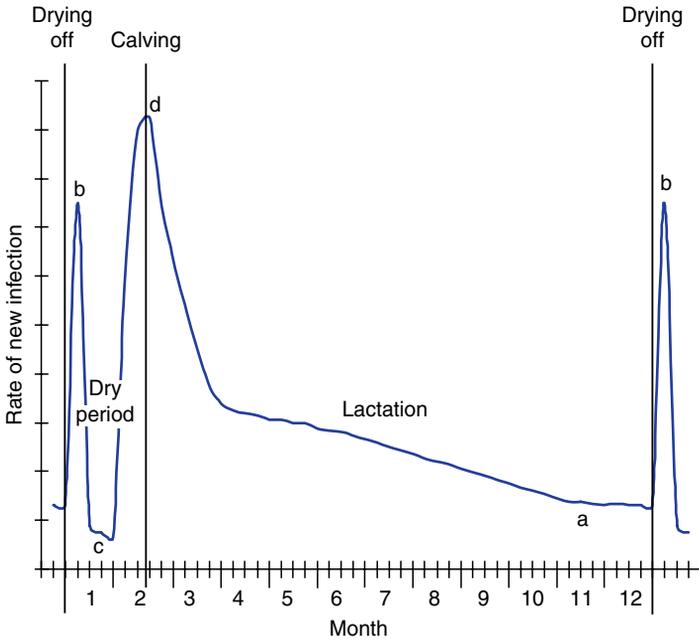


Fig. 4.3. This figure shows the incidence of new intramammary infections (IMIs) over a lactation. There is a low level in late lactation. This dramatically increases just after drying off, falls again in the mid dry period and reaches a peak around calving (Green *et al.*, 2002).

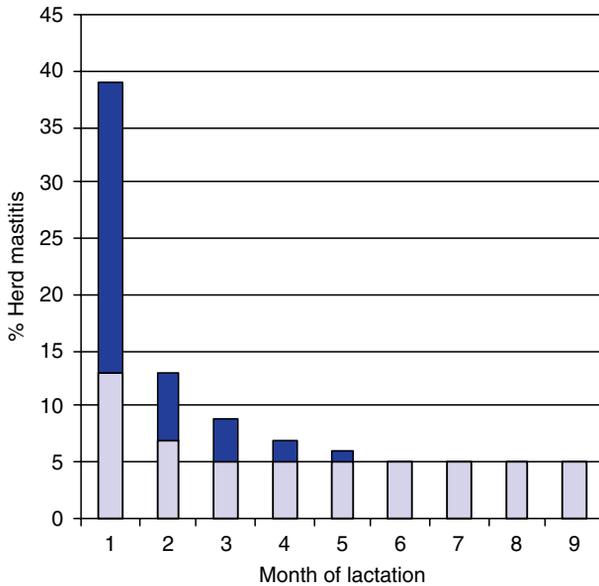


Fig. 4.4. New infections contracted during the dry period lead to clinical mastitis during the first 4 months after calving. The light blue bars represent clinical mastitis arising from lactation infections, and the dark blue bars are from dry period infections (Green *et al.*, 2002).

mastitis occur in the first 4 weeks of lactation, and, of these clinical cases, some 60% originate from infections that have become established during the dry period. This was despite the use of dry cow antibiotic therapy. It should also be noted that dry period infections continued to cause clinical mastitis up until the fifth month after calving.

The process whereby a keratin plug is formed in the teat canal during the first 2 weeks after drying off was described above. Unfortunately, many cows do not form an effective teat seal, and in these cows the risk of new infections is even greater. Woolford *et al.* (1998) in New Zealand showed that 97% of dry period mastitis infections were in 'open' quarters, i.e. quarters that had not developed a good teat seal. The slow development of the teat seal is shown in Fig. 4.5. Even at 45 days after drying off, 25% of teats had not formed an effective teat seal, i.e. they were still 'open'.

The effectiveness of the teat seal varies with factors such as the following:

- Overall production. Cows with higher total lactation yields form a less effective seal.
- Milk flow rates. Fast milkers form a less effective seal, and they are more likely to leak milk. For example, in a trial in the Netherlands, Schukken *et al.* (1993) showed that cows that leak milk are four

times more likely to develop mastitis in the dry period.

- Milk yield at drying off. The higher the yield at drying off, the higher will be the risk of an ineffective keratin plug forming. Dingwell *et al.* (2004) in the USA showed that new infections developed in 26% of cows with drying off yields of greater than 21 kg, but in only 16% of cows where the drying off yield was less than 13 kg. Bradley and Green (1998) have shown that every 1 litre increase in yield at drying off produces a 6% increase in the risk of a new dry period infection. It is therefore essential that the level of yield is decreased before drying off. This can be achieved by either feeding or management, but it should not be done by milking once a day or alternate-day milking.
- Teat-end damage. Dingwell *et al.* (2004) showed that cows with a significant level of teat-end damage were 1.7 times more likely to develop a new dry period infection. Teat end damage is a risk factor for mastitis in both the dry period and lactation therefore.
- Dry cow therapy. Woolford *et al.* (1998) showed that cows given dry cow therapy (DCT) were *twice* as likely to form a good seal. Presumably this is because teat canal organisms degrade keratin, and their removal with DCT leads to a more effective seal.

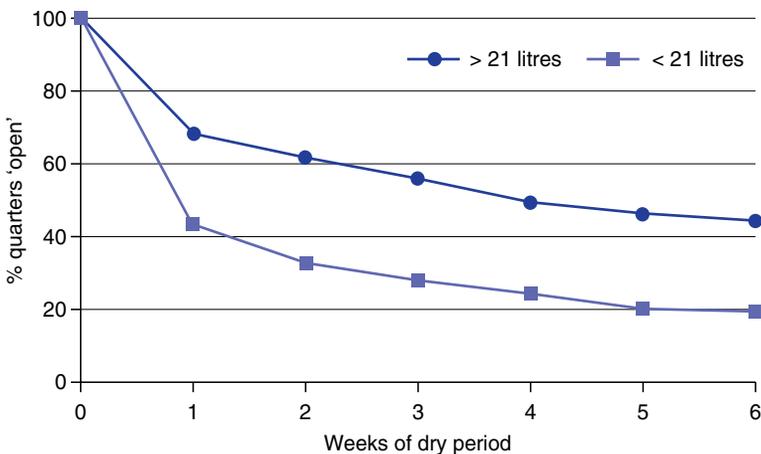


Fig. 4.5. Keratin plug formation. Although the keratin plug is a very effective teat closure mechanism, unfortunately many cows do not form an effective seal. This is especially the case in higher-yielding animals, cows with teat-end damage and cows with high yields at drying off (Dingwell *et al.*, 2002).

The correct procedure for drying off cows is given in Chapter 12. In addition to the above factors, the major methods of reducing dry period infections are: (i) dry period antibiotic therapy; (ii) internal and external synthetic teat sealants; and (iii) environmental management. These are discussed in more detail in Chapters 8 and 12.

The host immune response

In their work, Bradley and Green (1998) found that from 1200 quarters sampled during the dry period there were 154 dry quarters that developed coliform infections, and of these 13 (8.4%) developed clinical coliform mastitis with the same organism during the next lactation. In contrast, of the 1043 quarters that were not infected during the dry period, only 15 (1.4%) developed clinical coliform mastitis in the next lactation.

Quarters subclinically or latently infected with coliforms in the dry period are therefore almost six times more likely to develop clinical mastitis during lactation. DNA fingerprinting studies showed that it was the same organism that carried over into lactation. In fact, over 50% of all clinical coliform mastitis cases seen in early lactation are as a result of infections in the dry period, so, when investigating a herd outbreak, attention to dry cow environment, management and hygiene at dry cow tubing is essential. It would also seem logical to select a dry cow antibiotic that is effective against coliforms, and there are some data to show that framycetin may be more effective.

However, perhaps one of the most interesting features of dry period infections is not the number of clinical cases of mastitis that originate from the dry period, but rather the number of new infections that undergo spontaneous recovery. From the above, it can be seen that only 8.4% of quarters infected during the dry period developed clinical mastitis during lactation, i.e. over 90% of cases recovered. An important factor for the dairy farmer is to understand what modulates the immune response, i.e. what

makes the 91.6% of cows recover. There is no doubt that management and stress play a part. In a study of cases of toxic mastitis in Northern Ireland, Menzies *et al.* (2003) showed that cows with milk fever are 23 times more likely to get toxic mastitis, and that cows with assisted calvings are 11 times more likely to get toxic mastitis.

It is also well known that all cows undergo a suppression of the immune system during the 2 weeks before and the 2 weeks after calving, and this renders them much more susceptible to disease over this period. The extent of the immune suppression can be estimated by measures such as the speed of the neutrophil response to bacterial invasion and the efficiency with which selectins pull neutrophils through the capillary wall in response to inflammation (see Fig. 3.4). The evolutionary reasons suggested for this reduction in the immune response include the following:

- As the fetus is antigenically different from the dam, there is a risk that 'leakage' of fetal fluid into maternal circulation would lead to a hypersensitivity reaction.
- A reduced immune response will reduce reaction to trauma that might occur in the birth canal during parturition.
- Transfer of antibodies into colostrum may decrease circulating maternal antibody.

The influence of dry matter intake

All dairy farmers know the importance of getting a cow to eat after calving, and one of the major factors influencing the expression of the immune system is the level of food intake at this stage. Dry matter intake (DMI) starts to fall approximately 2 weeks precalving, reducing from around 2.5% of body weight to less than 2%, e.g. down to only 10 to 12 kg for a 600 kg cow (Fig. 4.6). On the day of calving, the rate of rumination slows or almost stops, and this further decreases food intake. Ample long fibre in the diet stimulates the resumption of rumen activity and hence feed intake. Cows that maintain a reasonable feed intake and those that

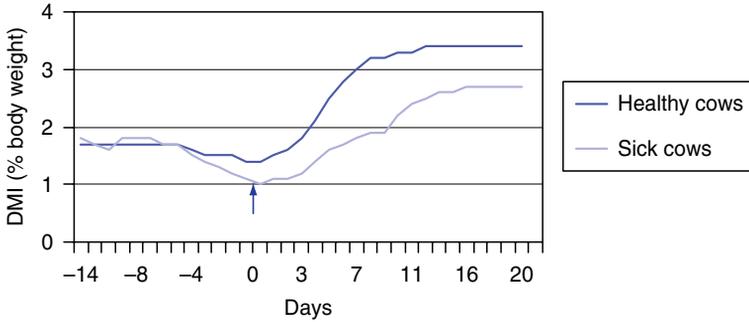


Fig. 4.6. Depression of intake at calving. All cows experience a fall in dry matter intake (DMI) around the time of calving (↑). The cows with a larger fall in DMI and those slower to recover intakes in early lactation are more likely to experience metabolic disorders such as milk fever, retained placenta, ketosis, displaced abomasum and mastitis.

quickly regain food intake after calving are less likely to develop metabolic disorders such as milk fever, metritis, ketosis, displaced abomasums, cystic ovaries, and of course, mastitis. Cows that are overfat have lower DMIs and are more likely to develop metabolic disorders and mastitis. On this definition, therefore, mastitis might even be considered to be a metabolic disorder, in that many cows become *infected* with mastitis pathogens but only a few are affected, i.e. progress to develop clinical disease.

Short dry periods

All dairy farmers also know that, if a cow has been dry for a very long time, she has an increased risk of developing mastitis and other metabolic disorders when she does eventually calve. A dry period of 8 weeks was originally considered ideal, but recent work has shown benefits from dropping this to 5–6 weeks or less. Shorter dry periods mean that the cow has an additional 2–3 weeks of lactation, and that she does not progress into the same level of ‘metabolic shutdown’. There is some evidence that cows with both shorter and longer dry periods have increased cell counts in the next lactation, but cows with shorter dry periods have less risk of developing clinical mastitis.

Less Common Causes of Mastitis

Bacillus species

Bacillus species are seen as both haemolytic and non-haemolytic Gram-positive bacilli with characteristic large, rough, dry, flaky colonies on blood agar.

There are two common *Bacillus* species causing mastitis: *B. cereus* and *B. licheniformis*. Great care must be taken with sampling, since *Bacillus* species can also be a contaminant of the teat canal and not associated with mastitis. It is essential to discard four to six squirts of foremilk before taking a sample.

Bacillus cereus

This is classically associated with infected brewers’ grains and may produce an acute, gangrenous mastitis (see Plates 4.3 to 4.6). It can also occur as a contaminant on dry cow syringes, warmed in a bucket of contaminated water for ease of administration. Bacteria (including *Pseudomonas*) infused at this stage may persist in the udder to produce acute mastitis after drying off or occasionally at the next calving.

Bacillus licheniformis

This is an environmental organism to which cows are particularly vulnerable if they lie

on waste silage left beside feed troughs. This is especially the case with maize silage, which undergoes more rapid secondary fermentation than other types and therefore produces a warm bed. Poor cubicle comfort, leading to increased numbers of cows lying outside, may also be involved. *Bacillus licheniformis* infections ascending into the vagina may also lead to an increase in endometritis ('whites'), low conception rates and abortions later in pregnancy.

Clinically, *Bacillus* species mastitis is often presented as a hard quarter with white clots. Although sensitivity tests often indicate that a wide range of antibiotics should be effective for treatment, response is often disappointing.

Yeasts, fungi and moulds

Yeasts, fungi and moulds grow slowly on blood agar and are best cultured on Sabouraud's media. Examples of yeasts include *Candida* and *Prototheca*, and *Aspergillus* is a common fungal infection. Yeasts will be seen as Gram-positive bottle-shaped organisms on smear.

Yeasts and moulds are common environmental organisms. They may cause mastitis if the straw or other bedding is wet and/or mouldy, for example from being stored outside. Yeast mastitis may occur if a large number of cows are lying out of the cubicles (free-stalls) or if the milker washes the teats but does not wipe them dry before applying the milking units. This is particularly the case if the water is contaminated and non-sanitized. On farms where wet teats are a problem, heavy contamination of teat skin leads to infection in bulk milk. It may be possible to culture *Candida* species (yeasts), *Aspergillus fumigatus* (moulds) or *Prototheca zopfii* (algae) from both bulk milk and cases of clinical mastitis.

Clinically, the mastitis is most commonly seen as a hard, hot and swollen quarter, with thick white clots. The cow may have an elevated temperature, especially with yeast (*Candida*) mastitis. Treatment with antibiotics is totally ineffective, as yeasts and fungi do not respond to anti-

biotics. Significant success has been reported from infusing 60–100 ml of a mixture of 1.8 g iodine crystals in 2 litres liquid paraffin, plus 23 ml ether, into the quarter, daily for 2–3 days. On each occasion, the infusion should be stripped out after 6–8 hours, otherwise the iodine can produce excessive irritation and be a cause of inflammation in itself. Intravenous sodium iodide or oral potassium iodide given concurrently sometimes improves response to treatment.

As the mixture is unlicensed, i.e. it is an 'off label' treatment, standard milk withdrawal times apply.

Relatively rare causes of mastitis

The following list includes some of the minor species causing mastitis:

- *Arcanobacterium pyogenes*. This is discussed in Chapter 13 under summer mastitis.
- *Corynebacterium bovis*: can cause sub-clinical mastitis and raised cell counts. Has been associated with poor/delayed postmilking teat disinfection. May also be isolated from the teat canal and not associated with mastitis.
- *Streptococcus faecalis*: present in faeces and a common contaminant in samples. Could be a cause of mastitis if isolated in pure culture.
- *Leptospira hardjo*: seen in conjunction with abortions and milk drop as part of the leptospirosis complex. Very difficult to culture.
- *Nocardia asteroides*: very hard quarter. Poor response to antibiotics.
- *Streptococcus zooepidemicus*.
- *Pasteurella/Mannheimia* species: environmental organisms. Have been associated with warming drying off tubes in contaminated water prior to infusion.
- *Serratia* species: can cause mastitis in both dry and lactating cows. Several species but *S. marcescens* is the most common. Non-pigmented strains are thought to be more pathogenic than pigmented strains.

- *Salmonella*: has possible human health implications.
- *Corynebacterium ulcerans*: has possible human health implications (sore throats).
- *Listeria monocytogenes*: has possible human health implications and has been associated with soft cheese.
- *Mycobacterium smegmatis*.
- *Yersinia pseudotuberculosis*: common infection of wild birds, especially starlings.
- *Haemophilus somnus*.

Specialist texts should be consulted for more details of these organisms. (See 'Further Reading' section.)

Culturing Milk for Bacteria

Culturing a pretreatment mastitic milk sample is a standard procedure in the investigation of mastitis. The major reasons for doing this are:

1. It is obviously important to know which organism(s) you are dealing with before tackling a mastitis problem. This is because of the differences in the epidemiology and subsequent control methods required for contagious and environmental mastitis.
2. A knowledge of the organism will also help to determine treatment strategies. For example with extensive *Streptococcus agalactiae* infection, blitz therapy might be considered and, with chronic *Staphylococcus aureus* infection, culling would be the likely option. This is dealt with in more detail in the treatment section in Chapter 12.
3. Knowledge of the organism involved will also help to determine if mastitis organisms are part of a concurrent TBC/Bactoscan problem.

Taking a milk sample

The quality of the results (and hence value for money) obtained from submitting a milk sample to the laboratory is to a very large

extent determined by the quality of the initial sample. The teat end is often heavily contaminated by a range of environmental bacteria, and by normal commensal teat-end organisms, but, of course, they are not necessarily the cause of the mastitis. Some may even penetrate the outer areas of the teat canal. To determine which bacteria are the causes of mastitis, samples must be taken very carefully. The following procedure should give good results:

1. Make sure the operator has clean hands. Wash and dry if necessary, or wear clean gloves.
2. Wash and thoroughly dry the teat if it appears dirty.
3. Strip out and discard the first four to six squirts, thus flushing out non-mastitic bacteria from the teat canal.
4. Predip and wipe.
5. Thoroughly scrub the teat end with a swab soaked in surgical spirit or similar, until the swab remains clean. Only then should the top be removed from the sample bottle.
6. Hold the bottle at an angle of 45° or less and draw out one squirt of milk in a diagonal direction. If the bottle is held vertically there is a much greater risk of dust and debris falling into the sample during stripping. One good 'draw' of milk is sufficient. It is not necessary to fill the bottle.
7. Replace the cap and label the bottle with cow identity, quarter, date and farm name.
8. Store the sample in the fridge (+4°C) until it can be transported to a laboratory. Ideally, samples should be plated out within 60–90 minutes, as this gives the best results. Storage for up to 72 hours at 4°C is acceptable. Freezing reduces bacterial numbers for some organisms (especially for coliforms) but can still give useful results. If possible, transport to the laboratory in ice packs.

Laboratory plating and incubation

Milk samples are cultured on agar plates. These contain special media (e.g. blood) that provide food for bacterial growth.

There is a wide range of techniques available, the variation suiting different needs. The method described in the following protocol is currently used by the authors, as it suits their requirements in veterinary practice. Although not the cheapest method, it allows fairly rapid identification of the major groups of mastitic organisms.

1. Preheat samples to at least room temperature to break down the fat globules, thus releasing trapped bacteria.
2. Use a sterile cotton wool swab to streak the initial plate. A standard 7.0 mm bacteriological loop contains only 0.05 ml milk and such a low volume could miss mastitic organisms that are only present in low numbers in the milk (e.g. less than 20/ml).
3. Plate out on to the following media:
 - Sheep blood agar (Columbia).
 - MacConkey.
 - Edwards.
 - Sabouraud, especially if yeasts and fungi/moulds are suspected (although these organisms will also grow slowly on blood agar).
4. Incubate plates at 37°C and examine after 24 hours and again at 48 hours.
5. Gram-stain colonies to determine the organism morphology (structure) and to determine whether it is Gram-positive or Gram-negative.

Bacteria are differentiated in many different ways: from their appearance on culture plates, their size, their shape and their reaction to a stain known as the Gram stain. For example:

- Shape: most bacteria are shaped as either
 - spheres (cocci), e.g. *Staphylococcus* or *Streptococcus*; or
 - or rods (bacilli), e.g. *Bacillus cereus*.
- Gram stain: bacteria stain either
 - Gram-positive: dark purple, e.g. *Staphylococcus* or *Streptococcus*; or
 - Gram-negative: pink, e.g. *E. coli*, *Pasteurella* or *Pseudomonas*.

Bacilli are often Gram-negative (but not always: *Bacillus* species are Gram-positive) and cocci are often Gram-positive. This distinction between

Gram-positive and Gram-negative bacteria is quite important when comparing the antibiotic sensitivity of different organisms.

- Haemolysis: some bacteria break down blood, to give a ring of haemolysis or 'clearing' of the blood around colonies growing on the agar plate, as seen in Plate 4.1.
6. Carry out other useful bacteriological differentiation tests, which include those for:
 - Coagulase.
 - Oxidase.
 - Catalase.

Antibiotic sensitivity testing

A single colony is taken from the original culture plate, added to a bottle of liquid culture medium (broth) and incubated for 4–6 hours, to increase bacterial numbers. It is then poured over the surface of a second agar plate. Bacteria should grow evenly over the surface of this second plate.

Small paper discs, each one containing a different antibiotic, are then placed onto the surface and the plate is incubated for a further 24 hours. If the bacteria grow up to the very edge of the disc (as with the antibiotic on the left of Plate 4.11), then this antibiotic will not be effective in treating the bacteria. On the right, the antibiotic has diffused out from the disc onto the agar

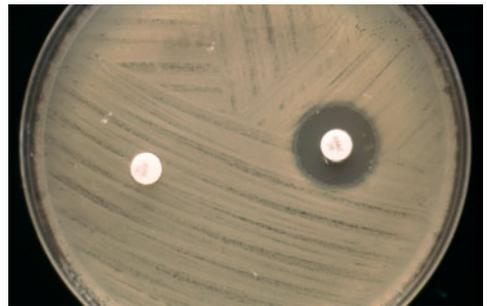


Plate 4.11. Antibiotic sensitivity plate. Only the antibiotic that causes a zone of inhibition around the disc (right) would be effective in the treatment of this infection.

surrounding the disc. Hence the clear zone of growth inhibition around the outside of the disc. The size of the zone of inhibition does not represent the likely efficacy of the anti-biotic used in the cow, but rather the concentration of the antibiotic in the disc and the ease with which it can diffuse through the agar. Other factors relating to efficacy of antibiotics are discussed in Chapter 12.

Interpretation of results

Even when bacteria have been grown and identified, often a mixture of organisms is obtained and there may still be difficulties in determining what is significant. The following are general guidelines:

1. A pure culture of any mastitis pathogen – highly probable cause of the mastitis.
2. A mixed culture, for example:
 - *Staphylococcus aureus* and *Streptococcus agalactiae*, plus other organisms such as *Streptococcus faecalis*; or
 - *E. coli* and *Streptococcus uberis*, plus other organisms.

In the above examples of mixed cultures, *Staphylococcus aureus* and *Streptococcus agalactiae*, or *E. coli* and *Streptococcus uberis* are the most probable cause of mastitis, and the other organisms are contaminants. In any sample containing a significant level of *Staphylococcus aureus* or *Streptococcus agalactiae* colonies, these organisms should be considered significant.
3. A contaminated culture, for example, *E. coli*, *Bacillus* species, *Proteus* and *S. faecalis*. These are all environmental organisms. There are so many bacteria present that the sample is obviously heavily contaminated and no useful information can be gained.
4. No growth. In any laboratory, one might expect as many as 25–35% of samples giving no growth, or no significant growth. This is often frustrating to the herdsman, who may have taken the sample very carefully from a cow obviously affected by clinical mastitis, e.g. with a

hard and swollen quarter, or recurrent milk clots. Possible causes of no growth include the following:

- *E. coli* infection – host response is so effective that all the bacteria have been destroyed by the time udder changes become visible (see page 29). This has been shown to be by far the most common cause of ‘no growth’.
- Intermittent excreter, e.g. with chronic staphylococcal or *Streptococcus uberis* infection the numbers of bacteria present at different times can fluctuate greatly, i.e. when the sample was taken the organism may only have been present in very low or undetectable numbers (see Table 4.5).
- Residual antibiotic from previous treatment is still present, inhibiting bacterial growth in the lab.
- Excessive delay between taking the sample and plating it out.
- Loop too hot.
- Sample volume too small (use a swab).
- Unusual organisms, not detectable following standard techniques, e.g. *Mycoplasma* or *Leptospira*.
- Traumatic or hypersensitivity mastitis, i.e. where no infectious cause is involved. Probably rare.

Total Bacterial Count, Laboratory Pasteurised Count and Coliforms

It is unlikely that farmers reading this text will want to carry out their own bacteriology. However, for those involved in laboratory and mastitis investigational work, the ability to perform total bacterial, laboratory pasteurized and coliform counts is invaluable. Details of interpretation of results are given in Chapter 10 (on TBC/Bactoscan).

Total bacterial count (TBC)

This is the total number of living bacteria per ml of milk, sometimes also referred to as the TVC, or total viable count. Some British dairy companies now require milk with a Bactoscan under 30,000 bacteria per ml: otherwise they impose penalties. The TBC

of milk consists of thermodurics, coliforms and many other organisms. Causes of high TBCs in milk and the organisms involved are discussed in detail in Chapter 10.

Laboratory pasteurized count (LPC)

Also known as the thermoduric (TD) count, this is a measure of the number of living bacteria present after heating the milk sample. High thermoduric counts are indicative of poor plant washing.

Coliform counts (CC)

The number of coliforms per ml of milk. High coliform counts are usually associated with dirty (faecally contaminated) teats. Values may be increased when there is poor housing and poor premilking teat preparation.

Pseudomonad count

Pseudomonad species are environmental coliforms, sometimes referred to as non lactose fermenting coliforms, or NLFs. They can be used as an indicator of environmental as opposed to faecal contamination.

Streptococcus uberis count

Raised levels of *S. uberis* can be associated either with environmental contamination or, and more likely, mastitic milk entering the bulk tank. The latter may be due to subclinical infections or to poor detection of clinical cases.

Total Staphylococcal and Staphylococcus aureus counts

High levels of staphylococci may be due to chronic infection, poor postmilking teat disinfection or poor teat skin quality.

Differential count

This is a semi-quantitative assessment of the proportion of different types of mastitis bacteria present in the sample. It can help in the investigation of both mastitis and TBC

problems. For example, if *Streptococcus agalactiae* is present in bulk milk it could be the cause of both a high TBC and a high somatic cell count.

However, a word of warning: the absence of *S. agalactiae* (or any other mastitis organism) from the bulk milk does not mean that it is not present in the herd. It simply means that it has not been cultured on this occasion.

Methodology

It is vital that samples remain refrigerated during transport to the laboratory, otherwise TBCs and other counts will increase dramatically. In order to obtain the results carry out the following procedures:

Total bacterial count

Dilute the milk sample 1:1000 by adding 0.01 ml of milk to 10 ml Ringer's solution, then pipette 1.0 ml into a Petri dish. Pour on 20 ml milk agar, cooled to 45°C. Allow to solidify and then incubate at 37°C for 48 hours. Count the colonies. The TBC is the number of colonies on the plate \times 1000.

Thermoduric count

Heat 10.0 ml milk for 35 minutes at 64°C \pm 0.5°C. Cool and dilute 1:10 with Ringer's solution. Pipette 1.0 ml of diluted milk into a Petri dish and add 20 ml milk agar cooled to 45°C. Incubate at 37°C for 48 hours and count as above. The thermoduric count is the total number of colonies \times 10. Values over 200 per ml suggest a wash-up problem.

Coliform count

Pipette 1.0 ml of undiluted milk into two Petri dishes. Pour 20 ml of violet red bile agar (cooled to 45°C) into each. Incubate at 37°C and count the colonies at 24 and 48 hours. Ideally coliform counts should be less than 10 colonies per ml of milk, although values of up to 20 colonies/ml are acceptable.

5 Milking Machines and Mastitis

History of the Milking Machine	61
Function of the Milking Machine	61
Vacuum pump	62
Interceptor vessel	63
Balance tank	63
Regulator	64
Sanitary trap	65
Vacuum gauge	65
Pipelines	65
Cluster	66
Receiver vessel	67
Recorder jar	68
Automatic cluster removers	68
Pulsation	68
Pulsation chamber	68
Pulsation rate and ratio	70
Pulsators	70
Liner	71
Liner shields	73
Robotic Milking	74
Maintenance and Machine Testing	74
Daily checks by milkers	74
Weekly checks by the manager or owner	75
Routine specialist testing	75
Static test	76
Dynamic test	76
The Milking Machine and its Relationship to Mastitis	78
Acting as a vector	78
Damage to the teat end	78
Colonization of the teat canal	79
Liner slip and impact forces	79
Undermilking	81
Overmilking	81
Stray voltage	81
Simple Machine Checks that can be Carried Out Without Specialist Equipment	82
Vacuum level	82
Vacuum reserve	82
Regulator function	82
Pulsation system	83
Liners and rubberware	83
Other checks	83
Observations to be carried out during milking	84
Wash-up routines	84
Butterfat	85

Protein	85
Minerals	85
Bulk tanks	86
Air lines	86
Circulation Cleaning	86
Rinse cycle	86
Wash cycle	86
Disinfection cycle	88
Summary of common problems associated with circulation cleaning	88
Acid Boiling Wash (ABW)	90
Manual Washing	91
If a Wash-up Problem is Suspected	91
Common Faults Found with Milking Machines	91

This chapter explains the function of the milking machine and how it can affect mastitis. It also describes simple machine checks that can be carried out without any specialist testing equipment to assess performance. Milking machine testing and maintenance are explained, along with common faults that are found. How to wash the plant after milking and common wash-up problems are discussed.

The milking machine is the dairy farmer's equivalent of a combine harvester. It is a unique piece of equipment as it is the only machine that harvests food from a living animal on a regular basis. Milking machines are used more than any other piece of equipment on the farm. Even so, they are frequently neglected, despite the fact that they are responsible for generating the majority of the dairy farmer's income. Milking machines can have an influence both on mastitis and on milk quality, particularly Bactoscan or TBCs.

History of the Milking Machine

In the early 1800s, a number of pioneers tried to devise a machine capable of milking cows. These people were not farmers, who had little interest in mechanization while labour was cheap, but plumbers, doctors, inventors and engineers. The first machine was patented in 1836 and consisted of four metal cannulae connected to a milk pail suspended under the cow. Needless to say, this design must have caused considerable dam-

age to the teat ends and also spread of infection from cow to cow. In 1851, two British inventors had the idea of using vacuum to milk cows. In 1895, the Thistle milking machine was developed. It used a steam engine to drive a massive vacuum pump and was the first machine to include a device (the pulsator) to relieve constant pressure on the teat.

In the early 1940s, only 30% of dairy farmers in England and 10% in the USA were milking by machine. Real development only started in the mid-1940s, when the post-war shortage of labour stimulated a concerted drive to develop a commercial milking machine that was cost-efficient. The development of the milking machine continues even now, with the introduction of more sophisticated electronics to further improve performance.

Function of the Milking Machine

The basic principles of machine milking are identical in all milking systems, from the sophisticated robotic milker or rotary parlour down to the milking bale: milk should be removed swiftly from the udder with minimal risk to udder health. Milkout occurs by applying reduced pressure, i.e. vacuum, to the teat end, which causes the teat canal to open, letting the milk flow out. This is assisted by the oxytocin-induced let-down reflex, which increases the pressure within the udder. A constant vacuum level should be maintained

throughout milking. The pulsation system is responsible for ensuring adequate blood circulation around the teat. In order to understand how the milking machine operates, it is important to know how and where the various components fit into the system. When trying to identify any component, it is advisable to work your way from the vacuum pump forwards so as to avoid confusion. The components of the milking system are described below, and this section should be read in conjunction with Fig. 5.1.

Vacuum pump

This is the heart of the milking plant. It creates vacuum by extracting air from the milking system. Air is removed from all the pipes, jars, claws and liners. Vacuum levels are measured in kilopascals (kPa) or inches of mercury ("Hg). One kPa is equivalent to 0.3 "Hg or 1.0 "Hg is equivalent to 3.33 kPa.

Vacuum pumps are rated according to the amount of air that they can extract at a set vacuum level, normally 50 kPa (15 "Hg). This measurement is expressed in litres of displaced air per minute (l/min), and in the USA is expressed in cubic feet per minute (c.f.m.). The vacuum pump must always be fitted with a belt guard to protect against



Plate 5.1. The vacuum pump shown has no belt guard to protect against injury and leaves staff at risk of becoming entangled in the belts (A).

injury. Plate 5.1 shows a vacuum pump with no belt guard.

The vacuum pump needs to extract more air than is necessary to operate the milking system. This overproduction can be measured and is called the **vacuum reserve**. Vacuum reserve is needed to allow for air admission, such as when units are put on or taken off, to ensure the machine

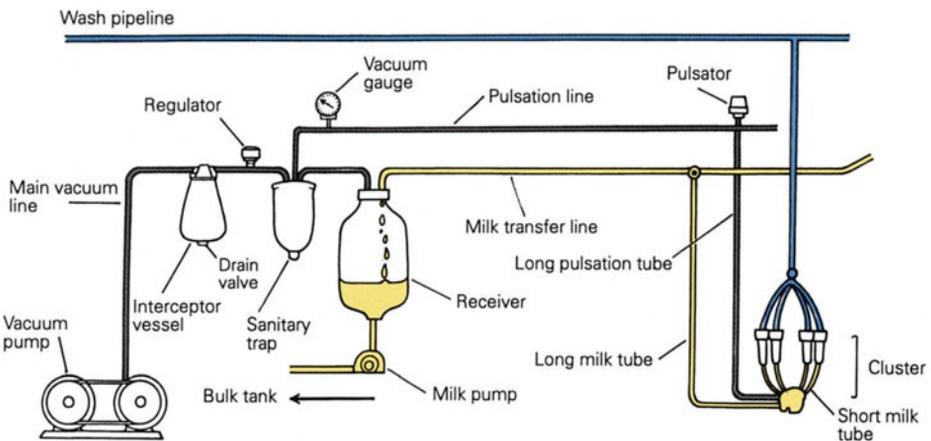


Fig. 5.1. A simple milking system. There is a huge variety of layouts in milking systems. This depends on factors such as age of the system, whether it has been modified, the amount of space available, manufacturer of the machine, etc.

Table 5.1. Air admission during milking (in litres of air/min), showing the importance of having adequate vacuum reserve if constant vacuum levels are to be maintained throughout milking.

Item	Air admission (l/min)
ACRs (per unit)	5–25
Air bleed per unit	4–12
Feeders (per feeder)	5–30
Gates (per gate)	10–42
Liner slip	28–170
Unit attachment	3–225
Unit fall-off (per fall)	570–1400

maintains a stable vacuum level during milking. Table 5.1 shows some examples of air admission that occur during milking.

The amount of vacuum reserve required for a milking plant will depend on the number of milking units and any other equipment that uses vacuum, such as ACRs (automatic cluster removers), pneumatic gates, feeders and teat dip sprayers. Allowance should also be made if more than one milker operates the plant. There must be sufficient vacuum reserve present to maintain vacuum stability throughout the whole of milking. For smaller milking systems, a large vacuum reserve will be required to ensure there is an adequate wash cycle.

Interceptor vessel

This is located close to the vacuum pump. Its function is to prevent any liquids or foreign matter from entering and damaging the pump. There is a drain valve at the base of the interceptor vessel, as shown in Plate 5.2, so that when the machine is turned off any liquids drain away under gravity.

Balance tank

This is found in some installations and is located between the interceptor vessel and the sanitary trap. It is a large hollow vessel up to 200 litres in capacity that acts as a vacuum reservoir (Plate 5.3). It is designed to



Plate 5.2. The interceptor vessel with drain valve at the bottom.



Plate 5.3. The regulator should always be fitted to the balance tank if one is present in the system.

improve vacuum stability during milking. Each balance tank has a drain valve at the base. In most installations, the vacuum lines for milking and pulsation feed directly off the balance tank.

Regulator

The vacuum pump extracts a **fixed** amount of air from the milking system. However, the demand for vacuum is **variable**, depending on how much air enters the system during milking, and so there must be some form of regulation to maintain stability. The vacuum regulator, sometimes called the vacuum controller, is responsible for maintaining vacuum stability. The regulator leaks air into the system as and when necessary so that a constant preset vacuum level is maintained at all times. The regulator should be sited in a dust-free location where it is easy to clean and inspect when necessary, as shown in Plate 5.4. Many regulators are now fitted with a **clean air system**, as shown in Plate 5.5, that draws air from the external environment. This avoids dust contaminating the regulator and entering the milking system.

The regulator filter should be cleaned regularly because if it becomes blocked it may be unable to respond rapidly to vacuum changes in the system. This will result in poor vacuum stability and increase the risk of fluctuations and thereby new infections.



Plate 5.4. The regulator should always be sited in a clean dust-free environment.



Plate 5.5. Regulators fitted with a clean air system are surrounded by a case (A). A pipe (B) feeds air from the external environment through the regulator and into the milking system. Although the surrounding area is dirty the system should prevent this dirt from entering the pipelines.

The regulator should be located on the balance tank, if one is present in the system. The balance tank acts as a reservoir for vacuum so there should be more stability and less turbulence at this point than on the pipes that feed in and out of the tank.

Regulators are rated according to the amount of air that they can admit into the system. Regulator capacity is measured in litres of air per minute (c.f.m. – cubic feet/minute – in the USA) and should equal the capacity of the vacuum pump. Any defect in regulator function may result in vacuum fluctuations during milking. The regulator should **always** be leaking air into the system. If this does not occur, it indicates that the plant is unable to maintain a stable vacuum level (i.e. inadequate vacuum reserve) or the regulator is faulty.

There are three types of regulator: weight-, spring- and servo-operated. Weight- and spring-operated regulators measure the vacuum level at the place where the air is admitted. For this reason, they do not respond very quickly to pressure changes. Servo regulators have a vacuum sensor fitted away from the air inlet valve and can correct vacuum fluctuations within milliseconds. Servo regulators are highly efficient and are now installed in all new parlours.

In some systems, it is the speed of the motor that partly controls vacuum, i.e. as

more vacuum is needed, the vacuum pump speeds up. If this energy-saving device has been installed, the regulator does not always leak in air.

Sanitary trap

This is located at the junction of the milk and air systems and is shown in Plate 5.6. Its function is to prevent any milk or liquids from entering those pipelines that carry only air, such as the line to the vacuum pump or to the pulsation system.

The sanitary trap should be made of a transparent material such as glass or pyrex and be located where the milker can keep an eye on it during milking. The trap is fitted with a floating ball valve so that if fluids build up, the valve rises and shuts off the vacuum supply. This causes all the milking units to fall off the cows, and in so doing protects the vacuum pump and air lines from becoming contaminated with milk.

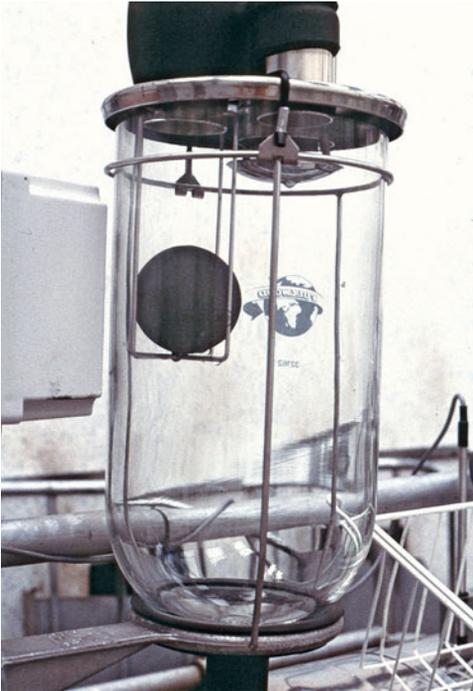


Plate 5.6. Sanitary trap with a ball shut-off valve to stop liquids entering the air lines.



Plate 5.7. The vacuum gauge must be easy to read and visible throughout milking.

Vacuum gauge

This should be located in the parlour so that it is visible to the milker throughout milking. Clearly gauges should be large enough to be read anywhere in the parlour (see Plate 5.7). Always check where zero (atmospheric pressure) is on the gauge. Older gauges move clockwise, while most new gauges move anticlockwise.

Pipelines

During milking, pipelines carry vacuum, milk or a mixture of the two. There should be as few bends as possible and no constrictions, as these will interfere with air or liquid movement. The free passage of air and liquids helps maintain vacuum stability. Dead ends must be avoided as they are difficult to clean and can lead to Bactoscan or TBC problems (see Chapter 10). Pipelines that carry milk must be made of material such as stainless steel or glass that can be cleaned and disinfected.

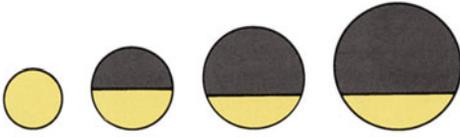


Fig. 5.2. The same volume of milk shown in pipes of different sizes.

Large-bore pipes are now fitted to most new milking installations. The effect of pipe size on internal volume is shown in Fig. 5.2. Four pipe sizes filled with an equal volume of milk are compared. The extra space in the larger bore pipe allows better movement of milk and air compared with that in the narrow pipe, which is flooded with milk. Large-bore pipes are more difficult to clean, however, and need more hot water plus air injectors (air blasters) to produce a physical swirling action that will clean the entire internal surface of the pipe (see Chapter 10).

Cluster

This consists of a clawpiece and four teat cups, each with its shell and liner, plus a short milk and a short pulsation tube. In many systems, the short milk tube is an integral part of the liner. The liners are connected to the long milk tube through the clawpiece (Fig. 5.3). The short pulsation tubes, one from each liner shell, are connected to the long pulsation tube on the outside of the claw. Milk is removed from the udder into the liner and then through the short milk tube into the clawpiece and out through the long milk tube.

An air bleed hole is fitted to each clawpiece. This leaks atmospheric air during milking to assist milk flow away from the udder. Air bleed holes admit between 4 and 12 litres of air per minute. In some systems, the air bleed hole is sited in the mouth of the liner (see Plate 5.12). This is said to give 'cleaner' milking, with less soiling of the teat.

The long milk tube is connected to either a recorder jar or the milk transfer line. It is important that milk is removed swiftly

from the udder. This will stop flooding in the clawpiece and the short milk tubes. Flooding means that milk from one quarter could pass to any of the other three teats, thus allowing cross-contamination between quarters. Flooding also leads to vacuum fluctuation. For this reason, the short milk tubes, the clawpiece and the long milk tube should be of sufficient size to make rapid milk removal possible.

Many years ago, clawpiece capacity was as low as 50 ml. However, as milk flow rates have increased, a capacity of up to 500 ml is commonplace today. The diameter of the short air tubes and short milk tubes has also increased. The difference in volume between a 13 mm and 16 mm long milk tube is 50%, and this can have a marked effect on vacuum stability at the teat end.

Milk flows through the short milk tubes, clawpiece and long milk tube into the recorder jar (if fitted), down the milk transfer line (this may be called the long milk line) and into the receiver vessel. The milk transfer line should be gently sloped to assist the passage of milk to the receiver vessel. Excessive agitation of the milk results in degradation of the fat.

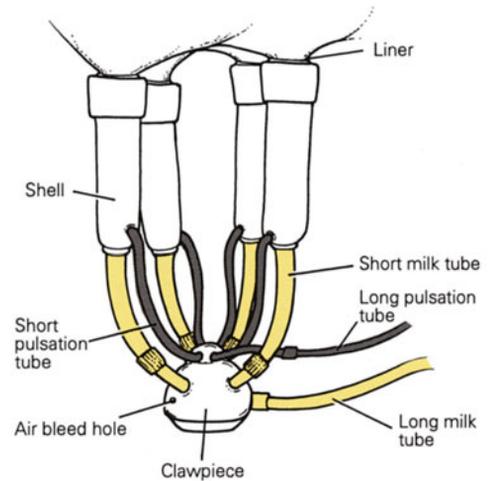


Fig. 5.3. The cluster consists of a clawpiece and four teat cups, each with its own shell, liner and short milk and short pulsation tubes.



Plate 5.8. A stainless steel receiver vessel is resistant to breakage but difficult to inspect.

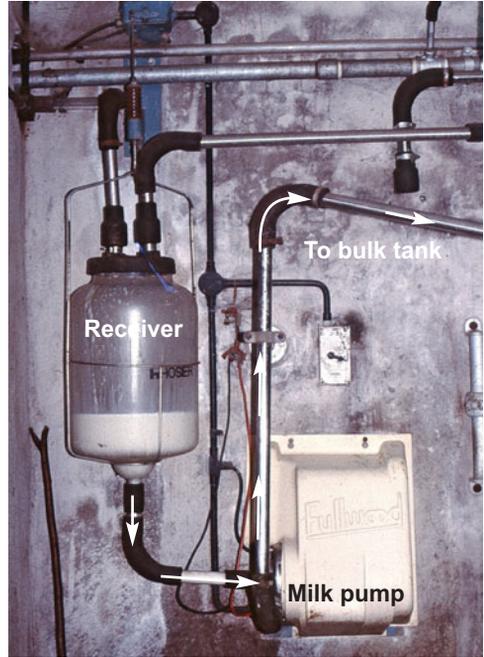


Plate 5.9. The milk pump pushes milk from the receiver vessel under atmospheric pressure into the bulk tank.

Receiver vessel

The receiver (see Plate 5.8) receives milk from one or more milk transfer lines. It may be made from either glass or stainless steel.

When milk builds up in the receiver, it triggers sensors to start the milk pump, which is connected to the base of the receiver. Milk is then pumped away from the receiver vessel by the milk pump through a milk filter, through plate coolers (if fitted) and into the bulk tank, as shown in Plate 5.9. The milk pump is therefore the 'break' between the vacuum system and the outside atmosphere.

Milking systems can be divided into two types depending on the level of the milk transfer line in relation to the cow: if the milk transfer line is below the level of the udder, then the system is called a low line, and if milk is lifted above the udder it is a high line (see Plates 5.10a and b). High line systems need to operate at a higher vacuum level, as they have to physically 'lift' milk from the udder into the milk transfer line.



Plate 5.10a. Low line system, where the milk transfer line is below the level of the udder.



Plate 5.10b. High line system, where the milk has to be 'lifted' above the udder.

Milk should not be lifted more than 2 m above the level of a standing cow.

Recorder jar

This is a vessel that holds and stores milk from an individual cow in the parlour. It allows the milker to see how much milk each cow has given. Milk travels from the recorder jar to the receiver vessel along the milk transfer line. In the majority of modern systems there are no recorder jars and milk is released from the long milk tube directly into the milk transfer line, and then on into the receiver vessel. This type of milking is called a '**direct to line**' system.

A milk flow meter may be fitted where the long milk tube enters the milk transfer line and give a digital reading of milk yield. This flow meter will also trigger the ACR.

Automatic cluster removers

More commonly known as ACRs, these are shown in Plate 5.11. ACRs are a labour-saving device intended to increase the efficiency of milking. ACRs also benefit the cow by reducing overmilking, which can result in teat-end damage. They remove the cluster automatically once the cow is milked out. Milk flow is measured by a sensor fitted in the long milk tube or flow meter. When milk flow falls below a certain preset level, the vacuum supply to the cluster is shut off. There is then a delay as air enters into the



Plate 5.11. ACRs shut off the vacuum to the cluster at the end of milking. To prevent the unit falling on the floor and becoming contaminated, a cord is activated, which gently lifts the cluster off the cow.

claw through the air bleed hole, making the vacuum level drop, and finally a cord removes the unit from the udder.

Most modern ACRs are adjustable and set to remove the milking unit once the flow rate falls to between 400 and 500 ml/min for twice a day milking and between 600 and 800 ml/min for three times a day milking.

Pulsation

Pulsation is responsible for maintaining blood circulation around the teat. This is achieved by making the liner open (milkout phase) and closed (massage phase) approximately 60 times per minute. The liner should be able to open fully and collapse completely below the teat with full and free movement. This is achieved by alternating atmospheric air and vacuum in the pulsation chamber, as shown in Fig. 5.4.

Pulsation chamber

This is the space between the liner and the teat cup shell. Air is extracted from the pulsation chamber through the short and long pulsation tubes. These join on the top of the clawpiece, as shown in Fig. 5.3. Note that there is no connection between the milk and pulsation lines on the clawpiece.

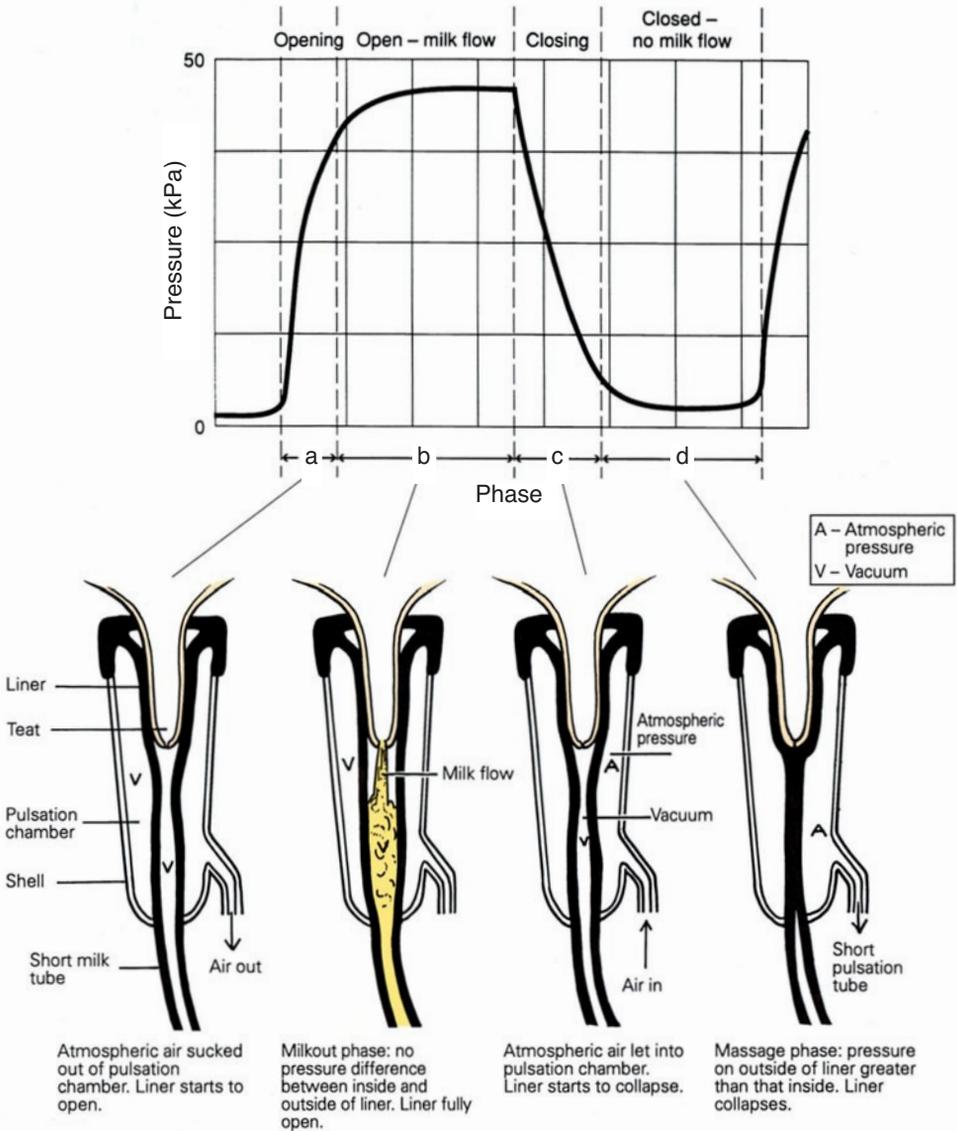


Fig. 5.4. Liner action on the teat. Pulsation is achieved by alternating atmospheric air and vacuum in the pulsation chamber. Each pulsation cycle can be traced on to a graph (above) to show the pressure changes that occur inside the pulsation chamber.

During milking, vacuum is constantly applied to the base of the teat. When atmospheric air enters the pulsation chamber, it forces the liner to collapse around the teat. This happens because the pressure on the outside of the liner is greater than that inside. When this occurs, milk flow stops

and blood is able to circulate around the teat. This is called the 'massage' phase.

When the atmospheric air is 'sucked' out of the pulsation chamber and replaced with vacuum, the liner is 'pulled' open. This occurs as there is no pressure difference between the pulsation chamber and the

inside of the liner and so the liner opens under its own elasticity. The elasticity of the liner is therefore very important. When the liner opens, milk flows away from the udder; this is called the '**milkout**' phase.

One complete liner movement is called a **pulsation cycle**. Each pulsation cycle can be divided into four phases – a, b, c and d, as shown in Table 5.2. Each pulsation cycle can be traced on to a graph to show the pressure changes that occur inside the pulsation chamber, as shown in Fig. 5.4. It must be remembered that this is not pulsation. It is just a graphical representation of the pressure changes that are occurring within the pulsation chamber during a pulsation cycle. Pulsation refers to the actual liner movement around the teat.

Table 5.2. The pulsation cycle. (From Spencer, 1990.)

Phase	Liner action on the teat
a	Opening
b (or milkout)	Open with full milk flow
c	Closing
d (or massage)	Closed: milk flow stops, blood circulates

Pulsation rate and ratio

The **pulsation rate** is the number of pulsation cycles per minute, and the rate is normally between 55 and 60 cycles per minute. The **pulsation ratio** refers to the length of milkout (a + b) compared with massage (c + d) and is expressed as a percentage. A pulsation ratio of 60/40 refers to 60% of the cycle as milkout and 40% as massage.

$$\text{Pulsation ratio} = \frac{a + b}{c + d} \times 100\%$$

To make matters even more confusing, there are two different forms of pulsation: single and dual.

Single pulsation

Single or simultaneous pulsation occurs when the movement of all four liners in a

cluster act in unison. Single pulsation may be referred to as 4 × 0 pulsation. All four quarters are milked out at the same time and then massaged at the same time. This results in 'slugs' of milk flowing away from the udder and possibly greater teat-end vacuum fluctuation, but results in a lower teat-end vacuum level during peak milk flow.

Dual pulsation

Dual or alternate pulsation is where the movement of two liners alternates with that of the other two. Dual pulsation may be referred to as 2 × 2 pulsation. So, while two quarters are being milked, the opposite two are being massaged. This results in a continuous flow of milk away from the udder. Dual pulsation results in more efficient milking than single pulsation as there are no surges of milk or airflow in the system during milkout. In plants with single pulsation, there is usually only one long pulsation tube, whereas with dual pulsation, there must always be two.

Pulsators

A pulsator is a device that provides alternate pulses of vacuum and atmospheric air in the pulsation chamber. There are two different types of pulsators: individual and master.

Master pulsation

This is controlled electronically and regulates the pulsation throughout the parlour. Each cluster often has its own 'slave' pulsator controlled by the master. They are designed to give uniform pulsation, no matter where a cow is milked in the parlour. If problems occur with master pulsation, then all milking units will be equally affected. All modern milking systems are fitted with electronic master pulsation.

Individual pulsation

Individual pulsation refers to a system where each milking unit has its own individual pulsator. All the pulsators operate

independently of each other and so cows may receive different pulsation rates and ratios depending on where they are milked in the parlour. If a problem occurs with an individual pulsator, then only that milking unit will be affected. Individual pulsators can be expensive and many manufacturers are now ceasing production of these because of the benefits of master pulsation.

Liner

The liner is the only piece of the milking machine that comes into direct contact with the cow. Liners are made from complex rubber or silicone material and have a mouthpiece and a barrel, and may have an integrated or separate short milk tube, as shown in Fig. 5.5. The degree of elasticity in a liner has a big effect on its efficiency and hence liners have a limited useful life.

The majority of European rubber liners are expected to last for 2500 milkings or 6 months, whichever comes first. Silicone liners have a much longer life of up to 10,000 milkings, but are more expensive.

Fig. 5.5. Liners are made from complex rubber or silicone material and have a mouthpiece and a barrel, and may have an integrated or separate short milk tube.

Fig. 5.6. Liners deteriorate with age, becoming collapsed and losing their elasticity, as shown in the liner on the right.

Liners should meet the following requirements:

- Have a soft, flexible mouthpiece that forms an airtight seal with the base of the teat, adjacent to the udder. This will minimize liner slip and unit fall-off.
- Have a barrel long enough to allow the liner to collapse fully around the base of the teat.
- Be easy to clean.
- Provide a rapid milkout with minimal teat injury.

Liners with a triangular barrel are used in some parlours. They are said to be beneficial because they produce a more even compression on the teat by applying pressure on three rather than two planes. One type of triangular liner has an air bleed at the mouthpiece to improve milk flow.

Liners eventually lose their elasticity and become collapsed as shown in Fig. 5.6. This occurs as liners always open and close in the same plane and explains why ‘wedging’ is sometimes seen at the teat end on some cows after unit removal (see pages 235–236).

There is one new type of liner that has an air bleed at the top of the liner (Plate 5.12). It is claimed that the benefits of this feature are a better milking performance and better ACR take-off as the clawpieces vent more quickly.



Plate 5.12. An air bleed at the top of the liner is claimed to give better milkout and smoother ACR function.

When liners become more worn, it takes longer for them to open and they will close early due to their tendency to collapse. Also, the overall effect is that milking time will be increased. Many milkers have noticed that milking time is reduced as soon as a set of collapsed liners is replaced.

Chemicals, especially chlorine products, will denature rubber and so reduce liner life. A rough inner surface of the liner may abrade teat skin and will certainly harbour bacteria. This will increase the risk of mastitis transmission and may affect Bactoscan or TBCs.

Liner usage can easily be checked by using the formula below. The number of cows refers to the total number of cows in the herd, i.e. milking and dry.

$$\text{Liner usage (no. of milkings)} = \frac{\text{No. of cows} \times 2^* \times \text{liner life in days}}{\text{No. of milking units}}$$

*Change to 3 if milking three times a day.

For example, in a herd of 160 cows milked twice a day through an 8 × 16 parlour (8 × 16

refers to 8 milking units with 16 cow stand-ings), and where liners are replaced twice a year, liners will have milked 7320 cows before being replaced:

$$\begin{aligned} \text{Liner usage} &= \frac{160 \text{ cows} \times 2 \times 183 \text{ days}}{8 \text{ milking units}} \\ &= 7320 \text{ milkings per liner} \end{aligned}$$

In this case the rubber liners have exceeded their useful life of 2500 milkings (check the manufacturer's recommendations for useful life of the type of liner you are using). They may need to be changed more frequently. The frequency of change can be worked out using the following formula:

$$\begin{aligned} \text{Liner life in days (i.e. frequency of change required)} &= \\ &= \frac{2500^* \times \text{no. of milking units}}{\text{No. of cows} \times 2^{**}} \end{aligned}$$

*Change if manufacturer's recommendations are different.

** Change to 3 if milking three times a day.

So, for this herd:

$$\begin{aligned} \text{Liner life in days} &= \\ &= \frac{2,500 \times 8}{160 \times 2} = 62.5 \text{ days or 2 months} \end{aligned}$$

The frequency of liner change can be checked quickly using the liner life charts in the Appendix at the end of the book. All the information required for this is the herd size, the number of milking units and the frequency of milking.

Liner choice is very important. The correct liner should be chosen to fit the teat cup shell. It must be able to collapse fully around the base of the teat; otherwise blood will be unable to circulate and this may lead to teat-end oedema (see pages 235–236) and other teat-end damage. Once liners have become worn and rough, not only can they cause damage to the teat end but they also become more difficult to clean (see Plate 5.13). Despite its appearance, the inside of such a liner is not smooth, as shown in Plate 5.14, which is an electron micrograph of a worn liner, and is magnified many thousand of times.

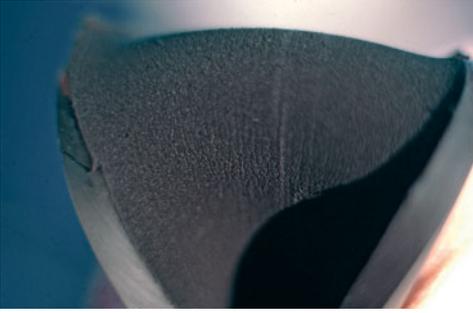


Plate 5.13. Worn, rough liners are difficult to clean and may harbour mastitis bacteria.

Liner shields

These may be fitted to the base of the liner barrel as shown in Fig. 5.7. Their function is to reduce the effect of any impact forces (see pages 79–80). Experimental work has shown that liner shields have helped to reduce the new infection rate by up to 12%. However, the siting of shields is critical. If they are situated too high up the liner they may prevent total liner collapse. There will then be an incomplete massage phase and teat-end damage may result.

Robotic Milking

Robotic milking has become more popular, and in 2009 there were about 8000 robots on more than 2400 farms throughout the world. The first robot or automatic milking system

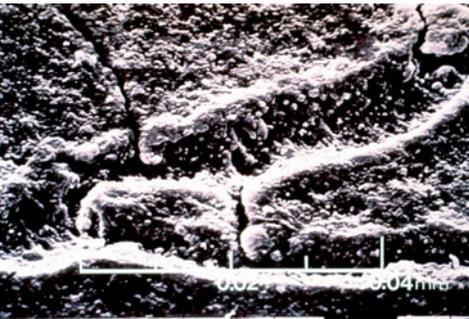


Plate 5.14. This is an electron micrograph of a worn liner. See how easy it is for bacteria to stick and be spread from cow to cow.

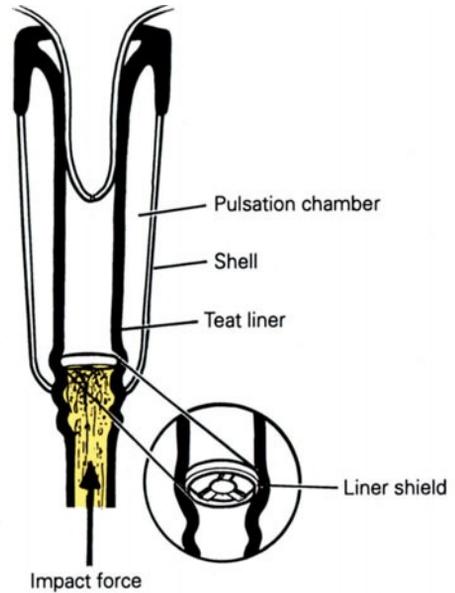


Fig. 5.7. Liner shields help to reduce the effect of impact forces.

(AMS) was installed in the Netherlands in 1992. The vast majority of robots are found in north-west Europe, with the Netherlands having the largest installed base and Scandinavia showing the fastest growth rate in the past few years. The principles of machine milking with a robot are identical to those of a conventional milking machine, with the exception that the robot has to attach the machine automatically (Plate 5.15).



Plate 5.15. A robotic milking machine attaching a teat cup.

People install robots for a variety of reasons, some of which are for personal lifestyle factors to improve the quality of their life, to increase milk yield, while others hope that more frequent milking and removal of teat cups on an individual teat basis will improve the health of their dairy herd. Some prefer not to have the task of routine milking, but the majority of users will tell you that the amount of time that is saved from using robots is not as great as many people think.

It is very important that the dairy farmers build their dairy system around the robot and not vice versa. Some farmers feel that they will be able to install a robot into an existing facility and that it will work efficiently. However, the majority of people find that this does not work.

It is essential that robots are well maintained and reliable, as they are milking cows 24-7. Engineering support has to be available 24-7 in the event of any breakdown or malfunction. There are a number of advantages with this technology (including allowing the cow to choose when she wants to be milked) that will have a positive impact on yield. Most robots will remove the teat cup from individual quarters when they are milked out, thus avoiding overmilking and maximizing teat condition. Electrical conductivity meters can help with early detection of clinical mastitis, and the liners are disinfected between cows, thereby avoiding cross-contamination.

Maintenance and Machine Testing

Like any other piece of equipment, the milking machine needs to be maintained correctly so that it operates at maximum efficiency. An inefficient machine may at best slow down milking or at worst decrease yield and increase the amount of mastitis.

It is important that all the people who use the plant know what checks need to be carried out. A machine that is not operating to its full potential will still remove milk from the udder although it may well cause a predisposition to mastitis. Problems with milking machines tend to be gradual in

onset and so checks need to be made regularly.

Some checks should be carried out daily by milkers, others at regular intervals by the manager or owner and others using specialist testing equipment on a routine basis. Most manufacturers now have a list of checks, together with a timetable of when these should take place.

The milking machine is a highly complex piece of equipment. It is used twice or sometimes three times every day of every year. Compare it to a motor car: car manufacturers recommend servicing every 5000 or 10,000 miles. Virtually everyone gets their car serviced at the correct intervals: if not, the performance of the car starts to decrease and breakdown is more likely.

The milking machine is no different. The average milking machine runs for 2.5 hours every milking. This is equivalent to over 1800 hours of use each year. If you compare the milking machine to a car travelling at 40 miles per hour (64 k.p.h.) over a year, the plant would have done the same amount of work as a car travelling 72,000 miles (115,200 kilometres).

Daily checks by milkers

The milkers are the first people who may feel that the machine is not operating correctly. Milking time may be increased or units may fall off for no apparent reason. Perished rubberware or parts such as worn valves, etc., should be replaced as and when they are identified. The milker should also check the vacuum level on the gauge during milking. If the milker is unable to identify and correct faults, then the farm manager or machine dealer should be called in to sort out the problem.

Weekly checks by the manager or owner

It would be unfair to leave all the responsibility for the milking machine to the milker. The manager or owner should check the plant regularly for any possible faults. Rubberware should be checked, liner condi-

tion inspected, the air filter on the regulator looked at and the oil level and belt tension of the vacuum pump checked.

Routine specialist testing

It is important that milking machines are tested by a qualified technician or adviser on a regular basis. Most farmers have their plant tested once a year, and some twice a year. There are some dairy farmers who have never had their plants tested since the day it was installed – sometimes a gap of up to 20 years. Parlours should be tested to meet specific standards such as those of the ISO (International Organization for Standardization), or national standards. These would be the minimum requirement.

All milking machines should be tested every 6 months. Plants that milk three times a day should be tested more frequently. In Arizona, where three times a day milking is the norm, all milking plants are checked every month to ensure that they are operating at maximum efficiency. They believe that the best test report is one where no problems are identified, meaning that the machine is working at its optimum performance. There are two types of test that can be carried out: a static and a dynamic test.

The static test is carried out between milkings when no cows are being milked. This is equivalent to an MOT or mechanical inspection of a car. It is very useful in identifying certain problems, but it does have limitations. The dynamic test is carried out during milking, and this is equivalent to a road test of a car. During the dynamic test the machine is tested under 'load' to see what, if any, problems are present. All parlours should have an annual dynamic test.

Static test

The static test includes the following.

Vacuum levels in the plant

Vacuum levels are checked at various locations throughout the plant to ensure that

there is no significant loss of vacuum between the pump and the teat end, and that the plant is set at the correct level. A drop in vacuum level would indicate that air is leaking into the system. The accuracy of the vacuum gauge is also checked.

Vacuum reserve

Vacuum reserve has already been described as being the production of vacuum over and above that needed to operate the plant. Adequate vacuum reserve is needed to ensure that stability of pressure is maintained in the plant throughout milking.

The ISO has made recommendations for vacuum reserve. It must be remembered that these are minimum recommendations, and ideally new plants should exceed these levels significantly. This will ensure that plants will still be able to operate to ISO standards as they get older and when their performance starts to become less efficient.

Systems with a low vacuum reserve will have difficulty in maintaining stable vacuum levels during milking. This may result in an increased number of liner slips and irregular vacuum fluctuations, which may affect the incidence of mastitis and poor milkout. Research has shown that herds where the milking machine has inadequate vacuum reserve, there is a correlation with high cell counts due to an increased level of residual milk left in the udder after milking.

If units or other items that need vacuum are added to the milking system without any increase in the size of vacuum pump, then the level of vacuum reserve will be reduced. This may affect the degree of vacuum fluctuation. The actual level of vacuum reserve is to some extent of academic interest. The important question is whether the vacuum requirements for milking and cleaning are satisfied.

Regulator function

It is important that the regulator functions correctly so that a stable vacuum level can be maintained throughout milking. Regulators commonly become blocked with dirt, thereby reducing the amount of air leak-

ing into the system, but occasionally mechanisms become defective. Regulators should be inspected and cleaned every week.

Pulsation system

The pulsation performance should be measured at each individual milking unit. In systems with master pulsation there should be little difference between units. In systems where there are individual pulsators, the differences in performance can be quite considerable.

General condition of the plant, rubberware, etc.

The plant should be examined for any perished rubberware, leaky valves, etc., and its overall condition noted. Liner condition should be assessed and the frequency of change checked to ensure that the liners are replaced at the correct intervals.

Dynamic test

The dynamic test is carried out during milking, as shown in Plate 5.16. Vacuum levels and fluctuations close to the teat end are recorded during the milkout of a few high-yielding cows at the furthest end of the milking system, i.e. at the greatest distance from the vacuum pump and receiver vessel. This is designed to test the plant under 'load'. The length of milking time, together with yield,



Plate 5.16. A dynamic test recording the vacuum levels and fluctuation during milking.

is recorded. The vacuum recording may be measured where the long milk tube leaves the milking cluster or in the short milk tube just below the liner shell. Rear quarters are preferred as they yield more than the fore quarters and this places the system under further load. The level and type of vacuum fluctuation are recorded.

There are two types of vacuum fluctuation that may occur at the teat end: regular and irregular. Regular vacuum fluctuation tends to be constant throughout milking. Figure 5.8 shows some different types of fluctuation that may occur during milking.

Regular fluctuations are caused by pulsation. Irregular fluctuations occur as a result of factors such as inadequate vacuum reserve, unit fall-off, liner slip, etc., and occur intermittently throughout milking. Liner slips are dangerous as they may result in reverse milk flow, leading to impact forces. Impact forces drive milk droplets up against or through the teat canal and are discussed on pages 79–80.

There are no fixed international standards for vacuum fluctuation. However, some researchers have suggested that levels over 10 kPa (3" Hg) are undesirable and may increase the risk of mastitis. Any irregular vacuum fluctuation is undesirable. A continuous recording at the receiver vessel or the sanitary trap is also made over a prolonged period to check that there are no vacuum changes occurring in the plant. If there is any vacuum fluctuation here, it is likely to be further exaggerated towards the teat end.

The behaviour of the cows should be noted during milking. Are they comfortable and content, or are they edgy and uncomfortable? Is there excessive defaecation or urination? The interaction of the machine with the cow should also be observed, along with teat condition assessment postmilking.

It is essential that all the findings from the milking machine test are recorded and a report left on the farm. Test reports should always be discussed with both the milker and the owner, and any faults identified related back to udder health. The best machine test report is the one showing that no faults have been identified. In some cases

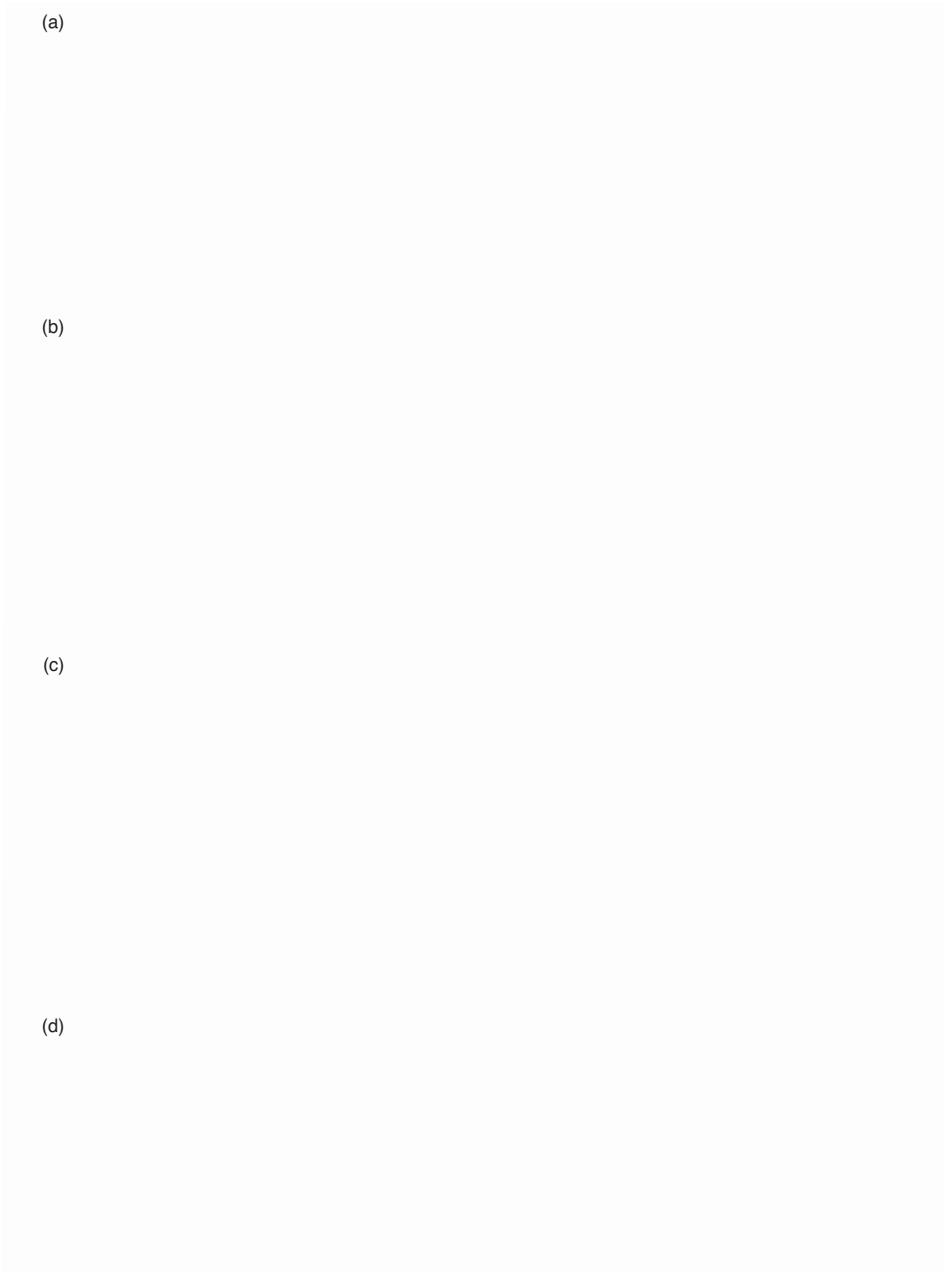


Fig. 5.8. (a) Good tracing showing little teat-end fluctuation. (b) When other milking units are attached, the vacuum level at the teat end drops, as shown by the arrows. This suggests insufficient vacuum reserve or a problem with the regulator. (c) There is a high level of teat-end vacuum fluctuation (20 kPa) from the start of milking to peak flow, which reaches a more acceptable fluctuation (7 kPa) towards the end of milking. The actual vacuum level varies between 13 and 36 kPa at peak flow and between 40 and 50 kPa towards the end of milking. (d) Teat-end vacuum level remains constant throughout, except when there is a high level (17 kPa) of irregular fluctuation caused by liner slip. This may result in impact forces.

when a test report has been left on the farm recommending immediate action, the message is ignored. This may occur because the significance of the fault has not been fully explained to the farmer and he sees no reason to take any action.

The Milking Machine and its Relationship to Mastitis

The milking machine can have an effect on the incidence of mastitis in a number of ways. These include the following:

- Acting as a vector.
- Damaging the teat end.
- Increasing bacterial colonization at the teat end.
- Creating impact forces.
- Undermilking.
- Overmilking.
- Stray voltage.

Acting as a vector

Disease organisms may be physically transmitted from the machine to cows, and in this way mastitis bacteria can be passed from cow to cow. This can occur through contaminated milk remaining on the liner between milkings (Plate 5.17). Research



Plate 5.17. Infected milk remaining on the liner may spread infection to the next six to eight cows milked.

work has shown that following the milking of an infected cow, *Staphylococcus aureus* infection can be spread to the next six to eight cows milked through the contaminated liner. The risk of this occurring is increased if worn liners are used because bacteria are able to adhere more easily to their roughened surface.

Infection may also spread between quarters during milking if there is flooding of milk back up the short milk tubes. If this occurs milk from all quarters will mix, allowing bacteria to contaminate uninfected teats. The amount of contamination will depend on how quickly milk is removed from the liner. Large-capacity claws help limit this effect.

Mycoplasma infections are also spread via the liner. While *Mycoplasma* is not a common pathogen in Europe, it is prevalent in hot desert areas. Affected animals are generally culled, as treatment is ineffective. For this reason, many milking installations have an automatic back-flushing unit that disinfects the milking unit between cows with boiling water. Back-flush units are being fitted in Europe to reduce cow-to-cow transmission of staphylococci and streptococci and also to ensure that the liner is clean before being put on the next cow.

Damage to the teat end

The teat canal is the primary defence mechanism in preventing new intramammary infections. Milking cows through a faulty machine that damages the teat skin or teat end will increase the risk of new infections. Damage to the teat skin, especially cuts and chaps, provides an ideal environment for the growth of mastitis organisms such as *S. aureus* and *Streptococcus dysgalactiae*. Damage to the teat end will allow bacterial colonization and give bacteria easy access into the udder.

One of the commonest and most significant forms of damage is hyperkeratosis. Hyperkeratosis is caused by overmilking, poor pulsation, high vacuum levels, milking with worn liners and/or rough removal of the cluster.

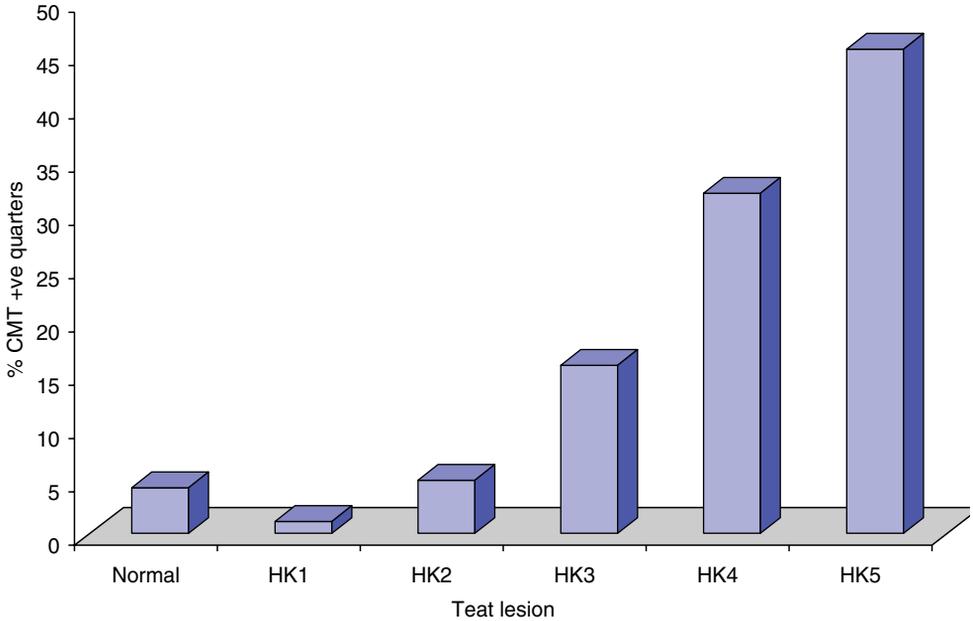


Fig. 5.9. The percentage of teats associated with a high cell count quarter according to the severity of hyperkeratosis. CMT = California Mastitis Test. HK1–HK5 = degree of hyperkeratosis scored on a scale of 0 (normal) to 5 (severe). (Lewis, 2000.)

Any damage to the teat canal is likely to increase the new infection rate. Figure 5.9 shows that quarters with high scores of hyperkeratosis have higher somatic cell counts and therefore higher levels of sub-clinical infection. Almost 50% of cows at score 5 were infected (CMT positive).

Colonisation of the teat canal

Pulsation is important as it allows regular removal of excess keratin from the teat canal during milking. Keratin acts as a type of blotting paper, mopping up any bacteria present and, as on the skin, natural sloughing (removal) of superficial cells helps in the removal of bacteria.

If there are problems with pulsation and the excess keratin is not removed, then there will be a build-up of bacteria in the teat canal. This accumulation is due to reduced milk flow rates, meaning that the excess keratin and bacteria are not ‘stripped’ away from the teat duct. These bacteria will be

able to colonize the canal and, if they penetrate the udder, the risk of mastitis is increased.

A worn or an incorrect design of liner, small-volume clawpieces, narrow-bore pulsation tubes and many other factors may affect pulsation. Problems with pulsation may also occur with short-barrelled liners, as the short barrel may cause there to be insufficient space for the liner to collapse fully around the tip of the teat.

Liner slip and impact forces

Impact forces result in milk particles being propelled from the short milk tube or clawpiece up against the teat end, as shown in Fig. 5.10.

Impacts occur when there is a pressure difference between the teat end and the cluster, often due to liner slip. This difference needs only to occur for milliseconds to create impacts. Milk may be driven at speeds of up to 40 miles per hour (64 k.p.h.). This

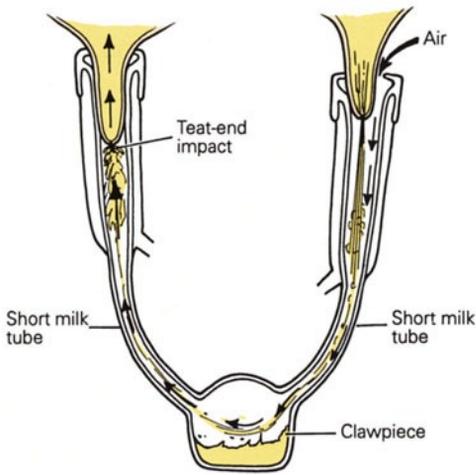


Fig. 5.10. Teat-end impacts are caused when air enters between the teat and the liner, leading to an imbalance of pressure between the teat end and the clawpiece.

force is such that penetration of the canal, which is open during milking (see page 23), may occur. If the milk is contaminated with mastitis bacteria then infection may follow. Pulling the milking unit off the cow without first shutting off the vacuum, machine stripping (see page 113) or liner slip (see Plate 5.18) may all result in impact forces.

Impact forces combined with poor pre-milking teat preparation can result in a high incidence of environmental mastitis. This risk will be increased when cows with dirty teats are washed but not dried, or when water contaminated with environmental bacteria collects around the top of the liner. If the cause of the liner slip is identified and resolved, and pre-milking teat preparation improved, the reduction in clinical mastitis can be immediate and very significant.

Liner slip may occur due to one or more of the following reasons:

- Poor unit alignment.
- Inadequate plant vacuum reserve.
- Cows with small, very large or splayed teats resulting in poor mouthpiece contact.



Plate 5.18. Liner slip creates impact forces that drive milk against the teat end.

- Nervous cows that fidget.
- Excessively large liner mouthpiece.
- Low vacuum levels.
- Poor liner design.
- Heavy cluster weight.
- High vacuum fluctuation during milking.
- Machine stripping at end of milking.

The majority of liner slips result in 'squawking' of air as it enters through the top of the liner. As most liner slips occur towards the end of milking, they pose two dangers. First, there is little resistance at the teat end because the teat canal is at its most open phase (see page 23). Second, if milk does penetrate the teat canal, because there is little milk left to be removed, it is more likely that bacteria will remain in the udder until the next milking, and this will increase the risk of mastitis developing.

There has been a considerable reduction in the incidence of impact forces over the past 20 or so years. This is due to many improvements in milking machines including:

- Larger pump capacity.
- Improved vacuum stability.
- Larger-volume clawpieces.
- Larger-diameter short milk tube.
- Larger air bleed holes.
- Liner shields.

A high incidence of liner slip can have a major impact on the incidence of clinical mastitis, especially if premilking teat preparation is poor.

Undermilking

At the end of milking about 0.5 litres of milk will remain in heifers and 0.75 litres in cows. In some instances, cows may be undermilked, and several litres of milk remain. In these circumstances, undermilking may affect somatic cell count due to the increase in the volume of residual milk and bacteria. These bacteria multiply to greater numbers, with an associated increase in somatic cell count. There may be an increased risk of clinical mastitis. *Streptococcus agalactiae* infections increase significantly if there is undermilking because of the high number of these bacteria shed compared with other mastitis pathogens.

Overmilking

Overmilking may lead to increased levels of mastitis due to the effect on teat damage. Research shows that there is more teat congestion and teat-end damage with overmilking, which results in more clinical mastitis and higher cell counts. Overmilking can be easily assessed by looking at cows towards the end of milking. If there is no milk flowing away from the cluster, cows are being overmilked (Plate 5.19).

If the clawpiece is empty at take-off, this also suggests overmilking. Units should be removed when milk flow drops to 400 ml per minute or before. Overmilking can also occur at the start of milking if there is poor teat preparation, resulting in a poor let-down reflex. This results in 'biphasic' let-down (see page 106).

Stray voltage

Stray voltage is where you have small electrical currents passing through the cow's

body. Stray voltage may occur as a result of poor or faulty wiring, faulty equipment and/or improper earthing. Most researchers agree that levels over 1 volt are significant to udder health.

The reactions of animals to stray voltage vary and depend on the path through the animal and the magnitude of the voltage. Stray voltage problems may be continuous or intermittent and often may be very difficult to detect. There are three general effects: behavioural changes, changes in milking characteristics and reduced production.

Often the first sign of stray voltage is that cows are reluctant to come in to be milked. When stray voltage is a problem, cows become nervous during milking. They are restless, fidgeting during the milking process, and may have to be pushed into the parlour. They may leave the parlour in great haste. This is because cows are frightened and realize that they may be subjected to stray voltage or tingling while they are being milked. There will be an increased number of defaecations and urinations. If cows are happy to come in to be milked and are calm throughout milking, stray voltage is unlikely to be a problem.

A poor milk let-down reflex occurs, resulting in incomplete milking and increasing residual milk in one or more quarters. This is because the cows are frightened, and this reduces the let-down reflex. The number of cows affected and the effect on the let-down reflex depends on the level of stray



Plate 5.19. Overmilking. There is no milk in the clawpiece.

voltage and the reaction of individual cows. Cows are more likely to kick the milking unit off, which can result in impact forces and incomplete milking, which results in more clinical mastitis and a rise in cell count. Incomplete milking will also reduce milk yield.

Simple Machine Checks that can be Carried Out Without Testing Equipment

There are a variety of simple tests that can be carried out to help identify possible machine problems without using sophisticated testing equipment. These are described in the following section and are designed to help identify possible problems. They are intended to complement but not replace routine machine testing, which remains an essential part of any mastitis control programme. Specialist testing procedures will not be discussed.

Vacuum level

- Check that the vacuum gauge reads zero before the machine is switched on. Vacuum gauges are frequently faulty.
- When the machine is turned on, watch the needle rise. Most plants should reach operating vacuum level within about 10 seconds. If it takes a long time to reach the operating level, then check that no valves have been left open – they may be leaking air into the plant.
- Tap the gauge to check it is not sticking.
- What is the operating vacuum level according to the gauge? Check this against the last milking machine test report.

Vacuum reserve

- Set up the plant for milking. For every five milking units, open one so that it sucks air into the system. If the vacuum level falls by more than 2 kPa (0.6"Hg), it suggests that there may be insufficient vacuum reserve. So, for a 20 × 20 parlour, you should be able to leak air in through four milking units.

- Stand in the pit so that you have a clear view of the vacuum gauge. Leak air into the system through one milking unit for 5 seconds. Check if the vacuum level has dropped. Close the unit and record the time it takes for vacuum to return to the normal operating level. This is called the **vacuum recovery time**. It should not exceed 3 seconds. In any system, leaking air in through one unit is equivalent to attaching a cluster to a cow. There should be **no fall** in vacuum level. If the vacuum drops, it indicates that there is a significant problem – possibly inadequate vacuum reserve or perhaps the regulator is faulty or dirty. The plant vacuum level should never drop when leaking air in through one unit.
- Now repeat the test, leaking air in through two milking units for 5 seconds. How far did the level drop and how long did it take to return to normal this time? When two units leak air into the system in small plants, there may be a small drop in vacuum. This should not exceed 2 kPa.
- In large milking systems, there should be no drop in vacuum level if you leak in air through two units.
- A crude test of vacuum reserve is to leak air in through one unit in five (four units in a 20 × 20 parlour) and watch the vacuum gauge. If there is plenty of reserve, the vacuum level will remain steady.

Regulator function

- First check that the regulator is clean.
- Stand close to the regulator during milking and listen. Can you hear air being admitted? The regulator should be continuously sucking atmospheric air into the system, as there should always be surplus vacuum (reserve) available throughout milking. What happens when units are put on, feeders cut in, etc.? If the regulator stops leaking in air, this indicates that the plant is unable to maintain a stable vacuum level, which may be due to inadequate vacuum reserve or a faulty regulator.
- Leak air into the system so that the vacuum level drops by 2 or 3 kPa. Listen to

the regulator. If it is working correctly, it should **not** admit air as the regulator is attempting to raise the working vacuum level back to normal. If it is sucking in air, then the regulator is faulty.

- Watch the gauge rise to normal after lowering the plant vacuum level. If the gauge ‘overshoots’ and rises to above the normal operating level and then settles down, it suggests that it is either dirty or faulty.

Pulsation system

- First check if the pulsation is master or individual, single or dual.
- Listen to each pulsator and check that it is working.
- Place a finger into a liner of each cluster and check that it is moving. In the case of dual pulsation, you will need to check two liners with alternate movement. If no movement is felt, it suggests that there may be defective pulsation in this unit.
- Measure the pulsation rate. Check this result against the last machine test report.
- Check the condition of the short and long pulsation tubes. A hole in a pulsation tube will affect liner movement. The liner may only partially open or may not open at all, as it will be unable to create a full vacuum in the pulsation chamber. This will depend on the size and the location of the hole.

It is easy to demonstrate the effect of inadequate pulsation to the milker: place bungs in two liners and ask the milker to put their thumbs inside the two open liners. Turn on the vacuum supply as for milking and kink the long pulsation tube. This will mimic continuous milkout without pulsation and will stop blood circulation around the teat or thumb. Watch the effect on the milker and see their red and swollen thumb afterwards, as shown in Plate 5.20.

Liners and rubberware

- While checking the pulsator action, feel the inside of the liner. Is it soft and smooth, or rough and cracked?

- If you have a pencil torch, shine it inside the liner and have a look.
- How often are the liners changed?
- How many milkings have they done between changes?
- How frequently should they be changed?
- Take a liner out of its shell: is it collapsed or round?
- Split the liner lengthways and look at its condition. (Always make sure that you have a replacement liner before you do this or milking will be difficult!) Is the liner clean?
- Check the condition of the rest of the rubberware for cracks, holes or splits.

Other checks

- Check the air bleed hole on the cluster. If it is blocked, milk will not flow away from the cluster easily. If milk runs back out from the liners when the cluster is removed, it indicates that the air bleed hole may be blocked.
- When was the machine last tested?
- What tests were carried out?
- Have all the problems identified at previous visits been corrected?
- Was a report left and were the results discussed fully?
- Who usually tests the plant?



Plate 5.20. If there is no pulsation, blood circulation stops as shown in the ‘thumb test’.

Observations to be carried out during milking

- Check that the units sit on the udder comfortably.
- Is there any evidence of liner slip?
- Are cows happy during milking or kicking and restless?
- Check the functioning of the ACRs.
- Are units pulled off while still under vacuum?
- Are cows under- or over-milked?
- Do cows kick at the clusters at the end of milking?
- Check that the vacuum gauge remains static during milking.
- Check teat condition after the unit is removed. Look for evidence of teat damage, such as small haemorrhages, sphincter eversion, distortion and cyanosis of the teat skin as soon as the cluster is removed from the cow.
- Is the regulator leaking air in throughout milking?
- Do units fall off for no apparent reason?
- Count the number of biphasic let-downs, which is an indication of poor let-down.

Further details are given in the parlour audit section at the end of this book.

Wash-up Routines

An efficient parlour wash-up routine will remove milk residues and bacteria from the plant. This will maintain milk quality, improve the appearance of the parlour and prolong the life of milking equipment. Problems with the wash-up routine will result in milk residue and bacterial build-up within the system. This will increase the Bactoscan and TBC (see Chapter 10).

The milking system is washed by the physical cleaning action of air and water, assisted by temperature and detergent chemicals, and then the plant is disinfected. No matter what system is used, the machine will not be adequately cleaned unless wash-up solutions come into contact with all soiled parts of the plant. All too often, poor cleaning is due to poor circulation of solu-

tions, blocked jetters, low temperature or an inadequate volume of wash solution.

The following will be required, irrespective of the type of milking system:

- A supply of potable water (water free from faecal contamination).
- An efficient water heater.
- A thermometer.
- Chemicals.
- Protective clothing.
- For circulation cleaning, one, if not two, wash troughs.

British Standards require a minimum of 18 litres (4 gallons) of hot water per milking unit for circulation cleaning or acid boiling wash. Less than this and the Bactoscan or TBCs may increase. Remember that some hot water may be used for other purposes, such as feeding calves, etc. Recorder plants (where there is an individual glass jar for each milking unit) or large-bore pipelines may need over 18 litres of boiler capacity per unit.

The milking system should be cleaned immediately after milking while the plant is warm and before milk deposits start to form on pipes. Two forms of cleaning are used: circulation cleaning and the acid boiling wash (ABW). Circulation cleaning is the most common method in the UK.

The milking system is designed to produce minimal turbulence of milk during milking because excess turbulence may lead to impact forces or 'buttering' of milk. However, during the wash-up routine, maximum turbulence is required to make sure that all internal surfaces of the plant are thoroughly cleaned. In some parlours, more vacuum reserve will be needed to draw wash solutions through the plant than is needed to provide stable vacuum during milking.

Direct to line and some other plants are fitted with air injectors, which admit 'slugs' of air to increase turbulence by bubbling and swirling water all around the pipes. Air injectors are essential in large-bore systems so that the entire surface of each line can be physically cleaned. Plate 5.21 shows an air injector sited at the junction of the wash line and the milk transfer line.



Plate 5.21. Air injectors are fitted to the milk transfer line to maximize agitation of wash solutions to ensure that large-volume lines are cleaned.

It is essential that all dairy chemicals are stored safely and used correctly. Protective clothing (goggles, gloves and aprons) should be worn when handling chemicals to avoid accidental injury. Chemicals must not be stored in the same room as the bulk tank to avoid any risk of milk taint occurring.

If problems occur with the wash-up routine, then milk films can build up, as shown in Fig. 5.11. These films provide nutrients for bacteria, which can then multiply and increase the Bactoscan or TBC (see Chapter 10).

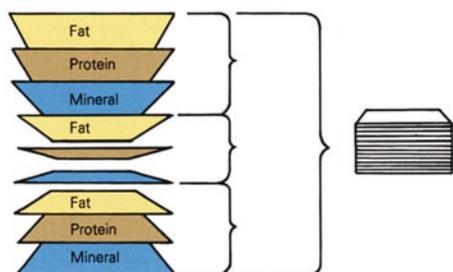


Fig. 5.11. If problems occur with the wash-up routine, milk films will build up in the plant. This diagram shows how milk films develop.

There are two types of milk soil: organic and inorganic. Organic soils are composed of milk fat (butterfat), protein and sugar. If these residues are left in the plant, they will harden as they dry. Inorganic soils result from mineral deposits, such as calcium, magnesium and iron, and are often referred to as ‘milkstone’.

Butterfat

The temperature and pH of the cleaning solution must be right in order to ensure that all butterfat is removed. Butterfat starts to solidify at temperatures under 35°C. This is an important consideration for the rinse cycle of circulation cleaning. Alkaline detergents are used to remove butterfat and must be capable of emulsifying (breaking down) the fat globules so that they can be removed from the system. If butterfat solutions build up in the plant, they trap other forms of milk soil and also have a detrimental effect on rubber components.

Protein

Protein films are hard to see. They adhere strongly to pipes and are difficult to remove. Alkaline detergents with chlorine added (i.e. chlorinated) can break down protein so that it can be removed from the plant. However, water at high temperatures can bind protein deposits back on to milk pipes.

Minerals

Calcium, magnesium and iron may cause problems if allowed to precipitate, particularly in hard water areas, where milkstone can easily build up on the pipes. This is seen as a chalky film. Acid solutions are used to remove and prevent the accumulation of mineral deposits and most dairy farmers run an acid wash (milkstone remover) through the plant on a regular basis. These washes usually contain phosphoric acid. This is usually carried out weekly in areas with hard water.

Bulk tanks

The bulk tank must be cleaned every time milk is emptied from it. Most tanks are now cleaned by automatic washers that rely on chemicals and jetters to complete the process. All internal parts of the tank that can come into contact with milk must be cleaned and disinfected. It is essential that the milker checks that this process has been carried out efficiently before milking.

While automatic washers do a good job, there is the risk that, because they are automatic, things can go wrong. The milker rarely looks at the tank to check on cleanliness. Occasionally problems do occur, such as when a wash jetter becomes blocked and so part of the tank is not cleaned. Another common fault is when chemicals that are feeding the automatic washer run out. This causes a build-up of milk film, in which psychrotrophic bacteria can multiply, resulting in an increased Bactoscan or TBCs. Psychrotrophs are bacteria that thrive under refrigerated conditions.

Air lines

Occasionally, milk enters the air lines. For example, this can occur if there is a split liner. In this case, milk will be sucked up through the pulsation chamber into the pulsation tubes and into the main pulsation line. If milk does enter the air lines, then this needs to be cleaned out. All air lines should be washed twice a year as a minimum.

Circulation Cleaning

Circulation cleaning is divided into three cycles.

- Rinse: removes excess soil.
- Wash: cleans the plant.
- Disinfect: removes residual bacteria from the cleaned plant.

When milking is completed, any milk in the receiver vessel and the milk pump should be drained. The milk pipe should then be dis-

connected from the bulk tank. The external surfaces of clusters and milking units should be rinsed clean (ideally with warm water) and the plant set up for the wash-up routine. This consists of attaching jetters to the clusters and then transferring vacuum to the wash lines so that wash water is drawn into the wash lines, through the cluster and back through the return wash line, as shown in Fig. 5.12.

Rinse cycle

Warm water at body temperature (38–43°C) should be rinsed through the milking system and run to waste until the water appears clear immediately after milking. This will remove the majority of any residual milk left in the plant. Many dairy farmers use a cold water rinse. Under no circumstances should cold water be used as it will congeal butterfat onto glass and stainless steel fittings and cool down the plant before the hot wash. Energy in the form of hot water will then be required to heat up the pipes before the hot wash. In addition, minerals and sugars in milk are more easily dissolved in warm water.

After the rinse cycle, the wash line valves should be shut off to prevent large volumes of air being sucked into the system. This air may cool down the plant before the hot wash. It is advisable to insulate the milk and wash lines in colder climates. This is essential where any of these pipes are exposed to the outside environment. Not only will it prevent excessive cooling during the wash-up process, but it will also prevent milk freezing during winter milking. An efficient rinse cycle should remove about 95% of all milk residues in the system and all the milk sugars. The remaining residues are removed during the wash cycle by chemical action.

Wash cycle

In circulation cleaning, the wash cycle relies on an alkaline detergent solution to remove butterfat. Chlorine is normally incorporated into the detergent to remove protein, but in this form the chlorine has no disinfectant

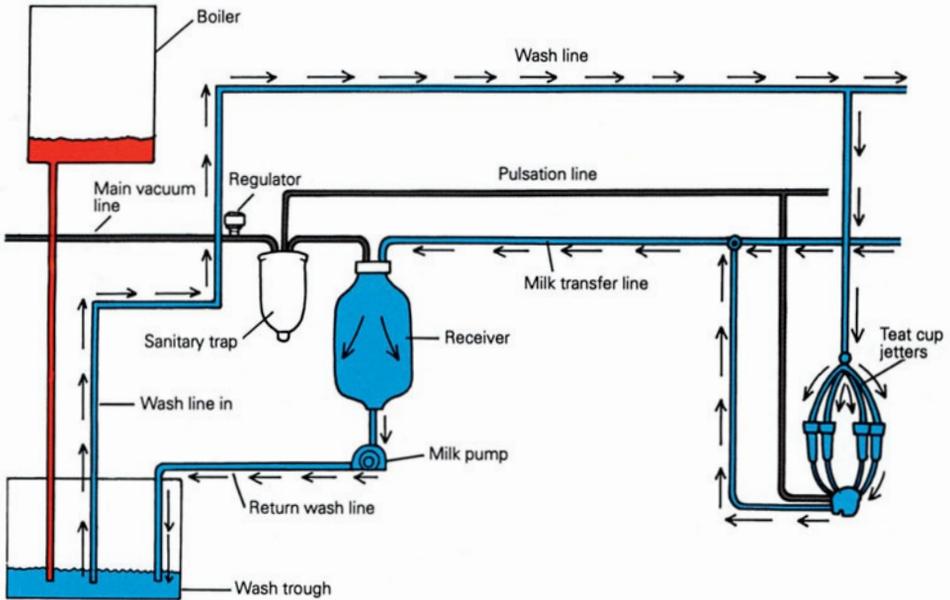


Fig. 5.12. With circulation cleaning, water is drawn in the wash lines, through the cluster, back through the milk transfer line and into the wash trough, where it is recirculated.

property. Wash-up solutions are very sensitive to temperature. In general, their cleaning power doubles for each 10°C increase up to a maximum of 71°C. Once they exceed this temperature, they tend to become unstable, vaporize and become less effective. There are, however, a few detergent solutions that are designed to be circulated in cold water, so always follow the manufacturer's instructions.

An adequate supply of hot water must be available. Maintaining the correct temperature of water in the boiler is essential. It is also important that the boiler has a large-bore tap so that the wash trough can be filled rapidly without heat loss.

The water temperature should be checked regularly against the boiler gauge to ensure that the thermostat and heater element are functioning correctly (see Plate 5.22). Sometimes the boiler gauge becomes faulty or the heating element becomes caked with mineral deposit. This is especially common in hard water areas. The best way to check on boiler efficiency is to fill a trough with hot water and measure the temperature in the



Plate 5.22. Checking the temperature of the hot wash solutions.

filled trough. This is the temperature that counts.

Detergent solutions must be used correctly and to do this you must know the volume of wash water. Check the manufacturer's instructions on how much detergent to use. If the solution is too weak it will be ineffective. If it is too strong, then it will be wasteful and may even corrode the stainless steel or rubberware in the plant.

Hot water is run into the plant and, as it travels around, it heats up the pipes and vessels. Only then should the correct amount of detergent solution be mixed into the circulating hot water. It should be circulated at 60–70°C for 5–8 minutes, or in accordance with the manufacturer's recommendation.

The circulation wash relies on the detergent action but also the physical swirling action of wash solutions. Air injectors create turbulence that is essential in order to have effective cleaning in large direct to line systems.

The milker should not hose down the outside of the recorder jars with cold water during the rinse or wash cycles as this will cool down the temperature of the jars and also of the circulating solutions.

If the rinse cycle has not been effective, a large amount of milk residue may be left in the system. This milk will inactivate some of the detergent, which will decrease the effectiveness of the wash-up routine.

Ideally a thermometer or temperature recording strip should be fitted to the return wash pipe to check that the wash solutions are being circulated at the correct temperature (see Plate 5.23).

If solutions are circulated for long periods of time, their temperature drops and then protein may be deposited back onto the pipes. Milkers have been known to let the hot wash cycle run while they go and have breakfast. At the end of the circulation wash cycle, the milking plant should be clean and free from any milk soil.

Disinfection cycle

The disinfectant rinse reduces the number of bacteria in the plant and helps maintain

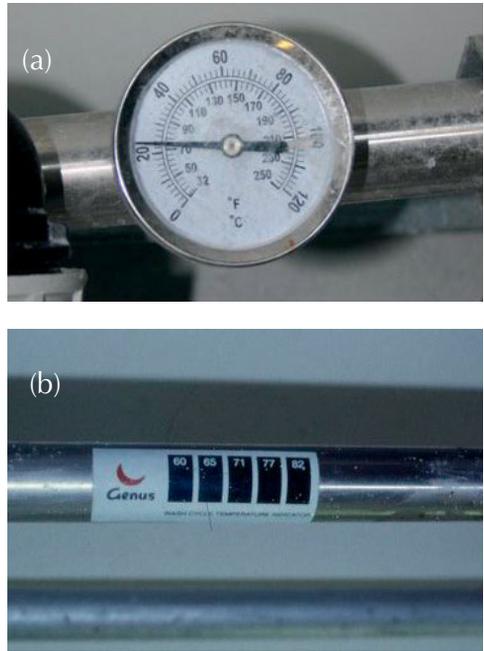


Plate 5.23. A thermometer (a) and a temperature strip (b) attached to the return wash line to check that wash solutions are circulated at the correct temperatures.

milk quality. Sodium hypochlorite is the most commonly used disinfectant, at a strength of 50 p.p.m. (parts per million). This solution may be circulated and then dumped to waste at the end of the cycle.

If you examine the internal surfaces of some rubber components in the plant and a black deposit marks your finger, as shown in Plate 5.24, this indicates rubber damage caused by too high a level of hypochlorite. The same effect may be seen in the black colour of the disinfection rinse shown in Plate 5.25. Remember that chlorine compounds reduce the life of all rubberware and liners.

Summary of common problems associated with circulation cleaning

- Water is not hot enough – this will make the detergent solutions less effective as they are temperature-sensitive.



Plate 5.24. Black deposits from rubber parts indicate too high a level of hypochlorite resulting in rubber corrosion.



Plate 5.25. High levels of hypochlorite in the disinfection solution strip away rubber.



Plate 5.26. If there is insufficient turbulence or flow of wash solutions, cleaning may be ineffective, as shown.

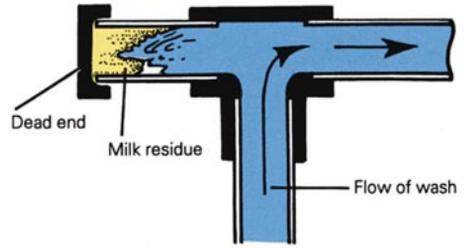


Fig. 5.13. Deposits frequently build up in dead-end areas as they are difficult to clean.

- Inadequate wash volumes – water may not come into contact with all internal surfaces. This may result in some areas of the system not being cleaned, especially the top part of the milk lines.
- Rinsing the plant with cold water after milking – this will cool down the warm plant and congeal butterfat. The hot wash solutions will then have to heat up the plant from cold, and the detergents will have to remove the butterfat deposits.
- Incorrect strength of detergents used – too little is ineffective while too much is costly and corrosive.
- Wash cycle continued in excess of the recommended time – the solutions will cool down and may re-deposit material back onto the internal surfaces.
- Build-up of deposits in dead-end areas that are difficult to clean, as shown in Fig. 5.13.
- Insufficient turbulence or flow of wash solutions – cleaning may be ineffective and deposits can accumulate on pipes (see Plate 5.26). Build-up of milk soil occurred in this plant due to inadequate flow of wash solutions.
- Blocked wash-up jetters – this may result in one liner or a complete milking unit not being washed. The effect will depend on where the jetter is blocked.
- Faulty air injectors – these will not create the physical turbulence that is needed to clean large-bore lines.

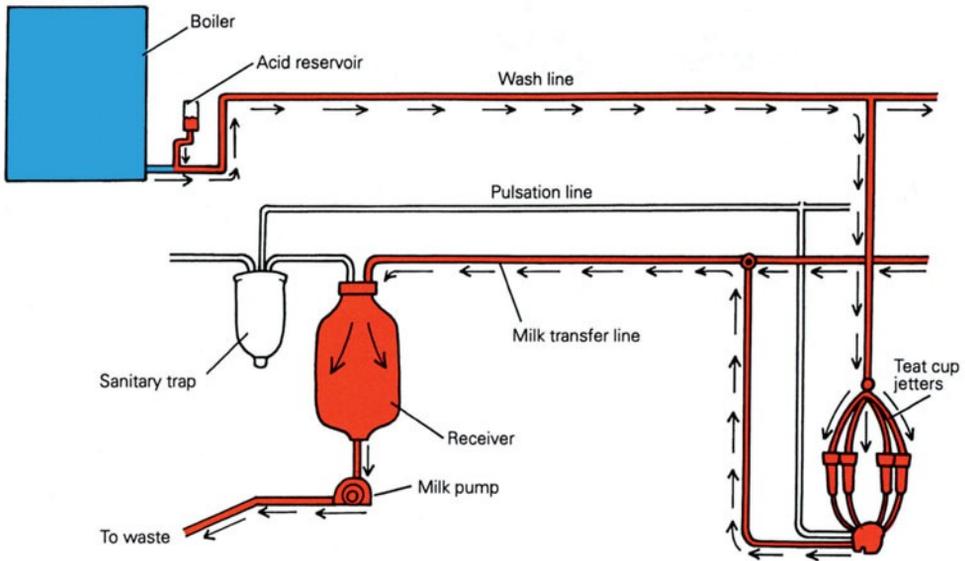


Fig. 5.14. The route of wash solutions with acid boiling wash. The boiling water and acid are run through the plant to waste.

Acid Boiling Wash (ABW)

An ABW relies primarily on heat for disinfection. Large amounts (18 litres or 4 gallons) of boiling water over 96°C are needed for each milking unit. This water is run directly from the boiler around the plant to waste, as



Plate 5.27. Acid boiling wash (ABW) relies on heat and acid for cleaning. Acid is put into the reservoir and released with the boiling water to run around the plant to waste.

shown in Fig. 5.14. It takes the same path as for circulation cleaning, the only difference being that the solutions are run around the plant to waste rather than circulated.

In order to be effective, all parts of the plant must reach and maintain a temperature of 77°C for the whole cycle, which lasts between 5 and 6 minutes. In the first few minutes of cleaning, a solution of dilute nitric or sulfanilic acid is run in with the boiling water to prevent deposits building up on any of the surfaces. See Plate 5.27.

The plant must be capable of withstanding high temperatures and acids. There should be no dead ends and the whole system should be as compact as possible to avoid excessive heat loss. This form of cleaning is not very popular as problems occur if the water temperature or volume is too low. ABW saves on dairy detergents and is a faster form of washing compared with circulation cleaning. However, it requires the boiler to heat the water to a very high temperature, which can require considerable amounts of energy.

Manual Washing

Dump buckets and their clusters can either be washed as part of the wash-up routine or manually using chemicals and brushes in any system. While this process is labour-intensive and time-consuming, excellent results can be achieved and in addition, the milker is able to visually check on the efficiency of the process. It is important that clusters of dump buckets are thoroughly cleaned and disinfected, as they are often used to milk colostrums from freshly calved cows, which are very prone to mastitis (see page 30).

Some farmers place clusters and other pieces of milking equipment in troughs with small volumes of detergent solutions, as shown in Plate 5.28, and expect good results. This has little effect as the solutions do not come into contact with all the internal surfaces and there is no physical cleaning action. In addition, if this method of cleaning is used on clusters that milk mastitic cows, then this must increase the risk of spreading infection, as liners may remain contaminated with mastitis organisms (see page 111).

If a Wash-up Problem is Suspected

The efficiency of the wash-up routine can be evaluated through laboratory testing of bulk milk, as described on pages 176–183. If a



Plate 5.28. Cleaning will be ineffective as there is insufficient detergent solution to come into contact with all parts of the cluster.

wash-up problem is suspected, the cause must be identified. Much can be gained by manually inspecting the system after the wash-up routine, for example by removing pipe ends and examining internal surfaces with a torch.

Look inside the following areas for any evidence of milk film or milk soil build-up:

- Liners.
- Milk transfer lines (especially the top of the line).
- Bungs and valves at the base of jars and lines.
- ACR flow meters and sensors.
- Receiver vessel.
- Dead-end areas.
- Milk pump.
- Bulk tank.

Common Faults Found with Milking Machines

Below are a variety of milking machine problems and their consequences:

- Mix and match parlours (see Plate 5.29). Some farmers or milking machine fitters do unusual things with parlours. Plate 5.29 shows a parlour with eight units milking through recorder jars and four units milking direct to line. Parlours must be either direct to line or a recorder jar system, not a combination of the two. The



Plate 5.29. A combination of a recorder jar and direct to line parlour, which had a very poor milking performance.



Plate 5.30. Excess bends in pipelines.



Plate 5.31. Hole in the short air tube.



Plate 5.32. Split liner.

milking performance in this parlour was very poor.

- Excess bends in pipes (Plate 5.30): these impede air- and milk flow and may reduce the amount of vacuum available at the teat end, increasing the risk of irregular vacuum fluctuations in the plant and leading to an increased infection rate.
- Hole in the short pulse tube (Plate 5.31): atmospheric air is sucked in through the short pulsation tube. This will prevent full vacuum levels being attained in the pulsation chamber, resulting in incomplete liner opening, which will slow down milking in that quarter. In severe cases the liner may not open at all, resulting in failure of that quarter to milk out.
- Split liner (Plate 5.32): a split liner is unable to open and close normally. This has two effects. First, it will result in incomplete milkout and massage, leading to teat damage, which may lead to an increased risk of new infections. Second, milk will get sucked into the pulsation chamber, up through the pulsation tubes and into the long pulsation line. This may affect pulsation itself.
- Congestion of blood in the teat (Plate 5.33): this can occur due to a variety of reasons, including:
 - Absence of pulsation, i.e. full vacuum constantly applied to the teats.
 - Incomplete or defective pulsation.
 - Excessive vacuum levels.
 - Poor liner design.
 - Using incompatible liners and shells.
 - Overmilking.



Plate 5.33. Purple teats after unit removal. This is caused by congestion of blood in the teat.



Plate 5.36. A flooded sanitary trap due to a faulty milk pump, which could not remove milk from the receiver vessel.

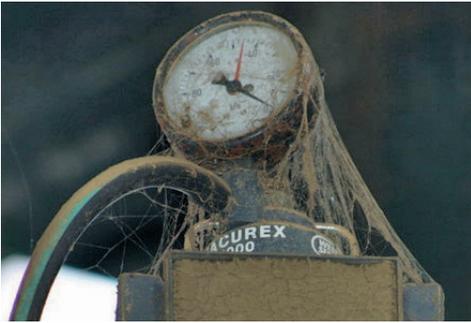


Plate 5.34. Dirty regulator. Blocked air filters may result in higher vacuum levels.



Plate 5.35. Multiple weight-controlled regulators.



Plate 5.37. Poor unit alignment, resulting in the cluster twisting on the udder.

- Congestion of the teat causes the cow great discomfort and the milk let-down reflex is likely to be reduced. If teat damage occurs, this will increase the likelihood of mastitis.
- Blocked regulator filter (Plate 5.34): a dirty regulator may be unable to respond rapidly to vacuum changes in the system, resulting in poor vacuum stability. This can increase the likelihood of irregular vacuum fluctuations thereby increasing the risk of new infections.
 - Multiple weight-controlled regulators (Plate 5.35), which act independently of one another. They all try to maintain stable vacuum in the system; however, as they respond slowly to pressure changes, it is likely that they may work against each other, resulting in poor vacuum stability.
 - Flooded sanitary trap (Plate 5.36): this sanitary trap flooded and the floating ball shut off the vacuum supply and all the units fell off the cows. This occurred due to problems with the milk pump, which could not pump the milk away from the receiver vessel quickly enough.
 - Poor unit alignment (Plate 5.37) due to long milk tubes or poor placement of the milking unit in relation to the position of the cow in the parlour may result in clusters twisting on the udder. This will increase the risk of liner slip, will slow down milking as the teats become twisted in the cluster and may also increase the risk of undermilking one or more quarters. Good unit alignment is essential to ensure efficient milkout.

6 The Milking Routine and its Effect on Mastitis

Minimizing Transfer of Infection	96
Foremilking	97
Mastitis Detection	98
Teat Preparation	100
Predipping	101
Drying Teats before Milking	103
Assessing Teat Preparation	104
Milk Let-down Reflex	105
Batch Preparation	107
Unit Attachment	107
Milking the Mastitic Cow	108
Unit Removal	111
Disinfection of Clusters between Cows	111
Residual Milk	112
Machine Stripping	113
Postmilking Teat Disinfection	113
Milking Order	113
Frequency of Milking	114
Robotic Milking	114
Summary of the Milking Procedure	115

This chapter describes the various processes that constitute a good milking routine and discusses the way in which they can protect herd cell count and reduce clinical mastitis, while at the same time speeding up milking.

A good milking routine will remove milk efficiently from the cow with minimal risk to udder health. It must include practices that limit the spread of contagious mastitis in the parlour, while minimizing the risk of environmental mastitis. This results in quality milk production with low bacterial contamination. The milking routine should be designed to achieve these goals but at the

same time it must be practical and labour-efficient. The milker needs to understand the scientific reasoning for each step in the milking process in order to achieve these aims.

It is important that a consistent milking routine is practised in the herd. Cows love uniformity. They are easily stressed and so rough handling or aggressive milkers are to be avoided. Cows can become nervous and this can affect their milk let-down reflex. The conscientious dairyman will benefit from a good routine by reduced levels of mastitis, increased milk production and a rapid milkout.

The beneficial effects of good hygiene at

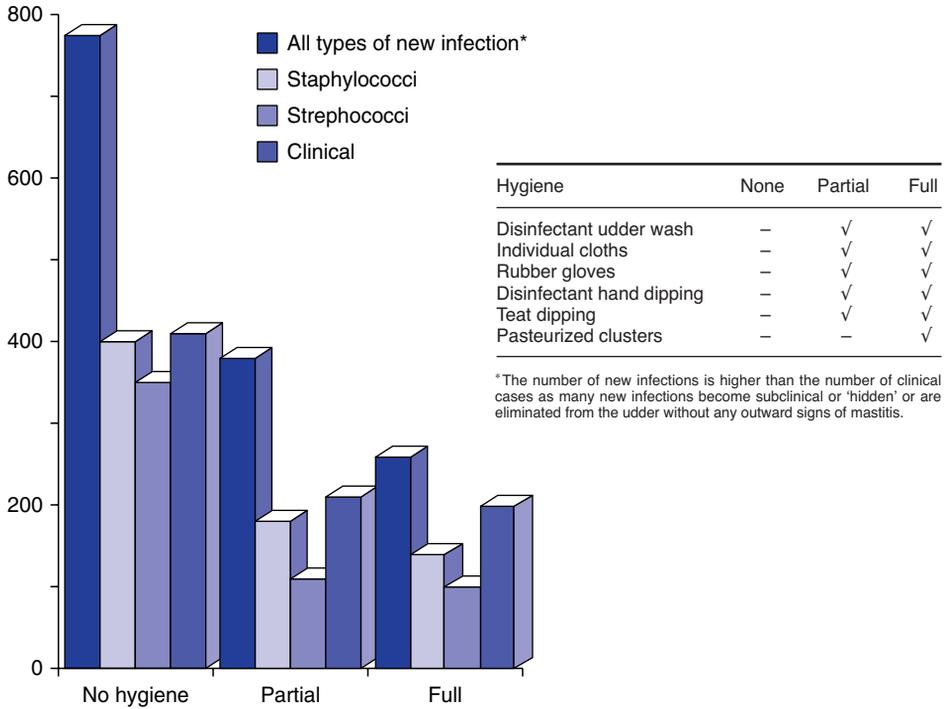


Fig. 6.1. Effect of different hygiene regimes on new infection rate and clinical mastitis. (From Neave *et al.*, 1969.)

milking time are shown in Fig. 6.1., which demonstrates how different hygiene practices can result in a reduction in clinical mastitis and also in the new infection rate. This is to be expected as contagious organisms are transferred between cows during the milking process.

In this trial there is only a slight reduction in infection levels between 'partial' and 'full' hygiene, indicating that at the time of this particular trial, the benefits of pasteurizing clusters between individual cows were limited.

Minimizing Transfer of Infection

Infection can be transferred from cow to cow during the milking process by:

- Liners.
- Hands.
- Cloths.

Control of transfer is based on wearing gloves, using a separate cloth for each cow and minimizing transfer on liners by maintaining liners in good condition and cluster flushing as necessary.

The milker can spread contagious mastitis as he handles each cow. It is extremely difficult to disinfect the rough surface of hands, let alone keep them clean during milking (see Plate 6.1). For this reason it is advisable to wear clean gloves, but it is essential to keep them clean throughout milking.

Trial work in 1966 (Neave *et al.*) showed that half of all milkers' hands were infected with mastitis organisms even before milking had started. Contamination increased during milking so that by the end all milkers' hands were infected.

In another experiment (Neave *et al.*, 1966), two groups of milkers' hands were cleaned in different ways. The first group were washed with a disinfectant solution,



Plate 6.1. Hands have rough surfaces that are difficult to clean compared with the smooth surfaces of rubber gloves.

and afterwards only 30% of hands remained contaminated. However, in the second group, whose hands were just washed in water, 95% remained infected.

Once gloves become worn or torn, they must be discarded. Many milkers wear disposable gloves that are discarded after each milking. Gloves themselves do not reduce the spread of infection. They only allow you to disinfect hands. To be effective, gloves must, of course, be rinsed frequently in a disinfectant solution during the milking process.

The use of rubber gloves is especially important when dealing with *Staphylococcus aureus* or *Streptococcus agalactiae* infections. *S. agalactiae* has been isolated from milkers' hands up to 10 days after their last contact with infected animals. Indeed, some relief milkers are known to have introduced infection into clean herds in this way.

Foremilking

Foremilking is the practice of hand milking each teat before unit attachment. It is recommended for three reasons. It:

- Stimulates the milk let-down reflex.
- Aids detection of mastitis.
- Flushes out the milk from the teat canal. This will remove most bacteria that have entered the teat since the previous milking.

Early detection of mastitis allows prompt treatment of clinical cases. This not only results in higher cure rates, but, more importantly, reduces the risk of spreading infection to the rest of the herd. It also stops mastitic milk from entering the bulk tank and this helps to avoid high bacterial and cell counts. In the case of *S. agalactiae* and *Streptococcus uberis* infections, up to 100,000,000 organisms per ml of milk can be shed from an infected quarter. This can account for the fluctuating Bactoscan or TBC levels that are frequently found in herds with an *S. agalactiae* or *S. uberis* problem.

Frequently there are clots in the first two or three strippings from cows but the remainder of the milk appears normal. This is probably a response to bacteria in the teat sinus but not in the udder itself. In such cases, only the foremilk needs to be discarded but the cow should be marked and carefully checked at the next milking.

Internal teat sealants are now commonly used during the dry period. It is important that milkers can differentiate between debris from these sealants and clinical mastitis. Internal teat sealants have a brighter white colour than mastitis clots; they feel rubbery and break down more easily.

Potentially, foremilk does have some disadvantages. It is time-consuming and may spread infection. For example, if you have a mastitis incidence of 45 cases per 100 cows per year, nearly 5500 teats will have to be foremilked to detect one case of mastitis when milking twice a day. This increases to over 8000 teats when milking three times daily.

Some feel that there is a greater risk of spreading infection from cow to cow as the milker's hands or gloves become contaminated with mastitis organisms, and they consider that this outweighs the risk associated with a failure to detect mastitis at an early stage. However, a good milker will be wearing clean gloves and will postdip cows, which will partially overcome these risks. Compared with the risk from liners, which are in contact with teats for very much longer, one would expect that the risk from regularly disinfected gloved hands is relatively low.

More and more farmers now foremilk as they see a large benefit from having a good milk let-down reflex, which speeds up milking and also maximizes yield. Foremilking is also the only accurate method to detect clinical mastitis.

Foremilking should be carried out before the teat preparation. Any milk that contaminates the milker's hands will then be removed before infection can spread to the next cow. Milk should be stripped onto the parlour floor rather than into a strip cup (see Plate 6.2) as strip cups tend to become reservoirs of infection rather than acting as an aid to detection. A black tile built into the parlour floor under each cow's udder allows easier examination of milk. When foremilk- ing, hold the teat between your finger and thumb and not in your whole hand, as this further decreases the risk of infection transfer. Some milkers use both hands and foremilk two teats at a time.

Mastitis Detection

Mastitis is inflammation of the udder. The appearance of the milk changes with the



Plate 6.2. Milk should be stripped on to the parlour floor rather than into a strip cup.

type of inflammatory response and it may look clotted, watery or stringy, as shown in Plate 6.3. It is not possible to be sure which organism is causing the mastitis simply from the appearance of the milk alone.

The milker may detect mastitis by one or more of the following methods:

- Foremilking.
- Change in the behaviour of the cow.
- Observation of quarter swelling.
- In-line mastitis detectors.
- Checking the milk sock or filter at the end of milking.

The importance of foremilk- ing has already been discussed and this is the best and most reliable form of mastitis detection. Some herds rely on the other methods mentioned above.

When a cow comes into the parlour on a different side or at a different time from usual, then this suggests that something is wrong. She may be sick or it could be due to other factors, such as bulling. Good stock- manship can help identify early cases of mastitis by picking out cows that act out of character.

There are occasions when the cow has a visibly swollen quarter but the milk appears normal (see Plate 6.4). If the cow is healthy and the milk is normal, this cow should not be treated but marked and checked carefully at the next milking.

If the cow is ill, this may be due to a very high temperature, e.g. from infection with *Streptococcus uberis*, or to toxins produced in the udder by a peracute form of mastitis, caused by organisms such as *Staphylococcus aureus* or *E. coli*. This may occur so rapidly that the milk still appears normal. In these cases prompt veterinary treatment will be necessary in order to save the cow's life. On other occasions, there is no swelling in the quarter and the milk shows very little change but the cow is again very sick, due to *E. coli* mastitis toxæmia; this is discussed in detail in Chapter 3.

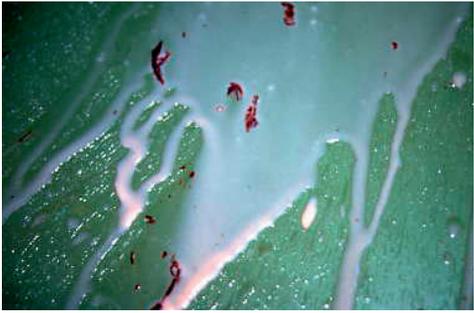
In-line mastitis detectors can be fitted to the long milk tube (Plate 6.5). They have a wire mesh filter through which most milk passes. Any clots present clog the filter. Milk



(a)



(b)



(c)



(d)

Plate 6.3. Various types of mastitic milk; (a) Brown, watery secretion, typical of *E. coli* infections; (b) Watery milk with some clots; (c) Viscous red/brown secretion is associated with gangrenous mastitis; and (d) Clotted milk indicating mastitis.



Plate 6.4. Udder palpation is useful when quarters become hot and inflamed, as shown.



Plate 6.5. In-line mastitis detectors are fitted to the long milk tube and, if clots are present, they clog the filter.

should be able to bypass the filter without impeding milk flow. Filters should be located in the long milk tube either at eye level or close to the clawpiece so that they are easily seen when the units are removed. All too often, they are sited in a location where they are difficult, if not impossible, to observe.

These detectors can give a false sense of security to milkers. Some assume that all cases of mastitis can be discovered using this method, even if filters are never checked. This is not true. Mastitis cases that produce watery milk or very small flecks can be washed through the detectors and cases missed. In-line filters pick up clotted forms of mastitis.

In order to be effective detectors must be examined after each cow is milked. In parlours fitted with recorder jars, these should be checked before milk is transferred into the bulk tank. Problems with Bactoscan or TBC and cell count occur when detectors are not checked, because mastitis goes unseen and mastitic milk enters the bulk supply. This is particularly the case with direct to line plants, which rely solely on detectors for mastitis identification. Here mastitic milk will have reached the bulk tank by the time clots are seen on the filter. However, despite their limitations, in-line mastitis detectors can still be an additional aid in detecting mastitis, but be aware that they can cause problems with vacuum stability at the teat end due to interfering with the milk flow in the long milk tube.

Examining the milk sock or filter after milking is also important, especially to check on the hygiene of the milkers. (The milk sock or filter is located between the milk pump and the bulk tank.) The presence of clots or large amounts of faecal contamination, as shown in Plate 6.6, indicates a poor milking routine and/or poor mastitis detection.

Some farmers rely solely on checking the milk filter for mastitis detection. When clots are found, the procedure is to strip out the entire herd at the next milking to identify the infected cows. In some instances, milkers stop stripping when one mastitic cow has been identified. This may leave other mastitis cases undetected. On other occa-



Plate 6.6. The presence of clots or large amounts of faecal contamination on the milk sock indicates a poor milking routine and/or poor mastitis detection.

sions, no mastitis may be found as the cow may have cleared up the infection herself. Even then, it would be best to foremilk the herd again at the next milking.

Clinical mastitis is detected by visual changes to the milk and udder. Some milkers carry out additional tests such as the CMT (California Mastitis Test) to decide if a quarter is clinical based on the CMT result. If a milker has to resort to further tests to try and detect if a quarter has clinical mastitis, the cow **does not** have clinical mastitis.

In all cases of clinical mastitis, it is advisable to collect pretreatment milk samples for bacteriological testing. This will allow the identification of the types of mastitis present on the farm so that specific control measures can be implemented.

Teat Preparation

One Somerset dairy farmer has a notice up in his dairy for his milkers, 'If the cows' teats are not clean enough to put in your mouth, then they are not clean enough to put the cluster on.' This sums up premilking teat preparation perfectly.

Good teat preparation is essential for clean milk production. It also helps to reduce the risk of environmental mastitis. The goal in teat preparation is to ensure that teats are **clean and dry** before the milking units are attached (see Plate 6.7). The best way to ensure that the cows are clean as they



Plate 6.7. Ensure that the teats are clean and dry before the milking units are attached.



Plate 6.8. Hairy udders are likely to trap dirt and hence increase the risk of environmental mastitis.



Plate 6.9. Long tails should be trimmed, otherwise they will spread dirt on to the other and teats.

enter the parlour is to make sure that they are kept in a clean environment. This is especially important during the housing period. If cows enter the parlour with dirty teats, then problems with environmental management need to be addressed.

Hairy udders, as shown in Plate 6.8, are likely to trap dirt and this adds to the amount of work the milker has to do. Udders should be clipped or singed. Similarly, excess hair on the end of the cow's tail should be cut off (Plate 6.9) and the sides of the tail clipped, ideally up to the vulva.

Predipping

Predipping is the best way to prepare teats and refers to the disinfection of the teat before milking. The aim is to reduce the number of bacteria present on the teat before the milking unit is attached. This will greatly reduce the number of environmental bacteria entering the milk, and in so doing will also reduce the risk of environmental mastitis.

Predip solutions must be fast in action. Only preparations with a proven rapid speed of kill (under 30 seconds) will be effective. To get the maximum benefit, clean teats should be coated in predip solution. A minimum contact time of 20–30 seconds is necessary and then the solution must be thoroughly wiped off the teat before the unit is applied. This ensures that no chemical contamination of milk occurs. Many herds have had a marked reduction in clinical mastitis, Bactoscan or TBC and improvement in teat condition when they predip.. Many cows also milk out more completely and faster when they predip as this further maximizes the milk let-down reflex. Predips are discussed in detail on page 126.

There are many herds that do not wish to pre-dip and so, if the cow enters the parlour with visibly clean teats, then dry wiping the quarters with a paper towel should suffice (see Plate 6.10). If the teats are dirty, as shown in Plate 6.11, they must be washed **and dried**. Grossly contaminated teats should be soaked before washing to allow the dirt to soften. This allows easier removal



Plate 6.10. Visibly clean teats can be dry wiped with a paper towel.



Plate 6.11. If the teats are dirty, then they must be washed and dried or predipped and dried.

of soil. Poor washing procedures assist the spread of bacteria rather than removing them. Washing of teats is best carried out using water from drop hoses. Warm water is recommended as it is more comfortable for both the cow and the milker. However, if warm water is used, it is essential that header tanks are kept clean and covered and preferable that a sanitizer is added to the water.

Contaminated water can be a source of

Pseudomonas infections (see page 47). Washing teats in the winter with cold water can reduce the milk let-down reflex. It may also have an adverse affect on teat skin condition. Some people wash the teats and udder with a power hose as the cows enter the parlour. This is not recommended as it must be painful to the cow and soaks the udder as well as the teats.

It is important to ensure that only the teats and not the udder are washed; otherwise, when the milking unit is attached, water runs down the wet udder and collects around the top of the liner, as shown in Plate 6.12. This is commonly referred to as ‘magic water’ because one moment it’s there and the next it’s gone! If sucked in through the top of the liner, at best it contaminates the milk (causing increased Bactoscan or TBC) and at worst causes liner slip, creating impact forces. Impact forces increase the risk of new mastitis infections (see pages 79–80) as there are likely to be high levels of *E. coli* and *Streptococcus uberis* in ‘magic water’. The less water used on teats the better. If



Plate 6.12. When the udder gets wet, water drains down and collects around the top of the liner and the teats. This is commonly referred to as ‘magic water’.



Plate 6.13. Automatic teat-washing sprinklers jet water up against the udder and teats to remove any dirt.

washed, it is **essential** that teats are dried prior to attachment of the cluster.

The addition to the washing water of even low levels of disinfectant, for example, 60 p.p.m. of iodine or 200 p.p.m. of sodium hypochlorite, is beneficial. It helps to keep the warm water and pipelines free from bacterial contamination. It also reduces the number of bacteria on the teat and helps to keep the milker's gloves or hands clean during the milking process.

In hot climates such as the desert areas of the USA and the Middle East, automatic teat washing in sprinkler pens may be used. Here there are two collecting yards before cows enter the parlour. The first yard is fitted with sprinklers at ground level that jet water up against the udder and teats to remove any dirt, as shown in Plate 6.13. The cows are then allowed to stand while the udders and teats drip dry before they enter the second collecting pen, from which they have immediate access to the parlour. Sprinkler pens reduce the amount of washing necessary in the parlour and so speed up milking. However, as they wet both the udder and teats, it is vital that the cows are thoroughly dry before entering the milking parlour. The system can only be used in hot climates and care must be taken that it does not signifi-

cantly increase standing times; otherwise lameness might result.

Washing but not drying teats before milking will increase the bacterial contamination in milk and this will raise Bactoscan or TBC. It also deposits bacteria in suspension on the teat end and in so doing increases the risk of environmental mastitis. Finally, excessively wet teats increase liner slip. In herds where the teats are washed but not dried before milking, the coliform count, which is a measure of the level of environmental contamination of milk, is high (see pages 173–174).

Drying Teats before Milking

Cows should be dried with a single-service paper towel or cloth before the milking units are attached. Under no circumstances should a communal udder cloth, as shown in Plate 6.14, be used, as this only spreads infection from cow to cow. Udder cloths often become so grossly contaminated that they are virtually impossible to disinfect. The only acceptable udder cloth is an individual cloth per cow that is washed, disinfected and dried between milkings. Such a cloth is easy to use and cleans teats well, but



Plate 6.14. Communal udder cloths spread mastitis bacteria from cow to cow.



Plate 6.15. Growth of bacteria from an udder cloth sampled during milking.

the uptake of this practice will depend on the costs of labour and energy.

The risk of spreading infection through the use of a communal udder cloth cannot be underestimated. Many farmers are convinced that because their udder cloths are placed in a bucket of disinfectant solution during milking all the organisms are killed. This is not correct.

Trial work has shown that *Staphylococcus aureus* can survive on udder cloths soaked in disinfectant solutions for 3 minutes. *Streptococcus agalactiae* can survive on cloths for 7 days and can be isolated after soaking for up to 5 hours in a 2% hypochlorite solution. This is much longer than the few minutes they would be soaked in disinfectant solutions during milking.

It is very simple to show how ineffective disinfectants are at killing organisms on udder cloths. This can be demonstrated to farmers who are convinced that use of these cloths poses no risk in spreading infection during milking. Get the milker to squeeze some liquid from an udder cloth onto a blood agar plate during milking. Incubate this for 24 hours in the laboratory. The growth of bacteria shown in Plate 6.15 comes from one such udder cloth. The results speak for themselves.

Paper towels are cheap and disposable and are the ideal choice for drying teats. In some countries old newspapers are used. It

is important that a separate piece of paper towel is used on each cow or else you may smear dirt or infection from the first cow onto the second and so on.

Medicated towels are recommended by some people. These are towels impregnated with a disinfectant and are designed for single use. They are only intended to dry and disinfect clean teats, not to clean and dry dirty teats. If they are used to wash dirty teats, there will be little, if any, benefit over the use of paper towels.

Assessing Teat Preparation

Teat preparation can be assessed by a variety of means. Obviously the cleanliness of the teats can be observed before the unit is attached. The milk sock (see Plate 6.6) can be checked after milking to see how much faecal matter is present. It is important to take into account the number of cows in the herd. A high level of faecal contamination is far more significant in a small herd than a large herd.

Carrying out coliform counts on bulk milk is another useful screening method. Alternatively, teats that have been prepared for milking can be wiped with a white towel to see how much dirt remains present, as shown in Plate 6.16. It is also useful to examine the inside of the liners during milking. If they are dirty, this indicates that teat preparation is suboptimal.



Plate 6.16. Teats can be wiped with a white paper towel after preparation to see how effective teat preparation has been.

Milk Let-down Reflex

Milk is extracted from the udder by applying vacuum to the end of the teat. This liter-

ally ‘sucks’ the milk out. It is important that the teat does not collapse during milking. This is achieved in two ways. First, the venous plexus at the base of the teat (see page 13) becomes engorged with blood, and this makes the teat become erect. Second, there is an increase in pressure within the udder during milking, which causes the teat to become full and turgid, thus making milk removal easier.

Release of the hormone oxytocin results in increased udder pressure. Stimulation of the udder and teats causes the pituitary gland at the base of the brain to secrete oxytocin. Oxytocin acts on the alveolar muscles in the udder, which then squeeze milk into the ducts. This results in a pressure build-up and produces the syndrome called milk let-down.

Oxytocin release occurs through two forms of milk let-down reflexes: ‘conditioned’ and ‘unconditioned’. Conditioned reflexes are those that the cow takes in through her eyes, ears and nose, such as the sound of the vacuum pump and the smell of concentrates. Unconditioned reflexes occur as a result of teat stimulation, such as washing, predipping, foremilk and drying.

The level of the conditioned reflex generally remains constant and so the aim of a good milking routine is to maximize the level of the unconditioned reflex (see Figure 6.2). This is where the benefit of a good and

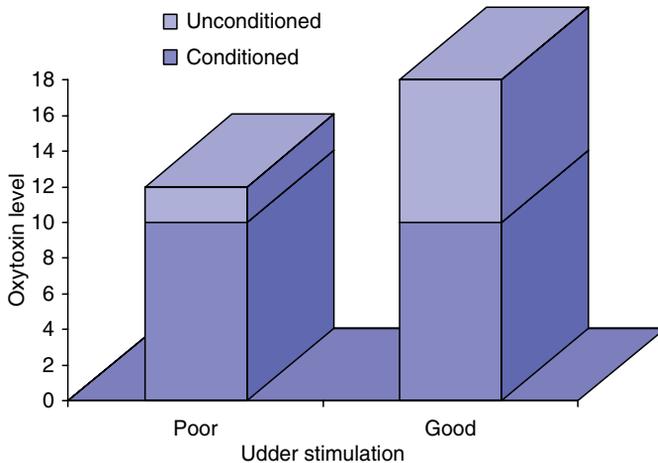


Fig. 6.2. Levels of oxytocin with good and poor udder stimulation.



Plate 6.17. A Lactocorder measures milk flow throughout milking.

consistent milking routine pays dividends.

Farmers report that cows milk out faster following good teat stimulation from foremilk and predipping. They also milk out more fully, resulting in higher milk yields.

The key factor in the speed of milkout is not the level of oxytocin in the blood but rather the timing of the oxytocin release. Studies have shown that teat or udder massage for 30–60 seconds immediately before units are attached results in faster milk flow rates. The oxytocin let-down reflex is relatively short-lived and does not extend

beyond 10 minutes. Cows should therefore have finished milking within 10 minutes of stimulation. Taking full advantage of the milk let-down reflex will result in faster milking and reduced teat damage, and ensure the most complete removal of milk from the udder, thereby improving production.

The effect of a good let-down reflex can be seen using a Lactocorder, a device that measures milk flow rates from individual cows. The Lactocorder is fitted in the long milk tube between the cluster and the milk transfer line or recorder jar (see Plate 6.17), and measures yield against time. Figure 6.3 shows the milking pattern of a cow where there has been no manual stimulation. It can be seen that there is an initial milk flow in the first minute of just over 2 litres per minute. This is the milk from the teats and gland cistern. However, a minute after attachment the flow rate has dropped down to just below a litre per minute and then, once let-down has occurred, it takes 7 minutes to harvest 13.7 litres of milk. This is called **biphasic milk let-down** (i.e. initial milk flow, then flow stops, then peak flow), and it can also be observed by simply watching the bowls in the claw.

Compare Fig. 6.3 with Fig. 6.4 where

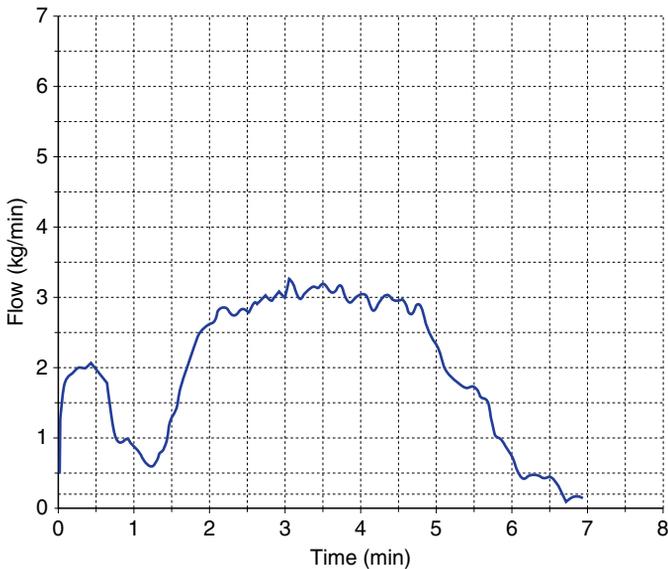


Fig. 6.3. Lactocorder tracing (solid line) showing biphasic milk let-down and overmilking: 13.7 kg milk in 7 minutes.

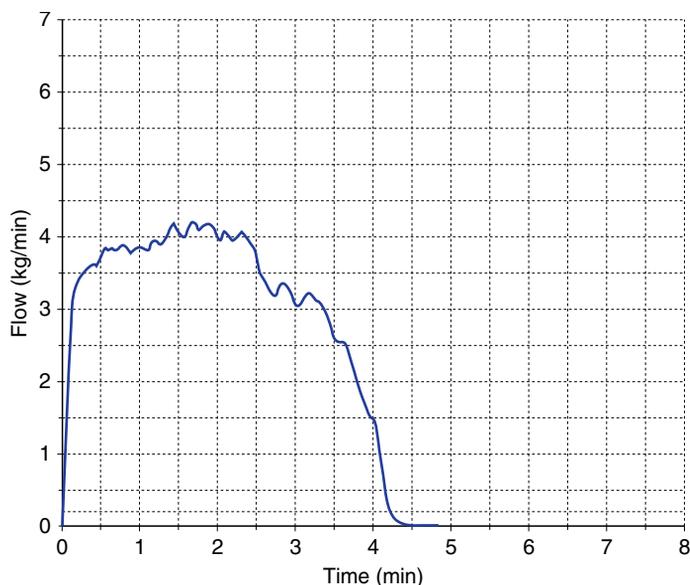


Fig. 6.4. Lactocorder tracing (solid line) showing excellent milk flow and rapid milkout: 14 kg in 4.25 minutes.

the Lactocorder measures a cow that has had 20 seconds of manual stimulation and where the unit was attached within 1–2 minutes after stimulation. This shows that milk flows at a rate of 4 litres per minute once the unit is attached, and this cow milks out in 4 minutes, having given 13.8 litres of milk, a saving of 3 minutes compared with the cow with no manual stimulation.

Batch Preparation

The short time spent stimulating cows with foremilk has a large payback in a reduced milking time. The unit has to be attached at the right time following stimulation and so batch preparation of cows is advised. Batch preparation is where the milker prepares a set number of cows and then attaches the unit within 1–2 minutes so that the cluster is attached to coincide with the oxytocin release.

This means that in a herringbone parlour, cows are prepared in batches so that the clusters are attached within 1–2 minutes after stimulation. An example is a milker

who foremilks and then predips the first cow, and then follows this routine down a batch of four to six cows. He then returns to the first cow, dries the teats and attaches the cluster and repeats this action on the remaining cows (see Fig. 6.5). He then moves on to the next batch of cows.

In a rotary parlour with two milkers preparing the udder and teats, it is easy to have a time delay of 60–90 seconds from stimulation to attachment. In herringbone parlours where there are two milkers, you can use the follower and leader principle, where the leader will predip and strip and the follower dry and attach. Farmers find that milking cows with a set routine and batch preparation speeds up milking.

Unit Attachment

An efficient milker will attach the units without leaking large amounts of air into the system. This helps to make sure that vacuum levels remain stable and reduces the risk of liner slip and impact forces.

Most units should be carefully aligned

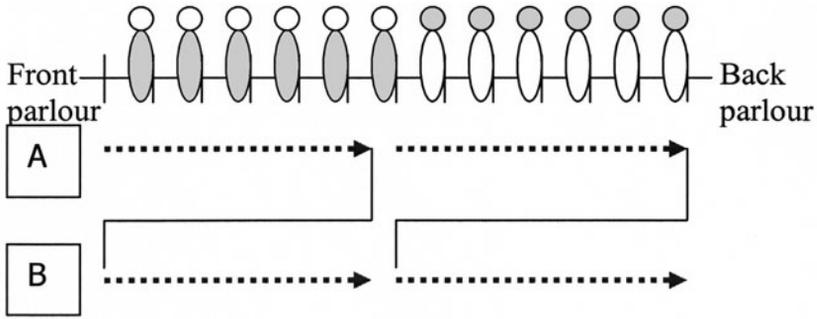


Fig. 6.5. The first task is to predip and strip the batch of cows (A). The milker then walks back and (B) wipes the teats dry and attaches the unit down the batch. He then starts on the second batch.

so that the cluster sits comfortably on the udder without twisting. This ensures that the cow will milk out evenly. Clusters that twist are uncomfortable for the cow, may result in the poor milkout of a quarter and will increase the risk of liner slip and the cow kicking off the unit (see Plate 6.18). They will also increase the risk of air being admitted through the top of the liner.

Units are designed to be attached with the long milk hose extending out through the hind legs or forwards towards the head of



Plate 6.19. A support arm helps to prevent the unit resting on the udder by taking the weight of the long milk tube.



Plate 6.18. Poor unit alignment. Clusters that twist are uncomfortable for the cow and may result in the poor milkout of a quarter and increase liner slip.

the cow. In the latter case, a support bar, as shown in Plate 6.19, is needed to avoid the unit twisting on the udder. Although commonly used in Europe and the USA, support arms are not frequently used in the UK.

On occasion, milkers have been known to place stones or bricks on top of the claw-piece to try to speed up a slow milker, as shown in Plate 6.20. This practice is not to be encouraged as it causes excessive pulling on the teats and this further increases any teat damage already present, and may indirectly slow milking down even further.

Milking the Mastitic Cow

In an ideal world, as soon as a new case of mastitis is detected the cow should be held

Table 6.1. Disinfection of teat cup clusters after removal from cows with mastitis. (From Bramley *et al.*, 1981.)

Treatment	Time	No. tested	% Clusters positive after cleaning	No. <i>S. aureus</i> /ml recovered per cluster
Cold water flush	5 s	19	100	100,000–800,000
Circulation of cold hypochlorite (300 p.p.m.)	3 min	19	100	50–2,000
Circulation of water @ 66°C	3 min	18	22	0–80
Circulation of water @ 74°C	3 min	85	0	0
Circulation of water @ 85°C	5 s	530	3	0–15

back and milked last. Unfortunately, this does not always happen as it can be time-consuming and there may be no facilities for separating and holding back individual animals. Milking infected cows last, or even better, in a separate group, eliminates the risk of mastitic or antibiotic-containing milk entering the bulk tank. (Don't forget to remove the milk line from the bulk tank first.) It also reduces the spread of mastitis to the rest of the herd via the milker's hands

and contaminated liners, and allows the milker more time to treat cows properly without slowing down the milking process. Remember that liner condition is also significant, as worn, cracked liners will retain more bacteria than smooth liners.

It is important to disinfect the cluster after milking mastitic cows. This will reduce the risk of transferring infection to other animals. Many milkers dip the units in a disinfectant solution for a few seconds. While this reduces the number of bacteria present, it does not kill all the organisms nor does it fully eliminate the possibility of spread.

Table 6.1 compares the efficiency of different methods of disinfecting clusters. It can be seen that after cold water flush for 5 seconds many bacteria were still present. Unfortunately, the data do not give the initial number of bacteria present. You need to circulate water at 74°C for 3 minutes in order to disinfect the cluster, but this is impractical during milking. The only effective method to sterilize the cluster during milking is to flush it with the water at 85°C for 5 seconds. Most farmers disinfect the cluster by immersing it in a solution of hypochlorite, iodine or peracetic acid solution for several minutes. While this does not sterilize the unit completely, it does remove the majority of the infection, minimizing the risk of cross-contamination.

In some of the dairy herds in the hot or desert areas of the world, where highly contagious *Mycoplasma* mastitis poses a real threat to udder health, back-flushing units are fitted to parlours. These disinfect the milking unit by passing water at 85°C



Plate 6.20. Milkers have been known to place stones or bricks on top of the clawpiece to try and speed up a slow milker. This is not recommended.



Plate 6.21. Back-flushing units with disinfectant solution will help to reduce the spread of infection from cow to cow.

through the liners, as shown in Plate 6.21. These units are very expensive and, provided good mastitis control measures are used, less benefit would be gained from them under temperate conditions, where *Mycoplasma* infections are rarely encountered.

The simplest way to reduce the spread of infection is to have a separate cluster for milking mastitic cows (see Plate 6.22). This means that they can be milked as and when they enter the parlour. This cluster can then be adequately disinfected between uses without slowing down the milking process (see Plate 6.23). Clusters can be disinfected in a solution of peracetic acid, hypochlorite or another suitable disinfectant. Many farmers have a separate cluster connected to a 'dump bucket'. By collecting mastitic milk separately, there is no risk of antibiotic contamination.

The dump bucket used on mastitic cows is also often used to collect colostrum from fresh calvers. Freshly calved cows are very susceptible to infection, as their resistance to disease is low at this time. If the cluster is not disinfected between uses, this may act as a source of new mastitis infections (see the contaminated liner in Plate 6.24). It is essential that these units are not neglected, that liners are changed frequently and that they are thoroughly cleaned after each milking.

Many farmers still milk mastitic cows into recorder jars and then drain this milk into a bucket or on to the floor. There are several dangers in doing this. The milker may

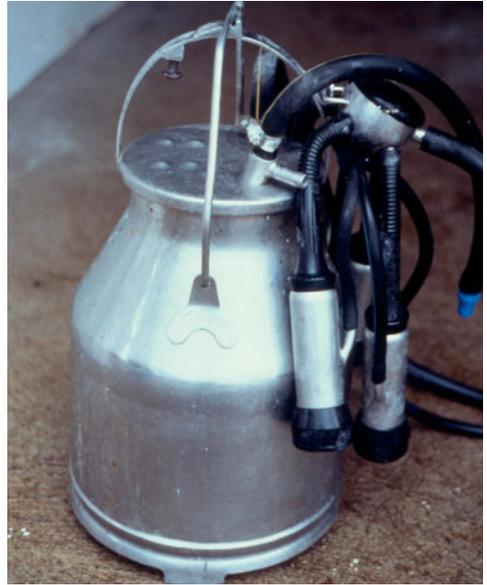


Plate 6.22. A separate cluster connected to a dump bucket will reduce the risk of spreading infection to healthy cows and eliminate the risk of antibiotic residues entering the bulk supply.

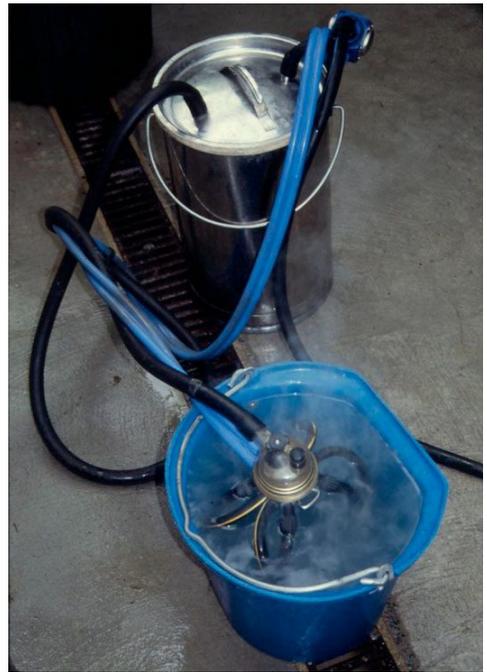


Plate 6.23. After milking a mastitic cow through the dump bucket, it is essential that the cluster is disinfected before it is attached to the next cow.



Plate 6.24. The liner of a dump bucket that has not been cleaned correctly. You can see the crud and the deposits inside the liner.

forget to dump the milk, so antibiotic residues contaminate the bulk supply. The valve at the bottom of the recorder jar may be faulty and milk may leak past the valve and into the bulk tank. Finally, antibiotics concentrate in butterfat and so jars that are not rinsed out thoroughly after milk is released may still result in antibiotic contamination from butterfat rings around the inside of the jar. Antibiotic residues are discussed in Chapter 15.

Unit Removal

Once cows are milked out, the vacuum supply to the cluster should be shut off. Atmospheric air enters the clawpiece through the air bleed hole, releasing the vacuum and allowing the unit to ‘fall off’ the udder. Where ACRs (automatic cluster removers) are fitted, they should operate in the same way. A cluster removed while still under vacuum can result in large impact forces and may cause teat sphincter damage.

There are three important adjustments on the ACR, namely:

- The milk flow rate, which will eventually trigger ACR activation.

- A delay between reaching this flow rate and vacuum shut off.
- A further delay between vacuum shut-off and ACR pull.

First, milk flow rates triggering ACR removal were traditionally set at 200 ml/min, but more recently this has been increased to 400 to 600 ml/min or 600 to 800 ml/min for high-yielding cows milked three times a day. Second, there has to be a delay between reaching this ‘trigger value’ and vacuum shut-off; otherwise a cow that has had a temporary drop in flow rate, e.g. from liner slip, will have the unit removed early.

Third, the delay between vacuum shut-off and ACR pull allows time for vacuum levels in the claw to vent, thereby reducing the risk of teat-end damage. If a significant number of cows kick at unit removal, it is likely either that the milk flow trigger is too low or that there is insufficient delay between vacuum shut-off and ACR pull.

Overmilking is not to be encouraged as it increases the unit time on, slows down milking and increases the risk of teat-end damage, clinical mastitis and high cell count.

Disinfection of Clusters between Cows

At the end of milking, a small amount of milk, about 2–4 ml, is held inside the mouthpiece of the liner. When the cluster is attached to the next cow, the milk from the previous cow will run down the inside of the liner and contaminate the teat of the next cow to be milked. This represents a risk of transfer of infection.

At one stage, dipping clusters into a bucket of disinfectant solution between cows was a popular procedure in the milking routine. It was generally believed that dipping units in a solution of hypochlorite for a few seconds would remove all bacteria from contaminated liners. The difficulty of completely disinfecting clusters has been shown in Table 6.1. Although cluster dipping will help to reduce bacteria numbers, it is unlikely to be totally effective in eliminating the spread of bacteria from cow to cow.



Plate 6.25. The flush solution is emitted from a small nozzle inside the mouth of the liner to minimize cross-contamination.

The MFE trial work in the late 1960s (page 1; Fig. 6.1) that showed the effect of pasteurizing clusters on clinical and sub-clinical infections was marginal, and so it was not incorporated into the five-point-plan that was recommended. However, it must be remembered that at this time herd size was small, yields were low, environmental mastitis was not a significant problem, and the major problem was subclinical mastitis.

Dairy herds have changed significantly since then. Back-flushing units will help to remove the both environmental and contagious bacteria and this can only be of benefit, especially in herds where environmental management or teat preparation is suboptimal.

A variety of cluster flush systems are in use. In one, the flush solution is emitted from a small nozzle inside the mouth of the liner (see Plate 6.25), while others incorporate pulses of air and a sanitized flush solution that enters the long milk tube and flushes through the cluster and out through the liners.

While there are ample data to show that this reduces the level of all bacteria present

in the liner (staphylococci, streptococci, coliforms, etc.), so far there are no good trial data to demonstrate a positive benefit in terms of cell count or reduced mastitis incidence. Liners are always flushed between cows on robotic systems.

Residual Milk

No matter how long you leave the milking unit on the cow, not all milk will be removed from the udder. The remaining milk is called residual milk. The amount of residual milk is usually 0.5 litres for heifers and 0.75 litres in cows.



Plate 6.26. Machine stripping is the process whereby a downward pressure is applied to the clawpiece with one hand, while the quarters are massaged with the other. This is not recommended.

There are a variety of factors that can increase the amount of residual milk:

- Disturbing or frightening cows just before or during milking, which will affect the let-down reflex.
- Delay between udder stimulation and attaching the teat cups.
- Irregular milking intervals.
- Teat injuries.
- Poor unit alignment, leading to incomplete milkout in one or more quarters.
- Poor ACR adjustment, leading to early cluster removal.
- Milking through a defective parlour.
- Liner 'creep', leading to poor milkout.

A good milking routine will help to keep the amount of residual milk to a minimum, thereby maximizing yield. If a large amount of milk remains in the udder, this may aggravate subclinical mastitis, especially with *Streptococcus agalactiae* infections. It can also decrease milk yield.



Plate 6.27. Immediately after the cluster has been removed following each milking, the entire surface of each teat should be coated in a disinfectant solution. Note the brown stain left by the iodine in the dip.

Machine Stripping

Machine stripping is the process whereby downward pressure is applied to the claw-piece with one hand, while the quarters are massaged with the other, as shown in Plate 6.26. The intention is to maximize milk removal and reduce the amount of residual milk in the udder.

There is a danger that, if machine stripping is carried out with great vigour and enthusiasm, air will be sucked in through the top of the liner, resulting in impact forces. Impact forces may cause a massive reverse flow of milk against the teat end (see pages 79–80). If bacteria penetrate the teat canal, a new infection may become established. For these reasons, machine stripping is not recommended.

Today's dairy cow is selected for good udder and teat conformation. In addition, the ability of modern milking machines to reduce the amount of residual milk has greatly improved, due to modifications to their design.

Postmilking Teat Disinfection

Immediately after the cluster is removed the entire surface of each teat should be coated in a disinfectant solution, as shown in Plate 6.27. This can be applied as a dip or as a spray.

The aim of postmilking teat disinfection is to kill any bacteria transferred on to the teat during milking before they have a chance to colonize or infiltrate the teat canal. Postmilking teat dipping is an essential method of controlling contagious mastitis. It is less effective against coliform and other environmental forms of mastitis (for which predipping is more important). Teat dipping is discussed in detail in Chapter 7.

After cows leave the parlour, they should exit along clean and freshly scraped passages and move towards freshly bedded housing. They should have access to food and water to encourage them to remain standing for between 20 and 30 minutes while the teat canal closes fully. If cows lie down immediately after milking while the teat canal is open, there is a risk that environmental bacteria may penetrate the udder, and mastitis may result.

In some herds, cows are kept standing in a passageway for prolonged times after milking. This can have adverse effects on lameness. A lame or sick cow should be able to lie down after milking if she so chooses. Cows naturally feed after milking, allowing time for the teat canal to close; they should then be able to lie down.

Milking Order

If a herd is divided into groups, then milking order should be considered. Many farmers group their cows, and the types of group will depend on the management. To help reduce the spread of mastitis cows should be milked in the following order:

- High yielders.
- Low yielders.
- High cell count cows.
- Mastitic, lame and other treated cows.

Late lactation cows are likely to have an increased amount of subclinical infection due to longer exposure to mastitis organisms throughout lactation. Therefore, the greatest risk of contagious mastitis will come from this group of cows.

A high cell count group can help limit the spread of infection within a herd, but most herds that have cell counts under control do not need such a group.

Frequency of Milking

There is a higher yield when the milking frequency is increased to three or four times a day, without significant effect on milk composition. At three times a day, yields can be increased by up to 15% in heifers and 10% in cows. This is due to the removal of the milk inhibitor protein, resulting in more milk being synthesized in the udder.

In addition, as milk is removed from the udder more frequently, flushing out any bacteria in the teat canal and udder (and despite a possible increased risk of new infections from extra milking), the mastitis incidence in herds that are milked three times a day tends to be lower than in their twice daily milked counterparts. Herds that are milked three times daily also have lower cell counts due to this flushing action.

Robotic Milking

Robotic milking has become more popular, with large numbers of robots on the continent and in the USA. The robot milks the cow in a very different way from what happens in a conventional parlour, as both teat preparation and mastitis detection are totally automated. Once an individual quarter is milked out, the liner on that quarter is removed. This minimizes overmilking on individual quarters. Disinfection of the liner between every cow is automated, along with postmilking teat disinfection. There are significant advantages to this technology, provided that it works and that the management and facilities of the farm suit robotic milking.

However, there are some potential problems. Foremilk is not visually examined and mastitis is detected from electrical conductivity. Any change in conductivity results in this milk being discarded and a warning message about that cow. She must be checked to decide whether she has clinical mastitis. Most robots allow farmers to adjust conductivity thresholds so as to reduce the number of false alarms relating to mastitis. The new robots compare individual quarter conductivity with results from previous milkings and look for changes to improve the accuracy of mastitis detection. This can be very useful for the early detection of mastitis.

Teats are cleaned using rotary brushes (see Plate 6.28), which use a combination of disinfectant solution and physical action to clean. Teats are not dried, which is a disadvantage. If teats are grossly contaminated before milking, then teat preparation may not be adequate, and the wet teats can contain large amounts of environmental bacteria. This will increase the risk of environmental mastitis and/or raise the TBC or Bactoscan.

Postmilking teat disinfection is usually from a central spray nozzle, which can only coat the inner surfaces of the teats. Outer surfaces may not be fully disinfected.

Not all cows may enter regularly to be milked. This can result in intermittent milking, and this may increase cell counts of



Plate 6.28. Robotic milking machines use rotating brushes to clean teats. Teats are not dried before the unit is attached.

such animals. It is important that cows are milked at the correct frequency. This may involve some form of feeding to encourage cows to enter the parlour.

Summary of the Milking Procedure

The aim of the milking routine is to milk clean, dry teats with a correctly functioning milking machine as efficiently as possible, thus posing minimal risk to udder health while maintaining milk quality. This is achieved by the following routine:

- Foremilk cows.
- Carry out teat preparation so that teats are clean and dry. Predipping is the gold standard. Predip, allow a contact time of 30 seconds and wipe dry.
- Attach the milking unit within 1–2 minutes of teat preparation.
- Check machine alignment so that it sits squarely on the udder.
- When the cow is milked out, shut off the vacuum and then remove the cluster.
- Coat teats with teat dip.
- Allow the cow to exit to a sheltered yard with access to food and water so that she is encouraged to remain standing for 20–30 minutes.

The additional time spent diligently carrying out these procedures will be rewarded by a lower incidence of mastitis, lower cell counts, cleaner milk production, increased milk yield, increased satisfaction for the milker and greater comfort for the cow.

7 Teat Disinfection

Postdipping	116
Predipping	117
Method of Application: Dip or Spray	117
Dipping	117
Spraying	117
Preparation and Storage of Dips	119
Chemicals Used in Post- and Predipping Disinfectants	120
Iodophors	120
Chlorhexidine	120
Quaternary ammonium compounds (QACs)	120
Dodecyl benzene sulfonic acid (DDBSA)	121
Hypochlorite	121
Acidified sodium chlorite	121
Foam dips	121
Viscosity and surfactant	122
Barrier dips	122
Postmilking Teat Disinfection	123
Removal of mastitis bacteria	123
Removal of bacteria from teat sores	124
Improving skin quality with dip additives	124
Automatic teat disinfection systems	124
Limitations of postmilking teat disinfection	125
Seasonal use of dips	126
Premilking Teat Disinfection	126
When does predipping not work?	127
Iodine Residues	128

This chapter examines the reasons for teat disinfection, the methods of application (dip or spray), the chemicals used, some of the associated management faults and the importance of chemical residues. Teat disinfection can be carried out immediately before milking (predipping) or immediately after (postdipping).

Postdipping

In postdipping (see also page 113), the disinfectant is applied as soon as the milking unit is removed. Teats must not be wiped dry after the postdip has been applied. Postmilking teat disinfection is one of the most important preventive measures in

mastitis control and an integral part of the 'five-point plan' (see page 1). It should be carried out in every herd, after every milking, throughout the year.

Predipping

In predipping (see also pages 101–102), the disinfectant is applied to the teats just before milking, and teats must be wiped before the cluster is attached. Predipping is a newer concept, aimed at reducing the incidence of environmental mastitis and reducing the TBC/Bactoscan (see Chapter 10). In most instances the disinfectants used for postdips differ from those used as predips, as different speeds of kill are required.

Method of Application: Dip or Spray

Because of the importance of removing bacteria from the entire teat (see page 21), it is essential that the whole teat, and not just the teat end, is disinfected. This is likely to be best achieved by dipping, although spraying can be effective if carried out conscientiously.

Dipping

Dipping uses less product than spraying (approximately 10 ml per cow per milking for dipping versus 15 ml for spraying) and, provided that it is carried out correctly, will provide excellent teat cover. The teat dip pot should be large enough to contain the teat without excessive spillage of dip and, at the same time, it should be full enough to ensure that small teats will reach and be covered by the dip solution (see Fig. 7.1).

Dual compartment anti-spill cups (pots) are also available (Fig. 7.2). When the bottom compartment is squeezed, dip is forced into the top. If the pot should then be knocked over (or kicked out of the milker's hand), it is only the dip in the top of the pot that is spilled. These cups often have a hook on the

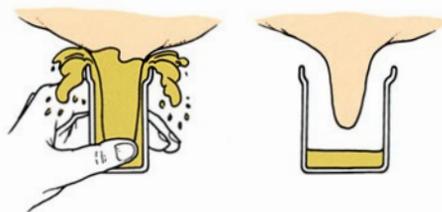


Fig. 7.1. (Left) A small, narrow teat dip cup that is overfull will cause the iodine to be wasted and the milker's hands to become stained, whereas an excessively wide cup with only a small quantity of teat dip (right) may mean that small teats are not adequately dipped.

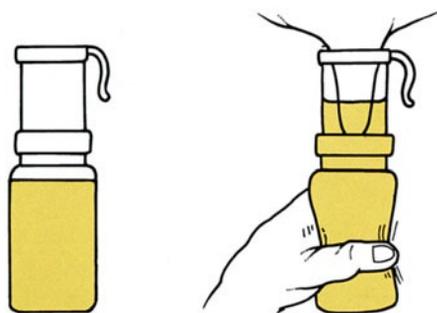


Fig. 7.2. Anti-spill teat dip cups are available. Only the dip in the top chamber is spilled if the cup is knocked over.

side, allowing them to be attached to the milker's belt (Plate 7.1) and therefore readily available for use. Whatever type of pot is chosen, it should be applied so that the rim makes contact with the udder, and the pot is then shaken to ensure total teat cover.

Teat dip pots should be cleaned regularly, to prevent contamination. Any dip remaining in the pot at the end of milking should be discarded and the pot cleaned before reuse at the next milking. If pots are hung in the parlour during milking, take care that they do not become contaminated, as in Plate 7.2.

Spraying

Teat spraying can also be effective, but must be carried out conscientiously. It is much



Fig. 7.3. Spray applied from the side using spray lances achieves only partial cover. Spray should be applied from the bottom of the teat in a circular motion to ensure total coverage.

Plate 7.1. Dip pots for predip and postdip are conveniently attached to the belt during milking.



Plate 7.2. Teat dip pots should be stored in the parlour in such a way that they do not become contaminated.

easier to achieve only partial cover than with dipping. Spray lances should be of reasonable length, with the nozzle pointing upwards and not directly out from the end (Fig. 7.3). Spray should be applied from beneath the teats while rotating the lance in a circular action below the base of the udder. At least two rotations will be needed to achieve full cover, one to the left to cover the left side of teats, and one to the right to cover the right side. A single rotation is simply not sufficient.

In herringbone parlours, some milkers open the gate, releasing the cows, and then try to apply teat spray as they walk past. Unfortunately, this results in very poor teat cover. If iodine disinfectants are used, it is easy to see when only one side of the teat has been coated, as in Plate 7.3. The disinfectant may well run to the end of the teat, thus eliminating teat-end colonies and reducing the most important aspect of mastitis transmission. However, the absence of disinfectant on one side of the teat could allow the establishment of a reservoir of mastitis pathogens on the untreated teat skin. You should also regularly check the spray lance. The two most common faults found are:

1. The nozzle becomes partially blocked, so that the spray is emitted from one side of the lance only.



Plate 7.3. Poorly applied spray leads to only one side of the teats being covered.

2. The nozzle emits teat disinfectant as a jet rather than a spray, and this results in poor teat cover.

These faults can be checked by spraying disinfectant on to a piece of white paper towel. The pattern on the towel is the emission pattern of the spray lance. Hand-held garden sprayers have been used, but most do not provide a sufficiently fine aerosol or wide enough spray angle to be effective.

The best method of checking teat cover is to examine teats immediately after disinfectant application, and preferably when the milker is not aware that this is being done, e.g. during a teat scoring visit. A lamp is needed for this, as shown in the section on teat scoring in Chapter 14 (see pages 233–234). In this system a ‘missed

cow’ is defined as a cow where the surface of one or more teats is less than half covered with disinfectant, i.e. quite a severe score. A good herd will achieve only 5% of cows ‘missed’. In poor herds, some 90 to 95% of cows may be ‘missed’. This clearly has a major effect on the spread of contagious pathogens.

A comparison between teat dipping and spraying is shown in Table 7.1.

Preparation and Storage of Dips

Some dips are bought ready to use, while others are supplied as concentrates and have to be diluted. Ready-to-use products are often more stable, as they have been carefully formulated and diluted with soft water. When diluting a concentrate, the instructions should be followed closely and, ideally, only enough solution for a few days should be made up to avoid deterioration. Hard water or bore hole water may not be suitable.

Unused containers of dip should be stored away from cold areas, since freezing may lead to separation of water from the chemical. When in use, make sure that the top of the drum is not left open in an area where large quantities of water are splashed. Water contamination will dilute the teat dip or, even worse, if contaminated by circulation cleaner rinse water, the dip may become denatured and ineffective.

Table 7.1. Comparison between dipping and spraying.

	Dipping	Spraying
Teat cover	Generally good	Good if careful
Volume used per cow/milking	10 ml	15 ml
Cost	Very cheap equipment	More expensive to install
Points to watch	Dirty teat dip cups Keep pot full Cows with very short or long teats	Blocked nozzles causing slow flow rates Solution running out during milking

Chemicals Used in Post- and Predipping Disinfectants

There is a range of chemicals used for both teat dips and sprays. It is not possible to be specific concerning the most effective disinfectant, because their properties vary. In the UK, some products have a Medicines Licence, i.e. the manufacturer has carried out field trials to show that the disinfectant is safe and is effective in the control of mastitis. Other products are simply sold unlicensed as a postmilking teat rinse, making no claims regarding mastitis control. Licensed products are clearly the safest option. The best dip is the one that fully covers every teat at every milking.

The most common products used are listed below.

Iodophors

These are probably the most widely used compounds in dips. They consist of 0.25–0.5% total iodine in association with a complexing agent, which essentially acts as a ‘reservoir’ of free inert iodine. As the free iodine is slowly used up by reacting with bacteria, more free iodine is released from the complexing agent reservoir to maintain a constant level of active ingredient in postdips of around 3–5 p.p.m. The problem of iodine residues is discussed on page 128, and a low-iodine residue dip described.

Iodophors have a low water solubility and require surfactants to bring them into solution. These are strong acids and this can be irritating to teat skin, hence most products contain significant levels of emollients. Like other teat dips, iodophors are not selective in their action. They have quite a rapid action, although like most other teat disinfectants, they react with any organic matter and so, if teats are badly soiled or heavily coated with milk or if the teat dip cup becomes contaminated with faecal material, then efficacy is markedly reduced.

One advantage of iodine is its colour. It stains skin and so it is easy to see how well teats have been covered after milking (though any stain left on the herdsman’s hands may not be appreciated). Iodine dip that has been excessively diluted looks ‘pale’ in the pot but can still cause staining. Some milkers dislike the smell, and inhalation of the fumes produced may cause unacceptable respiratory irritation, especially when teat spraying.

Chlorhexidine

Commonly used at between 0.4 and 0.8%, chlorhexidine has a wide activity against most bacteria and, because of its greater persistency on the teat, it is especially effective against staphylococci, which is why it is commonly used as a teat disinfectant for goats. It is less affected by organic material than most other disinfectants, although it is relatively colourless, making it less easy to see that total teat cover has been achieved. It is water-soluble so needs very little surfactant, and non-irritant, so products may be sold with only a low level of emollient. However, emollient may be added to improve teat skin condition.

Quaternary ammonium compounds (QACs)

These teat dips consist of the quaternary ammonium compound (the bacteria-killing component), a ‘wetting agent’ to assist in greater penetration of skin and dirt, pH buffers to stabilize the acidity of the product, emollients and water. Colouring agents may be added to show that the teats have been dipped, and thickening agents may give increased persistence on teat skin. Quaternary ammonium compounds are not irritant to teat skin, although careful formulation is necessary to maintain efficacy. Effectiveness against *Pseudomonas* and *Nocardia* is very doubtful and these bacteria have even been known to grow in QAC solutions.

Dodecyl benzene sulfonic acid (DDBSA)

Used at a rate 2.0% inclusion rate, DDBSA dips are non-irritant to teats and to the operator. They have a wide range of activity against most bacteria but are ineffective against bacterial spores. They have a longer length of action than some dips (and hence may confer some protection against coliforms) and work quite well in the presence of organic matter.

Hypochlorite

Hypochlorite is by far the cheapest product available, and is quite rapid in action. Its main disadvantage is that it rapidly reacts with organic material (milk, faeces and skin debris) and becomes ineffective. Used at the usual concentration of 4.0%, it can also irritate the milker's hands, cause damage and bleaching of clothing and result in quite marked drying of teats, especially when first used. These effects are partly caused by the inclusion of sodium hydroxide (around 0.05%), which is sometimes used to stabilize the product. Hypochlorite cannot be safely used as a teat spray, however, as inhalation illness may result. It is colourless and hence it is difficult to assess the efficacy of teat cover.

Ideally, hypochlorite should be introduced at a low concentration and then slowly built up to 4.0% (40,000 p.p.m.). Provided that weather conditions are favourable, teat skin often adapts well and the product can be used without severe reaction. There are anecdotal reports that its strong oxidizing action improves the rate of healing of teat-end damage (e.g. black spot, see page 229) and of viral skin lesions, such as pseudocowpox (see pages 227–228).

Due to formulation problems, if emollients are used they must be added immediately before to milking. Hypochlorite solutions are relatively unstable. They should be stored under cool conditions and with the lid closed; otherwise they can evaporate quite quickly and lose their potency.

There are hypochlorite derivatives available, for example, 5 g per litre

sodium dichloroisocyanurate, which are more stable and have a less severe skin-drying effect.

Acidified sodium chlorite

Combinations of sodium chlorite with lactic or mandelic acid form the antimicrobial compounds chlorous acid and chlorine dioxide, which are effective against most bacteria, yeasts and moulds. Acidified sodium chlorite compounds are two-part products, an activator and a base that are mixed immediately before use, as are added emollients and humectants. The final mix contains around 0.3% sodium chlorite, and barrier films can be incorporated.

Foam dips

Foams, for both pre- and postdips, are popular on some farms. The foam may be produced in a cup attached to a low pressure air line, or it may result simply by squeezing the base of a specially designed cup (Plate 7.4).

Foams are easier to apply than standard liquid dips, some of which are surprisingly difficult to repeatedly force into the upper compartment, and as a consequence may make your forearm ache. However, foam is, by definition, a liquid with air holes in it, so, although foams may appear to give good teat cover, the amount of chemical applied to the teat skin may be low.



Plate 7.4. A foam teat dip.

Viscosity and surfactant

Teat dips vary considerably in their viscosity and surfactant properties. Surfactant promotes the ability of the dip to penetrate cracks and crevices in teat skin, thereby removing teat skin bacteria, and viscosity influences the ability of the dip to remain on the teat after application. Plate 7.5 shows the result of using a cheaper low-viscosity dip – most of the product is on the floor under the cow.

Barrier dips

Barrier dips are only used postmilking. They are more expensive and thicker than conventional products, drip less and consist of a disinfectant, a gel and an alcohol – often isopropanol – which promotes rapid drying. As the dip dries, it leaves a plastic film

covering the teat end. Barrier dips are promoted on the basis that they last longer and hence provide protection against infection by environmental organisms between milkings. The residual plastic film may have to be removed when preparing teats premilking, and some barrier dips specify that predip must be used to remove residual barrier film at the next milking. Because of their high viscosity, it is possible to rapidly immerse a teat in barrier dip and withdraw the pot with no dip left on the teat. Barrier dips therefore take slightly longer to apply. With their thick film, their presence on the teat is easily seen (Plate 7.6), and this perhaps encourages the herdsman to take extra care with application.

Although they give a striking colour film over the teat, there is no evidence that barrier dips are any more effective than conventional products. Anecdotal reports of reduced mastitis could be due to more diligent application or to the need to use predip to remove the barrier prior to the next milking. There is concern by some that the very high viscosity of barrier dips may prevent their penetration into cracks and crevices in teat skin, and as such they may be less effective against teat skin organisms such as staphylococci. This is especially the case if there is a delay between unit take-off and application of the dip, leading to shortening of the teat canal. Aqueous dips might penetrate better because of the



Plate 7.5. Low-viscosity dips may run off the teats and on to the floor.



Plate 7.6. Barrier dips produce a striking film over the teat, but the evidence that they are consistently more effective is lacking.

hydrostatic pressure applied when the teat is immersed in the dip cup.

External teat sealants for dry cows are discussed on page 212.

Postmilking Teat Disinfection

There are three major reasons for carrying out postmilking teat disinfection, namely:

- Removal of mastitis bacteria from the teat skin.
- Removal of bacteria from teat sores.
- Improvement of teat skin quality.

Removal of mastitis bacteria

During the milking process, contagious mastitis pathogens can be spread from cow to cow by three vectors. These are:

- Hands.
- Cloths.
- Liners.

Control of transfer via hands is achieved by wearing gloves and rinsing hands regularly. Control of spread by cloths is by ensuring that communal cloths are not used. It is generally considered acceptable to use a separate cloth for each cow, but even then infection may have been spread from teat to teat of the same cow. Control of spread by the liner is by means of cluster flushing and this is discussed in Chapter 6. Despite these measures, it is still likely that there will have been some transfer of infection from cow to cow.

The liner represents the greatest risk because 2–4 ml of milk remains inside the lip of the mouth of the liner after unit take-off. Unless flushed, this milk will run down the inside of the liner (Plate 7.7) and will contaminate the teats of the next cow to be milked. This is why it is important to disinfect the **whole** of the teat and not just the teat end.

In addition to infection from other cows, there may be mastitis pathogens at the teat end that have arisen from the teat skin of the same cow. There is a risk that the drip of milk present in the teat canal at the end of

milking (Plate 7.8) will contain mastitis organisms, and these could potentially lead to new infections.

Unless removed, these bacteria multiply to form colonies and slowly penetrate the teat canal. It is the adhesive properties of the contagious mastitis organisms (see page 36) that allow them to do this. Once they have reached the udder, a new infection may be established.

Postmilking teat disinfection removes bacteria deposited during the milking process and, as such, it is an extremely important control measure against



Plate 7.7. Milk residues in the liner from the previous cow, which will run on to the teat of the next cow to be milked.



Plate 7.8. The drip of milk seen in the teat canal after milking represents a risk of establishing a new infection and needs to be removed by diligent postmilking teat disinfection.

contagious mastitis. The disinfectant should be applied as soon as the cluster is removed. At this stage, the canal is still open and so some disinfectant can penetrate the teat orifice. This ensures that those mastitis bacteria that have started to enter the canal will also be killed.

Removal of bacteria from teat sores

Any skin lesion that is infected will be slow to heal. Teat disinfection removes bacteria from the skin surface. This promotes healing and maintains teat skin in optimum condition. Rough, cracked or chapped teat skin, as in Plate 3.1, can be a reservoir for organisms such as *Staphylococcus aureus*, CNS (coagulase-negative staphylococci) or *Streptococcus dysgalactiae*. Thorough disinfection of the whole teat is important to ensure that all bacteria are killed.

Improving skin quality with dip additives

Teat skin has relatively few sebaceous glands, and so continual washing followed by exposure of damp teats to a cold and windy environment can remove protective fatty acids and lead to cracking. The most common additives to teat dips are:

- Emollients: they form a seal around the skin to prevent further water loss by evaporation. Similar products are used in udder creams.
- Humectants: these assist in drawing water into the skin.

Lanolin (emollient) and glycerine (humectant) are the most common additives and may be included at up to 10% concentration in the dip. As the level of additive increases, the proportion of disinfectant and hence the bacterial killing ability of the final product decrease (see Fig. 7.4). For this reason, additives are rarely included above the 10% level. If more additive than this is used, then the product may also become too thick and impossible to dispense through a spray line.

Automatic teat disinfection systems

Automatic teat disinfection systems, sited at the exit to the milking parlour, are available. The majority are activated by an electronic 'eye'. As the cow walks past, the 'eye' triggers a burst of disinfectant spray from a nozzle or a raised bar on the floor (Plate 7.9) and directs it on to the udder.

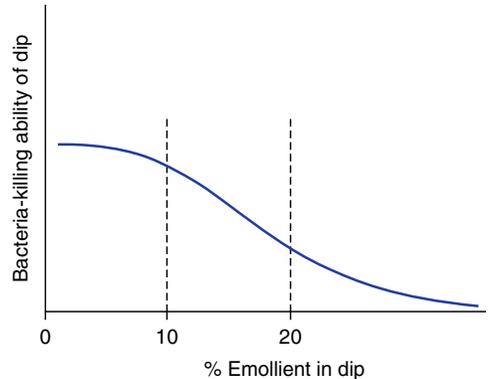


Fig. 7.4. Emollient levels above 10% significantly depress the bacteria-killing ability of the dip.



Plate 7.9. Automatic teat disinfection system sited at the exit of the parlour.

Automatic systems are being continually improved, but as yet none are as effective as thorough teat dipping. Their main disadvantages are as follows:

- Some cows rush past and may only receive a small amount of spray or may be missed altogether. This is partially overcome by trapping each cow in a head yoke as she passes along the spray race.
- Cows with very high udders will be sprayed on the inside of the thighs but full teat cover will not be achieved, whereas cows with low udders will get spray on the inside of the teats only.
- Faeces deposited on the spray jetter by one cow could be sprayed onto the following cow.
- Some spray systems have a jetter bar that can make contact with (and contaminate) the teats of cows with very pendulous udders.
- If sited outside the parlour, the disinfectant spray may be deflected away from the teats during windy weather.
- As most systems are sited at the parlour exit, they apply the teat disinfectant when the cows are leaving the parlour. This could be some time after the unit has been removed, when the teat canal has already started to close. Because of this, high cell counts associated with organisms such as *Corynebacterium bovis* have been reported with automatic teat disinfection.
- The system could run out of teat disinfectant without the operator knowing. Alarm devices should be fitted.

Automated dipping can also be applied from inside the liner (Plate 6.25). Whilst highly labour efficient, the system needs careful monitoring of teat dip cover.

Limitations of postmilking teat disinfection

Although a vital part of every mastitis control programme, postmilking teat disinfection has some limitations:

- It has no effect on existing infections – if teat dipping is introduced into a herd already heavily infected with contagious organisms, you cannot expect a rapid reduction in cell count or mastitis incidence. Although dipping prevents the transfer of bacteria and hence reduces the rate of **new** infections, it has no effect on **existing** infections. For example, in one trial over a 12-month period, a 50% reduction in new infections produced only a 14% reduction in the overall number of quarters infected.
- A herd with a high cell count that starts postdipping should not expect a rapid reduction in cell count. High cell counts will only decrease with treatment, dry cow therapy and culling.
- Its main effect is against contagious organisms – environmental infections are thought to be transferred on to the teat end between milkings and propelled through the teat canal by impacts during the milking (see pages 79–80), or they are the result of dry period infections (see pages 50–52). As postmilking disinfectants have a relatively short action e.g. 1–2 hours after application, they will have a limited effect against environmental mastitis. Premilking disinfection is therefore more important in the control of environmental mastitis.
- It may cause teat irritation – this is particularly the case during wet and cold weather. Some chemicals are quite irritant, although their adverse effects can be reduced or avoided by the inclusion of emollients. Under sub-zero conditions, some farmers discontinue teat disinfection. Disinfectants are temperature-sensitive; hence, during very cold weather, teat dips not only are more irritant, but also have a lower bacteria-killing power.
- It is inactivated by organic matter – all disinfectants are less active in the presence of milk or faeces. For this reason, it may be better to discard residual dip from the cup at the end of each milking, to clean the cup and to add new dip before the next milking.

Seasonal use of dips

Seasonal postdipping is common in some countries where it is considered that infection only spreads during winter. Some herdsmen have tried stopping postmilking teat disinfection during the summer, only to find that cell counts begin to rise due to a build-up of contagious organisms. The contagious mastitis bacteria will spread during milking throughout the year. In order to be effective, all teats must be disinfected after every milking, irrespective of the season.

Premilking Teat Disinfection

Premilking teat disinfection is an important control measure against environmental mastitis and reduces bacterial counts in bulk milk. Predipping also stimulates milk let-down and hence speeds up milking.

Teats should be clean prior to the application of the milking machine. They may be washed, and the use of a sanitizer in the water further reduces bacterial burdens. If washed, teats must be dried before milking.

Although dry wiping or washing and drying help to reduce bacterial levels on

teats, they are by no means as effective as applying a premilking disinfectant. Table 7.2 shows the benefits of teat disinfection over and above washing and drying, in an experiment where teats were first exposed to an experimental challenge of *Streptococcus uberis* 1–2 hours before milking.

Washing and drying produced a 43% reduction in the percentage of quarters infected. There was an additional 40% decrease in infected quarters when teats were dipped in a disinfectant prior to milking, even though previously the teats had been washed and wiped.

Predipping, first introduced in California, is now widely used in North America and is becoming increasingly popular in Europe. Its prime effect is against environmental mastitis.

One large field trial in the USA (see Table 7.3) involved four herds over a 3-year period. The cows were individually designated 50:50 as predip and control animals, although all were housed, fed and milked as a single group in each herd. Results showed a 46% reduction in the incidence of environmental infections caused by *Streptococcus uberis* and *E. coli* in those cows on which predip was used.

Table 7.2. Comparison of the effectiveness of different methods of premilking teat preparations. (From Galton *et al.*, 1988.)

Teat preparation	No. of quarters infected	% reduction	% further reduction
No preparation	27	–	–
Wash and dry	15	43	–
Wash, dry, then predip and dry	9	67	40

Table 7.3. The effect of predipping on the reduction of new intramammary environmental infections in four commercial dairy herds. (From Pankey *et al.*, 1987.)

Treatment	Number of quarters at risk	Number of infected quarters			% reduction
		<i>S. uberis</i>	Coliforms	total	
Control	553	31	41	72	–
Predip	619	18	21	39	46

Results from field trials in the UK have been similar, with one trial (Blowey and Collis, 1992) showing almost a 50% reduction in clinical mastitis incidence and another a 30% reduction. Predip should be applied before the application of the milking units. If the teats are grossly soiled, they must be washed and dried before the predip is applied. Predip needs a minimum contact time of around 30 seconds and then must be wiped off prior to the application of the machine. To achieve this many milkers adopt the following routine:

- Predip.
- Forestrip.
- Wipe.
- Apply.

This has the advantage of a longer period of predip contact time with the teat, and also the teat is damp when foremilked, making stripping easier. Others say that, for hygiene and teat cleanliness reasons, predip and wipe should be the last procedures prior to unit application. Whichever sequence is used, it is important to fully wipe the teat end to get full benefit from predipping. It is easy to wipe the barrel of the teat but leave a residue of dip and bacteria on the tip at the canal.

Clearly speed of action is important for predips. One novel licensed product with a high (50 p.p.m.) free iodine but low (0.1%) total iodine content is currently sold as having a very rapid action, with a 99.99% kill of teat surface bacteria within 30 seconds. This product is stable at pH 6.5 (most iodophor dips are acid) and hence can be used without an emollient.

Not only can predipping reduce environmental mastitis, but, if teat contamination is the cause of a high TBC/Bactoscan, then predipping will reduce bacterial counts. Improving housing conditions to avoid excessive soiling of the teats between milkings is clearly also important. It seems logical that, if the teat is left soaking in disinfectant for a period of time and then wiped, this must be a very effective method of removing dirt and debris. The effectiveness of predipping is

seen in some herds where coliform counts in milk may reach zero. As with postdipping, care should be taken to ensure that the pot does not become contaminated with faeces.

A further claim by the proponents of premilking teat dipping is that the teats are more moist and supple when the milking machine is applied, and this leads to less liner slip. Some say that teat condition will also improve, but this will clearly depend on the type of postdip used (e.g. high or low emollient). A marked improvement in teat skin condition could explain the anecdotal reports of predipping also leading to an improvement in cell count.

A few farmers have used standard postdip products as a predip, sometimes by diluting the postdip 50:50 with water. This should be avoided for three reasons:

- Postdips may not have the very rapid speed of kill required of a predip.
- The high iodine concentrations used in postdips could lead to residues if the postdip is used as a predip.
- If a diluted postdip is used as a predip, it is essential that full-strength solution is still retained for postdipping, otherwise postdip efficacy will be reduced.

The ideal situation would be to have a single product that could be used as both a pre-and postdip.

In summary, a comparison of the major points of predipping and postdipping is given in Table 7.4.

There will be some exceptions to the points in this table. For example, predipping will reduce initial *Streptococcus uberis* infections, and as such may help to reduce cell counts. Similarly, postdipping will prevent further spread of *S. uberis* and this may reduce the TBC/Bactoscan.

When does predipping not work?

Predipping is commonly implemented as a control measure against environmental mastitis. If it is going to be effective, then a response would be expected within a few

Table 7.4. Comparison of predipping and postdipping.

	Predip	Postdip
Season	Particularly during housing periods, depending on climate	Essential throughout the year
Speed of action	Must be rapid	Not important
Main effect against	Environmental mastitis	Contagious mastitis
Effect on:		
Cell count	Limited	Decreases SCC
TBC/Bactoscan	Decreases TBC (if dirty teats are contributory)	No effect on TBC

weeks (compared with postdipping, which may take several months before an effect is seen). There are however, circumstances where predip does not appear to be effective. These are:

- Environmental infections arising from the dry period.
- Mastitis due to heavy teat-end contamination immediately postmilking. The effects of this are shown in Table 7.5.

Table 7.5. The effect of an *E. coli* broth on new infection rates. Broth was applied to 20 teats either 1 hour before milking or immediately after milking.

Culture of <i>E. coli</i> applied to teat ends	One hour before milking	Immediately after milking
Number of infected quarters	2 in 40 (5%)	14 in 40 (35%)
Effect of predip	Good	Poor

Note that predipping would be less effective if the teats are contaminated immediately after milking.

Iodine Residues

Concern has been expressed about the widespread use of iodine products leading to increased milk iodine levels. Milk is certainly an important source of iodine for man. Most milks contain around 350 µg/litre of iodine. As the adult human daily dietary

iodine requirement is 150 µg, this would be obtained from the consumption of 430 ml (0.75 pints) of average milk. The majority of iodine in milk (around 70–80%) comes from the cow's diet (see Fig. 7.5). Widely differing diets can lead to great variation in milk iodine content. For example, in one trial (Blowey and Collis, 1992), bulk milk iodine ranged from 200 to 4000 µg/litre. Very small amounts of iodine may come from bulk tank cleaners and possibly from sanitizers added to teat washing water.

Perhaps surprisingly, more iodine residues are derived from postmilking teat disinfection than from predipping. This is due to a combination of factors:

- Premilking teat disinfectants are wiped from the teats before the cluster is attached.
- Iodine applied immediately postmilking will penetrate the teat canal.
- In herds where teats are only dry-wiped before milking, postdipping iodine residues will still be present on the teats at the next milking.
- There is good evidence that iodine can penetrate skin and then the teat wall and can pass into milk in the teat cistern.

Products are available with a low (0.1%) total iodine content but a higher (50 p.p.m.) free iodine level. This gives lower residues (and more rapid bacterial kill) than conventional

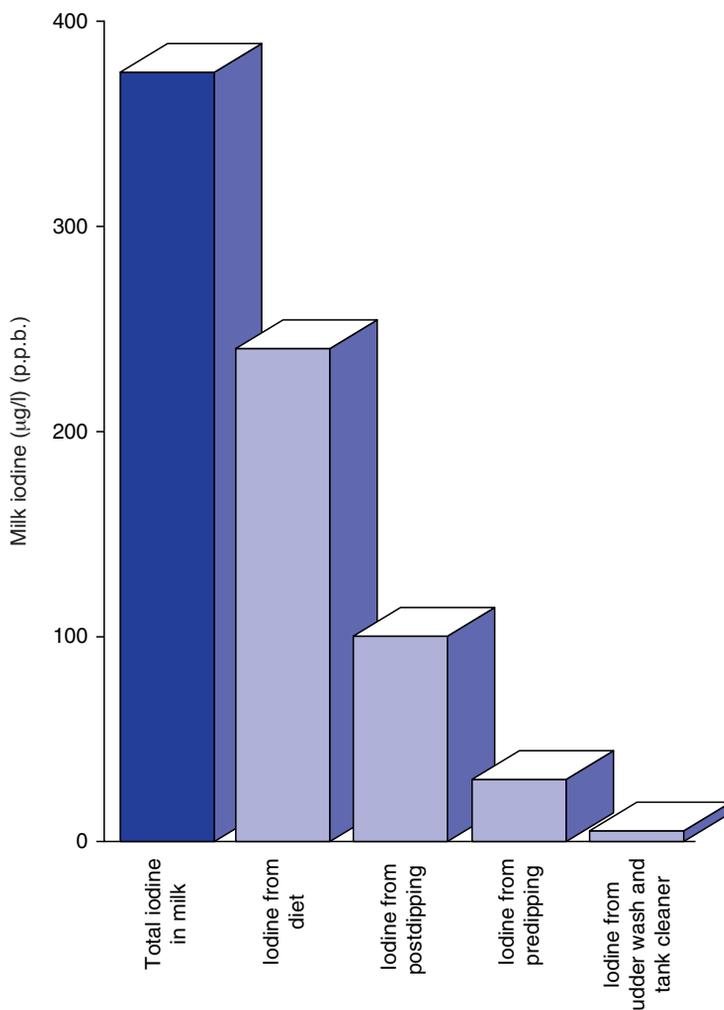


Fig. 7.5. The relative importance of different sources of iodine in milk. There is an enormous variation between farms in the amount of iodine from the diet.

iodine dips with 0.5% total iodine and 2 p.p.m. 'free' iodine and, as such, it is ideal for predipping. It is also stable at pH 6.5 and can be used without an emollient.

When iodine teat disinfectant is applied by spray, there is an increase in atmospheric iodine. Levels do not reach values high enough to represent a human health hazard (Blowey and Collis, 1992), although the vapour may irritate some herdsmen.

Suggested maximum dietary iodine intake limits for the UK are 2000 µg/day (13 times the dietary requirement). A daily intake of only 500 ml/day (less than a pint) of some extreme farm milks would be needed to reach these limits. Most of the milk consumed is from mixed sources, however, and it is therefore unlikely that these extremes would occur with purchased milk.

8 The Environment and Mastitis

Variation in Environments	131
Bedding Types	131
Straw	132
Sawdust and shavings	133
Sand	133
Ash	133
Shredded paper	134
Mats and mattresses	134
Bedding amounts	135
Cubicle sanitizers	135
Space allowances	135
Importance of Ventilation	136
Cubicle (Free-stall) Systems	138
Size	138
Division height	140
Cubicle length	140
Neck rails	141
Brisket boards	141
Cubicle base	142
Management	144
Straw Yards	144
Stocking density	145
Bedding	145
Yard design	146
Sand Yards	147
General Environmental Considerations	148
Avoid high stocking densities	148
Clear away waste food	148
Handle cows gently	149
Rubber parlour floor surface	149
Avoid draughts	149
Heat stress	150
Establish a postcalving group	150
Dry cow hygiene	151

Maintaining a clean and comfortable environment for cows is of major importance in both mastitis control and in the production of clean, quality milk. Comfort and cleanliness also influence the incidence of lameness. It is well known that the incidence of mastitis is related to the degree of bacterial contamination of the teat, and especially the teat end. This chapter is primarily concerned with those factors that help to keep cows clean.

Variation in Environments

Dairy cows are kept under a very wide range of conditions. They may be grazing pastures during a dry summer, or plodding through muddy gateways during a wet spring or autumn. They may be housed in open yards (Plate 8.1) in the hotter climates of Arizona, California, Israel or Saudi Arabia, or in cubicles (free-stalls), cowsheds or straw yards (Plate 8.2) in Europe and the more northern parts of North America.

Whatever the environment, the two major factors that can lead to an increase in mastitis and bacterial contamination of milk are:

- Housing – confinement leads to much closer cow-to-cow contact and therefore a greater opportunity for faecal contamination.
- Humidity – damp conditions facilitate the movement of faeces on to udders and



Plate 8.1. Cows in an open sand yard, typical of hotter climates.



Plate 8.2. Straw yard, typical winter housing for temperate climates. Although very comfortable for the cows, a high degree of management is required to avoid mastitis.

allow greater multiplication of environmental organisms.

Often when cows are housed in winter from pasture, there is an increase in mastitis. Part of this will be associated with cow-to-cow contact, and part will be because, in the UK at least, housing probably coincides with increasingly damp weather.

Large numbers of *E. coli* (1000 (10^3) per gram) are normally excreted in the faeces. This can increase considerably (up to 10^6 per gram) in a freshly calved cow fed on a high-concentrate ration. Even worse than this is the warm mixture of milk, bedding and faeces that can sometimes accumulate at the rear of the cubicle of a high-yielding cow leaking milk. Bedding like this may contain up to 1000 million (10^9) *E. coli* per gram and represents a serious challenge to the mammary gland, especially during milk leakage when the teat canal is open and highly susceptible to mastitis. Milk leakage is a major problem for high-yielding dairy cows.

Bedding Types

Growth of bacteria is dependent on the presence of four major requirements:

1. Food.
2. Warmth.
3. Moisture.
4. Mid range pH.

If any one of these requirements is absent, then bacterial growth is restricted. For example, if the bed is very dry, it will reduce mastitis, as will the use of products that lead to a very high pH. If the bedding is inorganic material, e.g. sand, it should be ideal because it is inert and does not support bacterial growth.

Both the type of bedding and the way in which the bedding is managed can have a marked effect on coliform levels. This is shown in Table 8.1, which compares four housing systems. It is interesting to note that no cases of coliform mastitis were seen in the 150 cows housed over the winter in systems 1, 2 and 3 (sand, straw and well-stored sawdust) but seven cases occurred in 3 months in only 24 cows housed in system 4 (damp sawdust). Table 8.2 shows how different types of bedding support the growth of different organisms on teats. Sawdust was the worst bedding for both total coliforms and *Klebsiella*, while straw produced very high numbers of environmental streptococci on teat skin. This agrees with clinical, on-farm experiences, where *Streptococcus uberis* mastitis is

commonly associated with straw yards. Damp and badly stored sawdust may have a high coliform count. However, provided that it is stored dry (i.e. not allowed to ferment) and is kept dry on the cubicle beds, there is no reason why sawdust or shavings should not be used as bedding material. They can be particularly useful when rubber mats and automatic scrapers are in use. Cubicles bedded with sand and ash are probably ideal and will reduce the incidence of both *E. coli* and *S. uberis*, but sand may cause problems with slurry systems.

The following section describes the main properties of each bedding type.

Straw

Straw is an organic material and hence supports bacterial growth. This is especially the case if the straw is damp. Normal straw has around 12% moisture (88% dry matter (DM)), but this can reach 30% if the straw is baled at a time of sea mist, stored under plastic sheet which prevents air

Table 8.1. Coliform levels using different housing systems. (From Bramley *et al.*, 1981.)

Group	Housing system	Number of coliforms/g bedding	Cases of coliform mastitis
1	Sand cubicles	37,000	0
2	Straw yards	47,000	0
3	Well-managed sawdust yards	44,000	0
4	Poorly managed sawdust yards	66,000–69,000	7

Table 8.2. Comparison of growth of mastitis organisms in three different types of bedding. (From Rendos *et al.*, 1975.)

	Bacterial counts (geometric means)					
	Sawdust		Shavings		Straw	
	Bedding ^a	Teat ^b	Bedding	Teat	Bedding	Teat
Total coliforms	5.2	127	6.6	12	3.1	8
<i>Klebsiella</i>	4.4	11	6.6	2	6.5	1
Streptococci	1.1	38	8.6	717	5.3	2064

^aCount per g/used bedding ($\times 10^6$).

^bCount from teat swab.

flow, or stored outside (see Plate 8.3). Not only does wet straw absorb less moisture, but it might contain yeasts and moulds that could cause mastitis. Damp straw is especially associated with outbreaks of *S. uberis* mastitis (Fig. 4.2), and hence straw to be used as bedding should always be stored under cover.



Plate 8.3. Straw bales that are not stored under cover get damp and predispose to mastitis if used for bedding.

Sawdust and shavings

Sawdust and shavings are also organic and, like straw, support bacterial growth. If sawdust is used it should be kiln-dried and not from fresh-cut wood. Kiln-dried shavings are around 90% dry matter, whereas sawdust from recently felled wood may have as low as 70% dry matter, or even lower if it has been stored outside in the wet. There is clearly little point in using bedding that already contains 30% moisture. If it feels warm in the pile it is dangerous to use. Some manufacturers are now producing a fine wood chip from waste pallets, etc., to be used as bedding. This should drain well, but will support bacterial growth.

Sand

Sand is probably the ideal bedding. Provided it is deep enough (ideally 4 to 6"), it provides and produces good comfortable clean bedding and hence reduces both mastitis and lameness (Plate 8.4). Being inor-

ganic, provided that it is clean, it does not support bacterial growth. However, if the sand at the rear of the cubicle goes black and damp, then it should be dug out and replaced with clean sand. This happens when the sand has become contaminated with urine, milk or faeces; then, with the warmth of the cow lying on it, quite high bacterial levels can be produced. A sand cubicle will always have a rear lip. This is an advantage to the cow as she will be able to push against the lip with her hind feet when rising to stand, and this increases cubicle comfort.

The type of sand used must also be chosen carefully. If the clay content is too high, the sand will become compacted and go hard, especially at the rear of the bed, where there is often more moisture. This leads to cow discomfort, pooling of moisture at the back of the cubicle and hence an increased risk of mastitis. Squeeze a sample of sand in your hand. If it is compacted into a ball and retains its shape when released, the clay content is too high. Like most other bedding types, sand is best stored under cover, otherwise it may be too wet before it is even used as bedding.

Ash

Ash, a waste product from paper-, cardboard- and wood-fired power stations, has



Plate 8.4. This sand cubicle (which has a lip) gives good comfort, but note that, in the adjacent cubicle, it is damp at the back. If the sand in this area goes black, then it should be dug out and replaced with clean sand.

recently been introduced as a bedding material and seems to have some significant benefits. In addition to its intense drying and water-absorbing properties, ash has a very high pH of 9 to 11, and this in itself reduces bacterial growth, including that of *S. uberis*. Care must be taken that teats do not become excessively dry or affected by superficial burns, especially if a 'barrier' postdip is being used, because the ash tends to stick to the film of barrier dip. Most farms use ash in combination with other bedding. It mixes with sand especially well (Plate 8.5), producing a dry material that does not become compacted. Ash can also be used as a 2–4 inch layer on the base of a yard to retard the rate of fermentation of the straw bed, and ash on concrete provides a good, firm, non-slip surface.

Shredded paper

Shredded paper has been used as bedding, but has not become popular. It is not particularly absorbent and when wet it tends to become matted and solid. It may also stick to cows' flanks where it looks untidy. Mixtures of cardboard and wood chip are better, as is shredded waste plasterboard from the building industry.



Plate 8.5. Ash from wood- and paper-fired power stations can be mixed with sand to produce excellent bedding. Its high pH prevents bacterial growth, especially of *Streptococcus uberis*.

Mats and mattresses

The ideal bedding should be soft and yielding, to encourage the cow to lie down, but at the same time strong enough to prevent damage from the movements of the cow. It should also be clean and hygienic. Mattresses (Plate 8.6) and rubber mats are good, and represent both increased comfort and a saving in bedding costs, but they must be kept dry and lightly bedded, as in Plate 8.6, otherwise there is a risk of mastitis and hock sores and of cubicle rejection. Some bedding should always be used, the amount depending on the softness of the bed. This is for two reasons. First, it is to keep the bed dry; otherwise the sweaty skin will leave a damp area and predispose to mastitis. This is especially the case if there has also been milk leakage on to the cubicle bed. Second, a small covering of bedding is needed to provide an anti-friction surface, as the cow slides across the bed when she is lying down. Inadequate bedding leads to hock sores.

If the rear edge of the cubicle mattress is slippery, then the cow may slip as she pushes with the toes of her hind feet to stand, and this can increase the incidence of lameness. This has led to many farmers returning to cubicles with a rear lip. Mattresses are often softer and more comfortable than mats, and should ideally



Plate 8.6. Cow mattresses are often formed from canvas sacks filled with rubber chippings. The mattress must have sufficient bedding; otherwise the rear of the cubicle will be both damp and slippery.

extend to the rear of the cubicle; otherwise the hock may be uncomfortable due to lying on a ridge.

Bedding amounts

Clearly it is not just the type of bedding that influences mastitis; the quantity of the bedding used and the frequency of renewing it are also important. Ample bedding, especially with dry materials such as straw or shavings, is essential to keep cows clean. The absolute amounts required will depend on cubicle design, the presence or absence of mats or mattresses and the space within the building. If space is limited, e.g. the cubicle passages are narrow, or there is less loafing (relaxation) area, then more bedding will be required. A very rough guideline to the amounts of bedding required (kg per cow per day) is given in Table 8.3.

The amounts given in Table 8.3 are approximate figures only. Clearly the more bedding that is used the greater will be the cleanliness of the cows. For example, in Plate 8.7 straw use was 5.0 kg per cow per day, giving a good covering even across the passage. The cows were very clean, and lameness and mastitis were low, although with straw the risk of *S. uberis* remains.

Table 8.3. Approximate bedding and sanitizer (lime) requirements (kg/cow/day) for cubicles and yards. Amounts will vary enormously with factors such as stocking density, ventilation, weather and diet.

Bedding material	Cubicles with		
	Cubicles	mats or mattresses	Yards
Straw	2.5	1.0	15
Sawdust	2.0	1.0	nu ^a
Sand	8.0	1.0	10
Ash	4.0	1.5	nu
Cardboard	2.0	1.0	nu
Lime	0.05	0.025	nu

^anu = not used

Cubicle sanitizers

Addition of small quantities of lime (see Table 8.3) or other proprietary cubicle sanitizer powder will help to dry the beds, and its high pH acts as a disinfectant. *S. uberis* can multiply at up to pH 9.5, however, so significant quantities need to be used. Make sure that it is hydrated or slaked lime (calcium hydroxide, Ca(OH)₂) or even ground limestone (calcium carbonate, CaCO₃) and not quicklime (calcium oxide, CaO), as the latter will cause severe teat burns. Lime should be added to the cubicle bed first, then the bedding placed on top, as this will further reduce the risk of excessive teat drying. This has not been done in Plate 8.6. A range of other proprietary cubicle sanitizers are available, their main claim being that they are less likely to cause teat skin burns.

Space allowances

In the introduction to this chapter, it was stated that housing cows increased their degree of contact and that this predisposed to mastitis. It follows, therefore, that providing more space, in terms of both air volume and floor area, will be beneficial. Original cubicle buildings were only 10 ft to the eaves, the roof was supported by the cubicle division, and the passageway between cubicles was only 8 ft in width. Modern cows



Plate 8.7. A wide passage and high levels of straw bedding help to keep cows clean and reduce both mastitis and lameness.

are, of course, much higher-yielding, but buildings are now erected at 20 ft to the eaves and with passage width between the backs of cubicles up to 15 ft wide. This change from 8 ft to 15 ft passages gives the cows almost double the space and, of course, there is half the amount of slurry in the passageway.

Most farms are stocked at no more than 90 to 95% cubicle occupancy, i.e. there are 5 to 10% more cubicles than cows in the building. This is especially for the high-yielding group, and is often a requirement of farm assurance audit schemes. There is considerable discussion over whether buildings should be two-row or three-row barns, as this affects the feed space allowance, and will have an indirect effect on mastitis and lameness. If the cubicles are 4 ft in width, then a two-row barn has 2 ft of feeding space per cow, which is ideal, whereas a three-row barn has only 1.3 ft per cow feed space. If space is limited, cubicles are uncomfortable or the building is poorly ventilated, then cows will lie outside on concrete (Plate 8.8), with the obvious hygiene and mastitis consequences.

Space allowance is also important in straw yards, particularly for transition cows and fresh calvers, the two groups that are at greatest risk of contracting new infections (see section on dry period infections on pages 50–52). A generous allowance would be 8 sq.m of bedded area per cow, increas-

ing to 10 sq.m for fresh calvers. If housing options are limited, then in the short term increased space allowance can be provided by outside loafing areas, although provision may need to be made for shelter and/or shade in periods of inclement or very hot weather.

Importance of Ventilation

Cows are extremely wet animals (Plate 8.9). The fluid produced by a high-yielding dairy cow consuming large quantities of food is enormous. Approximate figures are:

- 4–5 litres per day from skin and respiratory tract (treble this on a very hot day).
- 20 litres per day in urine.
- 30 litres per day in faeces.

High-yielding cows also produce large amounts of heat, around 1.5 to 2.0 kW/hour, depending on the level of yield. It is therefore vitally important that buildings are designed to be well ventilated in order to remove this heat and humidity, and that stocking densities are kept in reasonable proportions, to avoid a build-up of heat and moisture. Under current UK conditions, it is unlikely that cows will get too cold. The major reason for housing them is for ease of feeding and to protect the land (from foot poaching, i.e. foot damage to the pasture)



Plate 8.8. A combination of uncomfortable cubicles and warm weather encouraged a large number of cows to lie outside, with a consequent increase in mastitis.



Plate 8.9. As can be seen from the amount of moisture being exhaled, cows are extremely ‘wet’ animals. They excrete over 50 litres of water per day in the urine, faeces, breath and sweat, in addition to the milk that they produce.

and not to protect the cows. Hence, if cows can be kept as close to lower environmental temperatures as possible and protected from the direct effects of driving rain or direct sunlight, this should be ideal. Damp, hot and humid conditions predispose to mastitis. Heat stress leads to excess standing, predisposing to mastitis, and is discussed at the end of this chapter.

Long, narrow, blind-ended straw yards where you can ‘feel’ the humidity and stale air at the far end are particularly dangerous (see Fig. 8.15). Poorly ventilated cubicle buildings with a low roof, where condensation drips on to both cows and bedding on a cold morning, will predispose to mastitis and respiratory diseases such as IBR (infectious bovine rhinotracheitis). If you can’t see the far end of the shed because of condensation and mist or if condensation is dripping from the roof on to the cows’ backs (Plate 8.10) then ventilation is totally inadequate.

Some suggested ways of ensuring adequate ventilation include:

- Ensure an adequate apex outlet at the roof. Air will only flow into a building if it can get in and get out again. This is best achieved by having a 23–30.5 cm gap (approx. 9–12”), plus a 15 cm (approx. 6”) ‘upstand’ on the final sheet of roofing material (see Fig. 8.1). A cross-flow of air across the upstand produces an extractor effect. The conventional roofing cowls with a narrow outlet simply do not allow sufficient air flow.



Plate 8.10. Condensation dripping from the roof joists is considered to be a sign of poor ventilation.

Fig. 8.1. Leaving a 23–30.5 cm gap at the apex of the roof and adding a 15 cm upstand improves ventilation.

(a)

(b)

Fig. 8.2. (a) Conventional roof sheeting in which the edges are faced downwards and adjacent sheets overlap. (b) It has been suggested that ventilation can be improved by reversing the sheets so that the edges face upwards and a gap of 1.3–1.9 cm is left between each sheet.

- If a new building is being constructed, turn the roofing sheets upside down and leave a 1.3–1.9 cm (approx. 0.5–0.75”) gap between each run of sheets (Fig. 8.2a and b). Provided that there are sufficient animals in the building to produce heat and an upward flow of air, under most conditions this seems to prevent rain from entering and improves ventilation. It also reduces roofing costs, as fewer sheets are used.
- A similar effect may be obtained in existing buildings by using an angle grinder to

cut narrow slots into the top of every fourth to sixth ridge of the roofing sheets (Fig. 8.3). If this is done close to the apex of the roof, it will have a particularly good effect.

- Clad the sides and gable ends of the buildings with Yorkshire boarding (spaced vertical boarding), leaving a 12.7 cm (approx. 5") gap between boards. In many buildings, alternate boards are adequate, especially if facing a feed passage, or, if the area is reasonably sheltered from rain, then the building is best with no sides at all.
- Avoid multiple-span buildings (Fig. 8.4). By far the best air flow is achieved when buildings stand singly. Also avoid buildings of excessive span, e.g. more than 18.3 m wide (approx. 60').
- Ensure adequate drainage. Standing water increases the humidity within a building and further predisposes to mastitis. Straw yards with earth or sand floors and cubicle houses with good drainage or slatted passageways both reduce the amount of standing water.
- In older wooden buildings air flow can often be improved by cutting out part of the fronts, as in Plate 8.11. Provided that



Plate 8.11. As this wooden cubicle house was close to an adjacent building, removal of most of the front not only improved cow comfort but also improved air flow and overall ventilation.

there is a rail or similar to stop the cow passing through and that there is no direct exposure to rain, this will be a big improvement.

Cubicle (Free-stall) Systems

The most important features of cubicle systems are their design and management. Cubicles should be designed to be comfortable for cows and to be in constant use, but to stay reasonably clean.

Uncomfortable cubicles will often stay clean, simply because the cows do not use them, but so many cows then lie outside on the concrete that mastitis (and lameness) becomes a problem. Plate 8.8 shows a typical example. In this instance, a combination of warm weather and uncomfortable cubicles led to a large number of cows lying outside, with a resultant increase in mastitis.

One of the most important factors determining cubicle acceptance is pre-partum heifer training. Although training is vital, cubicle design is also important, and is discussed in the following section.

Size

This must depend on the size of the cow, but for modern large Holsteins cubicles

Fig. 8.3. Ventilation can be improved in existing buildings by cutting slots into the top of every fourth to sixth ridge of the roofing sheets.

Fig. 8.4. Avoid a multiple-span building such as this. The best ventilation is obtained from a single-span building that is not excessively wide.

Fig. 8.5. A single row of cubicles needs to be 2.4 m (8') long (above). Two facing rows can be 2.3 m (7' 6") long (right).

2.3–2.4 m long by 1.2 m wide (7' 6"–8' long by 4' wide) are reasonable dimensions (see Fig. 8.5). Length seems to be the most important dimension affecting cubicle acceptance. If cubicles are too wide, then cows may not lie straight, and this leads to soiling of the bed, as in Plate 8.12. Lying diagonally can also be a result of the lower edge of the metal cubicle division being too high, e.g. more than 22" above the cubicle bed.

Where there is a double row of facing cubicles (Fig. 8.5, right), space-sharing at the front makes a 2.3 m length acceptable. The



Plate 8.12. If the cubicle is too wide, or if the lower edge of the rear cubicle division is too high, cows may lie diagonally, leading to soiling of the bed.

cubicle should be such that a cow can sit in it, with her head held extended forwards to ruminate. If the cubicle is too short, she has to sit with her neck flexed (Fig. 8.6), which makes it difficult for her to regurgitate the cud. In addition, there is insufficient lunge space for her to regain the standing position.

Cubicle discomfort also encourages cows to stand for excessive periods of time and predisposes to lameness. Cubicles that are too narrow, or which have excessively rigid divisions, can lead to compression of the rumen when the cow is lying down. This can further impede rumination, as well as discouraging cubicle acceptance. Figure 8.7 shows one such design. Note how the rumen of a large cow would be directly compressed by the cubicle division. By removing both the upright bar AB and the horizontal CD, and replacing them with a length of rope under tension, as in Fig. 8.8, the cubicle becomes much more comfortable.

Fig. 8.6. (Left) A cubicle that is too short forces the cow to sit with her neck excessively flexed, thus impeding rumination. (Right) Space is needed at the front of the cubicle to allow the cow to extend her neck during regurgitation of the cud, and to allow forward lunging when rising to stand.

Division height

The height of the division is also important, especially at the front of the cubicle. If height PQ (Fig. 8.7) is too short, it will be uncomfortable for the cow when she is standing and she may also have to depress her neck when sitting 'space-sharing' with the adjacent or opposite cubicle. This further reduces comfort and impedes rumination. Clearly optimum height varies with cow size, but 1.32 m (4'3") for Holsteins has been suggested. The rear upright above R (Fig. 8.7) is best eliminated to give the cantilever division seen in Fig. 8.10.

Fig. 8.7. Cubicles with excessively rigid divisions can be uncomfortable. Pressure is exerted on the rumen (circled).

Fig. 8.8. A flexible division gives greater cow comfort. The lower cubicle rail can be replaced by a rope under tension. A two-stranded rope is brought under tension by rotating a piece of wood (see arrows) fixed between two strands. When the rope is taut, the wood is tied to the top cubicle rail.

Cubicle length

Cows may get too far forward with long cubicles and defaecate on the bed when lying or standing. Some animals also occasionally shuffle far forward on their knees, finishing up so close to the front wall (as in Fig. 8.9) that they either have great difficulty in standing or are totally unable to rise. This most commonly occurs with an uncomfortable base. Ideally, a cow should have at least 1.2 m (4 ft) of forward lunging space to enable her to stand easily.

Fig. 8.9. Some cows move so far forward in the cubicle that they are unable to lunge forward and therefore find it very difficult to stand up.

(a)

(b)

Fig. 8.10. (a) Neck rails may either be fixed to the top of the cubicle divisions or (b) preferably suspended above the divisions.

Neck rails

Encouraging the cow to remain in the correct position in the cubicle can be achieved by using either bricket boards or neck rails or both. Neck rails can either be attached to the top of the cubicle divisions, or suspended above, as shown in Fig. 8.10. In either case, they should be positioned approximately 30–45 cm (approx. 1'–1' 6") from the front of the cubicle, although this varies enormously depending on cubicle length. The suspended rail, positioned to be 7.5–10 cm (approx. 3–4") below the neck height of a standing animal, is preferable, since rails attached to the cubicle division may be so low as to discourage cubicle acceptance. Both have the same disadvantage, however, in that once she is lying down, there is nothing to prevent the cow shuffling into the position shown in Fig. 8.9. However, when she stands up, the

Fig. 8.11. A bricket board prevents the cow shuffling too far forward. It should be angled forward towards the front of the cubicle to reduce knee damage.

presence of the rail on her neck will encourage her to reverse to the rear of the cubicle and thereby urinate and defaecate into the passage.

Bricket boards

Bricket boards sited 1.72 m (5' 8") from the back of the cubicle (Fig. 8.11) will certainly stop the cow shuffling too far forward. However, when she stands up she can still easily stand over the front of the board and defaecate onto the rear of the cubicle. A neck rail is therefore needed in conjunction with a bricket board.

Bricket boards should have rounded edges rather than the sharp square edge shown in Fig. 8.11. Soft, moulded, pillow-shaped, plastic tubes are ideal. They must not be too high, however, or they may prevent the natural 'one front leg forward' position adopted by a proportion of sitting cows.

A long, pyramidal concrete shape, 0.38 m (15") high was once used between two facing rows of cubicles (Fig. 8.12 and Plate 8.13), as a means of correctly positioning cows in the cubicle. When seated, the cow could no longer go too far forward in the cubicle and yet the height CD meant that she could extend her neck over the top of the

Fig. 8.12. A pyramid of concrete between two facing rows of cubicles (or a triangle against a wall) prevents cows moving too far forward, while at the same time allowing sufficient space for chewing the cud and lunging forward to stand. This is no longer popular as it may lead to cubicle rejection.

pyramid into facing rows of cubicles. When fully standing she had to keep her feet back behind B and hence defaecated into the dunging channel. However, when rising to

stand, one foot had to be placed on T, the slope of the concrete, to push herself upright, and many cows found this uncomfortable. The system is still in use in some housing situations, but is not popular.



Plate 8.13. A concrete pyramid 0.38 m high between two facing rows of cubicles was once used to prevent cows going too far forward. It is no longer popular.

Cubicle base

Limestone, earth, sand and concrete have all been used for cubicle bases. The first three all suffer the disadvantage that they gradually become eroded to form pits, which at the rear of the cubicle can become filled with damp, soiled bedding, which will then represent a source of mastitis infection. Plate 8.14 shows a typical example.

Figure 8.13 shows the three major points of contact when a cow is lying in a cubicle. These are the positions of the two knees (A and B) and either the right hock (as in Fig. 8.13) or the left hock, according to which side the cow is lying on (C). These three contact points are clearly recognizable in many cubicles: look for the three areas where straw bedding has been scuffed away, often exposing bare concrete. Unless a concrete base is used, cows continually lifting themselves eventually erode depressions at the front or back of the cubicle, leading to discomfort. The rear depression can also become soiled with faeces and wet bedding, as in Plate 8.14. If sand is in use, it needs to be deep enough to remain spread over the



Plate 8.14. Badly soiled cubicle beds represent a mastitis risk.

The majority of farms now have concrete bases in their cubicles. Although these are certainly easier to keep clean, they can be hard and uncomfortable and this may lead to cubicle rejection. Mats, mattresses or deep bedding are essential.

Animals accustomed to cubicles with a lip often touch the lip with their toe before stepping into the dunging passage. Removal of this lip (for example by concreting the cubicle base) can induce apprehension in some cows because they do not know when and where to step down and this too can lead to cubicle rejection. Increasingly people prefer cubicles with a lip (such as that shown in Plate 8.4). Additional effort may be needed to keep the rear of the cubicle clean (and correct positioning of the cow is vital), but a lip positions the animal better: her tail lies inside the cubicle and not in the slurry passage, and the lip is used as a point of contact for the hind feet when the cow pushes to stand up.

Cubicles with a high kerb (e.g. greater than 12.5–15 cm or 5–6") were once thought to be a problem, as heifers especially may have been nervous about reversing from a high step. However, provided animals have been trained in advance, step height is probably not a major factor for comfort, and steps of 250 mm (10") are acceptable.

The slope of the cubicle floor is important and should be 10–13 cm (4–5") from front to rear, that is from Q to R in Fig. 8.7. A

Fig. 8.13. A, B and C are the major contact points between the cow and the cubicle base and must be well bedded.

whole cubicle base. Mats and mattresses will prevent this unevenness developing, although bedding is still needed to prevent hock abrasions.

An example of an advanced case of hock abrasion is shown in Plate 8.15. The lesion is first seen as hair loss over the bruised skin, which is followed by fluid accumulating in the hock bursa. (A bursa is a small 'shock-absorber' pouch, which acts to protect a protruding portion of bone and allows skin, muscles, tendons, etc., to glide over the bony surface.) Only when the skin is broken would the swellings shown in Plate 8.15 become infected.



Plate 8.15. Gross hock swellings, the result of lying on a hard surface. The hair loss and scab on the skin surface show the major points of abrasion.

cow much prefers to lie uphill. A level or, even worse, downward-sloping cubicle could lead to rejection.

Management

Cleaning and renewing the bedding of the cubicles and yards should ideally be carried out during milking, so that as cows exit from the parlour they are able to walk back along clean passageways, past fresh food, and then lie down in clean cubicles. Ideally, all soiled areas should be scraped from the backs of the cubicles at least twice daily (and preferably every time the herdsman walks past). If using straw, sawdust or shavings, fresh bedding should be added daily, although, if straw usage is liberal, it may be sufficient (but not ideal) to bed the cubicles twice weekly, scraping fresh straw from the front to the rear of the cubicle every day, as required. A small quantity of hydrated lime ($\text{Ca}(\text{OH})_2$) or even ground limestone (CaCO_3) sprinkled on to the rear of the cubicles once or twice a week (as in Plate 8.16) also helps to keep the bed dry, as lime absorbs moisture. Lime should be applied and then covered with fresh bedding (e.g.



Plate 8.16. Hydrated lime both dries and disinfects cubicle beds. However, do not use an excess as it can also produce drying and cracking of the teats.

straw or sawdust). This prevents direct and excessive contact of lime with teats, which could otherwise lead to cracking. Do not use quicklime, as this will produce severe teat burns.

The importance of regularly renewing the bedding is shown in Fig. 8.14. When sawdust was added to cubicles on a weekly basis (A), coliform levels were quite high. Levels declined when daily bedding was carried out (B), but the situation soon de-teriorated when there was a return to the original system of weekly bedding (C).

Cubicle passages should be scraped at each milking, and ideally this should be carried out before the cows return to the cubicles (Plate 8.17). This keeps the teats as clean as possible during the first critical 20–30 minutes after milking, when the cow is more susceptible to mastitis, because the teat sphincter has not fully closed. It also reduces the amount of faeces carried back on to the cubicle beds by soiled feet.

Straw Yards

Straw (loose) yards (Plate 8.2) are certainly good for cow comfort and, if given the choice, cows would opt for yards rather than



Plate 8.17. A semi-automated system. The tractor is scraping slurry from the passageway, a brush has removed soiled material from the rear of the cubicle on the right, and clean sawdust is being added to the cubicles on the left. The milking is then ready for the cows to enter after milking.

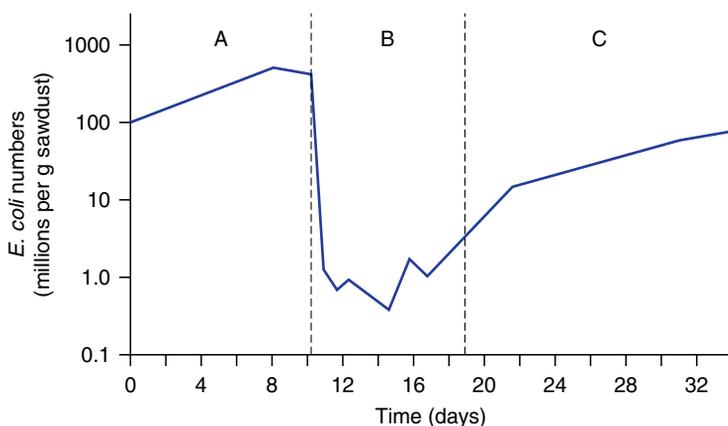


Fig. 8.14. The importance of regular renewal of cubicle bedding. *E. coli* numbers were high when fresh sawdust was added weekly (A), fell rapidly when daily bedding was introduced (B), but soon deteriorated on return to weekly bedding (C). (From Bramley, 1992.)

cubicles. However, they are not without problems. Whereas with cubicles cows can be positioned to drop urine and faeces into the passageway and hence keep the teats and udder clean, with straw yards there is a greater chance of faecal soiling of the udder. Hence there is generally an increased mastitis risk, especially when yards are badly designed or poorly managed, but, because they are more comfortable, there is usually a lower incidence of lameness. Straw usage is much higher (almost ten times more) than in cubicles and hence both bedding and labour costs are higher.

Stocking density

Stocking densities tend to be lower, because fewer cows can be housed in a straw yard than if the same building were used for cubicles. Current recommendations are that cows need at least 6 sq. m (65 sq.ft per cow) of bedded area, plus a further 1.8 sq. m (20 sq. ft) for feeding and loafing, i.e. 8 sq. m (85 sq. ft) per cow in total, and that fresh calvers and transition cows be given 10 sq. m (110 sq. ft) per cow. Higher figures than this may be needed for large cows.

Bedding

Yards should be bedded at least once a day, preferably during morning milking, and, as with cubicles:

- Cows should be encouraged to stand and eat for 30 minutes after milking (but not if this means locking them into an overcrowded and draughty passageway).
- Passageways leading back to the yards should be scraped clean before the cows walk along them.

The straw used for bedding must be clean, dry and free from fungi and moulds. Straw stored outside will significantly increase mastitis risks, and herd outbreaks will occur with damp and mouldy straw, even if liberal quantities are used. Mastitis caused by yeasts and moulds is a particular problem, because of its poor response to treatment.

Straw beds tend to heat up. Fortunately, the anaerobic fermentation that occurs in the compacted lower levels does not support the growth of *E. coli*, but surface temperatures in normal yards are around 40°C and this promotes bacterial growth (Plate 8.18). There is some suggestion that the use of excessive quantities of straw can lead to overheating and that this can increase coliform counts. Yards should be cleaned out frequently, at



Plate 8.18. Muck from a straw yard ferments and heats to around 40°C. Only the upper layers are likely to contain mastitis organisms.

least every five weeks. If left longer, there is an increased risk of mastitis. Some farms clean out as frequently as every 2–3 weeks and suggest that this uses less straw. After cleaning out, hydrated lime or power station ash can be spread over the floor before renewal of bedding. This reduces the rate of fermentation and increases the time before the next batch of bedding in the yards starts to heat up.

A hard-core base may be better than concrete, in that it permits better drainage. However, this is only likely to be of major importance in yards where the concrete base

is flat and poorly drained. If the straw squelches when you stand on the bed, then it's too wet.

Yard design

One of the most important aspects of straw yards in relation to mastitis is the design of the yard. Long, narrow yards (as in Fig. 8.15, on the left) are more easily soiled, because cows have to walk across a greater distance to get to the rear. The positioning of the water troughs (W) in the example shown is also very poor as the only access to them is through the bedded area.

The design shown on the right is far better. Access to the water troughs (W) should only be from the feeding passage (P) and hence this avoids excessive soiling of the bedded area (and it is less important when the water trough overflows).

Opinions differ on the value of the barrier BC. By restricting access, areas AB and CD become more soiled, but the area behind BC (H) stays cleaner, and cows prefer to lie against a wall if possible. Some systems have a continuous step approximately 30 cm high (12") running from A to D. This helps to retain the straw bedding and gives full access to the yard. The depth of the yard



Fig. 8.15. Design of straw yards: long, narrow, poorly ventilated yards with badly placed water troughs should be avoided (left). A more useful design is shown on the right.

(AE) should be at least 7.3 m and, preferably, no greater than 9.1 m (24–30 ft), with a feed passage (P) of 3.5 m (approx. 12 ft) minimum and a food trough (or floor area) (F) of 0.76 m (2' 6") per cow. Scraping the passage (P) twice daily further reduces faecal soiling of the bed.

Ventilation is equally important in straw yards to that in cubicle houses and cowsheds, and can be improved by providing roof insulation. However, this is rarely done, because of cost. The ideal humidity for dairy cows is around 70%, whereas many buildings reach 85% or higher in the UK during winter. High humidity also increases the heat stress in cows kept in hotter climates and should be avoided if possible.

Sand Yards

In hotter climates and desert regions cows are housed on sand corrals or yards and may have access to cubicles or a slatted area under cover. Shaded areas are vital and in the absence of specific shade cows tend to congregate and shelter along the edges of buildings, as seen in Plate 8.19. More commonly, tall constructions providing shade are erected, and cows will lie in their shadows. The dimensions and siting of such shaded areas are vital. Ideally they should provide shade over different parts of the sand yard throughout the day, so that all areas under shadow are also exposed to the drying influence of the sun at least once each day. Plate 8.20 shows cows lying under a sun shelter.

During the dry season, corrals should be cleaned out every 6–8 weeks. The top surface of the sand is scraped off and removed. In the Middle East, soiled sand (sand and dry faeces) is a valuable commodity for horticulture. Fresh sand is brought in to top up the yard (Plate 8.21). Sand provides good drainage and the heat of the sun dries the faecal pats, which are then broken up each day by a tractor and scraper (Plate 8.22).

During wet weather sand yards become very muddy (see Plate 8.23) and cleaning teats prior to milking becomes a major task. The risk of environmental mastitis increases



Plate 8.19. In the absence of specific shelters, cows congregate along the sides of buildings to obtain shade.



Plate 8.20. Artificial shade from the sun in hotter climates. Note how the cows specifically lie in the areas under shadow.



Plate 8.21. Yards should be cleaned and fresh sand provided every 6–8 weeks.



Plate 8.22. Sand yards need to be scraped daily to break up dried faecal pats.



Plate 8.23. Under wet conditions, open sand yards become a real problem.



Plate 8.24. In temperate climates, access to clean, open loafing and feeding areas helps in mastitis control, reduces lameness and improves heat detection.

substantially. If possible, cows should be kept in cubicle areas, preferably with fans as a cooling system, until the yards dry out.

General Environmental Considerations

There are certain points of general management that are applicable to all housing systems.

Avoid high stocking densities

Tightly packed cows create high humidity and are often under stress, especially younger heifers. Whenever possible, a large loafing area should be provided (Plate 8.24). In many parts of the world this need not be totally under cover, since cows are prepared to go outside in quite low temperatures, as long as it is neither raining heavily nor extremely windy. In hotter climates, loafing areas can be used at night.

The provision of adequate loafing (and feeding) areas also aids in heat detection and helps to reduce the incidence of lameness. This is because cows that have enough space to walk around are likely to suffer less damage to their feet than cows that stand still for long periods of time. In addition, if they are able to move away from other cows, not only are they more likely to show better signs of oestrus (heat) behaviour, but it will be easier to identify such cows.

Clear away waste food

Waste silage or other food left lying beside the trough encourages cows to lie outside (see Plate 8.25). It can also be a good culture medium for environmental mastitis bacteria, particularly *E. coli*, *B. licheniformis* and *B. cereus*, and by contaminating the teats can produce high TBC/Bactoscans. Areas around the feed troughs should therefore be cleaned regularly.

Handle cows gently

There is now a considerable volume of evidence to show that stressed cows are more prone to infections, and this includes mastitis. If rushed along roads and through doorways, they may injure or excessively soil their teats. If forcibly driven into the milking parlour, the cows' let-down is likely to be inhibited, with the consequences of longer milking times, increased teat-end damage and depressed yields (see section on heifer milk let-down, pages 14–15). Make sure the backing gate allows them plenty of room in the collecting yard, and allow the cows to flow into the parlour at their own speed. If the gate is pushing the cows forward too hard, they will become stressed and then more difficult to handle in the parlour, with poor milk let-down. The difference between quietly and roughly handled cows very soon becomes apparent from their reaction to visitors.

Most farms now have a foot bath after the parlour exit. To avoid contamination of 'open' teats, the bath should not be too deep (around 70 mm), the solution should be changed daily to avoid excess contamination and, for large herds, it should be wide enough to allow two cows to pass at the same time.

Rubber parlour floor surface

Increasing numbers of dairy farms now have rubber matting fixed to the parlour floor (Plate 8.26). Although primarily aimed at lameness, a rubber floor also has some advantages in mastitis control. Because it is more comfortable, cows stand more quietly and fidget less, and this is reported to lead to reduced liner slip. Cows are said to flow into the parlour better, thus reducing overall milking times. This should reduce both mastitis and lameness. Another potential advantage is that rubber flooring overcomes the erosive damage caused by some barrier dips. Plate 8.27 shows how the cement surface of a parlour floor has been eroded by one commercial dip. The presence of a white aggregate on the surface makes it quite diffi-



Plate 8.26. Rubber flooring in the milking parlour is claimed to have advantages for both lameness and mastitis control.



Plate 8.27. The cement surface of this parlour floor has been eroded by a barrier teat dip, exposing a white aggregate. This makes detection of mastitis in the foremilk more difficult.

cult to identify mastitis during foremilking, whereas milk is much more easily inspected with black rubber flooring.

Avoid draughts

Chilling of the udder may reduce the immune response and hence the cow's ability to counteract infections that have penetrated the teat canal. Chilling of teats will undoubtedly lead to cracking and chapping, and this further predisposes to mastitis. Cows must not be left standing for 20–30 minutes (to allow the teat canal to

close) in exposed yards or draughty passageways, especially when the teats are still wet with teat dip. It is better to allow them to return to feed.

Heat stress

At the other end of the spectrum, it is also important to keep dairy cows cool, and even in the UK heat stress can become an issue, leading to increased mastitis. Heat stress can affect cows at surprisingly low temperatures, for example, early changes may be seen at as low a temperature as 24°C, especially if the humidity is high. This has led to the use of the temperature humidity index (THI), which is described by the equation:

$$\text{THI} = \text{temperature} + (0.36 \times \text{dew point}) + 41.2$$

Buildings can reach high temperatures in the middle of summer, especially if there is a high number of translucent roof sheets, effectively turning the building into a greenhouse. No more than 10% of the roof should be Perspex sheeting to avoid this effect. The situation is made worse by the heat produced by high-yielding cows. A 40-litre cow produces 1.7 kW heat per day, rising to 2.2 kW at 60 litres.

There are a range of clinical signs for heat stress, including panting, sweating, tail swishing and decreased feed intake. Cows get very dirty because they stand for longer periods and stand in groups, especially near water troughs, where they splash water with their tongues. This combination of damp, excess standing, close cow contact and dirty coats leads to increased mastitis.

A wide range of control options are available, all aimed at reducing the building temperature and increasing air flow. Removal of internal walls helps to improve air flow through the building, and Yorkshire boarding can be removed from around the outside of the building. Fans are of great value as they produce a cooling effect by increasing air movement. If humidity is not too high, misters will provide an additional cooling effect

Perspex roof sheets can be painted to reduce the greenhouse effect, and cubicle

occupancy rates should be reduced, e.g. by turning the 'lows' outside, maybe at night, and allowing the 'highs' to run into the 'lows' area. This will also allow the building to cool.

Other control options include planting trees to provide shade; the evaporation from their leaves also reduces air temperature. As food intakes will fall, maximize the palatability of the ration and provide an ample flow of cool water. In Florida, cows are allowed to walk into deep cooling ponds.

Cross-breeding, e.g. with Jersey or Brown Swiss, may help in the longer term

Establish a postcalving group

All cows undergo a period of immunosuppression during the periparturient period, rendering them more susceptible to a whole range of diseases, including mastitis. This was discussed in detail in Chapter 3 (pages 26–28). The immunosuppression will be even more pronounced if the cow is concurrently subjected to high levels of environmental stress from defects in housing, feeding or management. For this reason, many larger herds now have an immediate postcalving or maternity group of cows, which are retained in a more 'gentle' environment than the remainder of the herd. This can be done by keeping them in a small group, at a lower stocking density and perhaps in a straw yard for the first 1–2 weeks after calving and before introducing them to the main herd and to cubicles.

It has been found that this can increase yields and decrease lameness. Perhaps surprisingly, cubicle acceptance is improved when the change from straw yards to cubicles occurs. Of course, it is vitally important that this group should be retained in a clean and well-bedded yard, at a low stocking density, otherwise mastitis problems could be exacerbated. Predipping should definitely be carried out in this group, if it is not already in routine use.

The disadvantage of the system is that for the milker it means an additional group to bring into the parlour, and for the cow it introduces an additional group change.



Plate 8.28. If dry cows and heifers are being fed from a feeder, as in this case, make sure that the feeder is regularly moved so that the cows are not tempted to lie on contaminated ground.

Frequent changes of social grouping are stressful for the cow. It has been estimated that when a cow is introduced into a new social group she suffers ten aggressive interactions every hour for the first few days, i.e. 240 interactions every 24 hours.

Dry cow hygiene

The hygiene of dry cows is often overlooked. As discussed in Chapter 4 (pages 50–54), the critical periods are the first 2 weeks after drying off and the 2 weeks prior to calving. If more than one in 12 cows or heifers that are calving develop mastitis in the first 4 weeks of their lactation, or if cell counts are high (more than 15% above 200,000) in fresh-calved heifers, then this is said to indicate an environmental dry period infection. Control measures to be considered include improved environment (Plate 8.28), hygiene at dry cow tubing, use of internal teat sealants, reducing yields prior to drying off and minimizing teat-end damage. Environmental hygiene at these times is vital, and stocking densities should preferably be even lower than for milking cows. If at pasture, it has been recommended that cows close to calving should be moved to a clean paddock every 2 weeks and not returned to the same paddock for at least 4 weeks (Plate 8.28). This advice can pose considerable practical difficulties, however, because there are often only one or two well-drained fields near the parlour where close-to-calving cows can be carefully watched.

9 Somatic Cell Count

Why Cell Counts are Important	153
Financial penalties	153
Legal compliance	153
Reduction in milk yield	153
The suitability of milk for manufacturing or liquid milk consumption	154
Measurement of Cell Counts	154
Automated testing	154
DCC cell count tester	155
California Mastitis Test (CMT)	155
Agitation of the bulk tank before sampling	155
Factors that Affect Somatic Cell Counts	155
Mastitis	157
Type of mastitis organism	157
Age	157
Stage of lactation	157
Diurnal and seasonal and variations	158
Stress	158
Milking frequency	159
Day-to-day variations and management factors	159
Herd Somatic Cell Counts	159
Very low herd cell counts	160
Individual Cow Somatic Cell Counts (ICSCCs)	162
Interpretation and Use of Cell Count Data	163
Culture	163
Early dry cow therapy	163
Drying off an individual quarter	164
Treatment during lactation	164
Milking order	165
Culling	165
Withholding milk from the bulk supply	165
Evaluating treatment efficacy	166
Study Herd	166

This chapter describes why cell counts are important, how they can be measured, the factors that result in raised counts and action on high cell count cows, along with an example of how individual cell count data can be used.

The somatic cell count (SCC) is the number of cells present in milk ('body' cells as distinguished from invading bacterial cells). It is used as one indicator of udder infection. Somatic cells are made up of a combination of white blood cells and epithelial cells. White blood cells enter milk in response to inflammation, which may occur due to disease (see pages 27–28), or occasionally to injury. Epithelial cells are shed from the lining of the udder tissue. White blood cells make up the majority of the somatic cells, especially when the cell count is raised.

The SCC is quite a crude measurement and there are a variety of factors that will affect the result. In general, it is the contagious mastitis organisms that are responsible for high cell counts, as they make up the majority of subclinical infections. This is because the body continues to send in large numbers of white cells while attempting to remove this subclinical infection.

The SCC is measured in thousands of cells per ml of milk. The results are normally expressed in thousands to the farmer, e.g. a count of 250 refers to 250,000 cells per ml of milk. It is impossible to have a cell count of zero.

There is no reason why any dairy herd should not have a mean annual rolling herd cell count under 150,000. This means a low level of subclinical infection and minimal damage to the milk-producing tissues, thereby maximizing milk yield and ensuring the production of quality milk that will attract a premium price.

Why Cell Counts are Important

Financial penalties

All dairy companies in the UK have a payment system that penalizes farms with high

cell counts. High cell count milk has less yield for processing and the shelf life of liquid milk is reduced. The penalties vary and the majority of companies have an escalating scale of penalties once the average cell count exceeds 250,000. Many companies also offer 'bonus payments' if the cell count is under 200,000 or 250,000. Farmers respond positively to these payment schemes as there is a real financial incentive to produce low cell count milk.

Legal compliance

Most countries set a maximum cell count level above which milk cannot be collected off farm. In the EU, once the 3-month geometric mean cell count exceeds 400,000 for more than 3 months, milk cannot be collected off farm. In the USA, this threshold was 750,000 in 2009.

Nearly every dairy company now has some system of financial penalty that is imposed if the cell count or Bactoscan/TBC (total bacterial count; see Chapter 10) of bulk milk rises above a certain threshold. This is intended to ensure that the milk produced is of the highest quality. Farmers who do not meet these production standards are financially penalized according to the quality of their milk.

The level of penalty will depend on the use of the milk (cheese, liquid) and on supply. If there is a large supply pool, a cheese maker is likely to impose cell counts at a lower threshold to encourage quality milk for processing.

Reduction in milk yield

Most farmers are well aware that, as the herd cell count rises, there is a corresponding drop in milk yield. This occurs as a result of damage to the milk-producing tissue caused by mastitis bacteria and the toxins they produce.

One Canadian study showed that milk yield drops by 2.5% for every increase in cell count of 100,000 above a base figure of 200,000. This is shown in Fig. 9.1. For

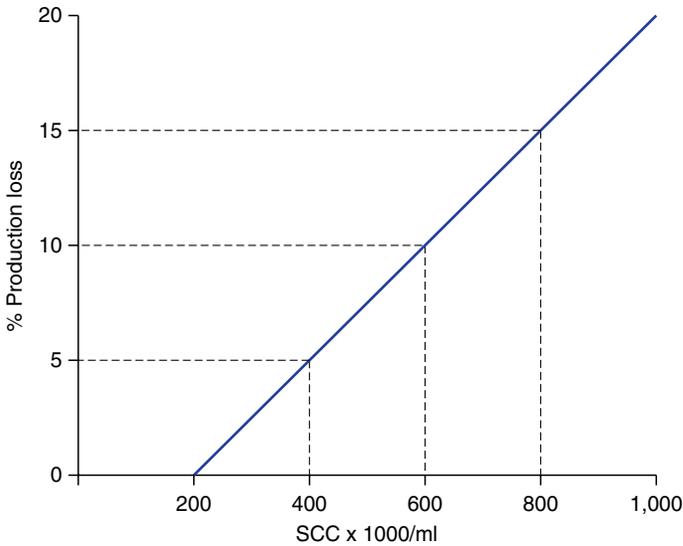


Fig. 9.1. Effect of herd cell count (SCC) on milk production: milk yield drops by 2.5% for every increase in cell count of 100,000 above a base figure of 200,000. (Adjusted from Philpot, 1984.)

example, a herd with an average yield of 7000 litres with a count of 360,000 can be expected to have a 4% loss in yield due to subclinical mastitis, or 280 litres per cow in lost production.

The suitability of milk for manufacturing or liquid milk consumption

The final and most important concern about high cell counts is the acceptability of milk to retailers and the manufacturing industry. It must be remembered that the quality of milk is only as good as when it leaves the farm. Poor-quality milk always remains poor-quality milk.

High cell count milk has a reduction in casein, lactose and calcium, and an increase in the enzymes plasmin and lipase (see page 17). Table 9.1 shows the compositional differences between low and high cell count milk.

Reduced casein levels result in a reduction of manufactured product. Reduced calcium levels result in poorer cheese clotting, higher fat losses and higher moisture retention. Increased plasmin results in lower yields of proteins and affects the stretch

properties in mozzarella cheese, gives a weaker body to yogurts and poorer water-binding properties. Plasmin withstands temperatures of pasteurization and continues to act in the final processed product. Lipase breaks down milk fat, resulting in rancid and off flavours.

Measurement of Cell Counts

Automated testing

The majority of laboratories use a Fossomatic cell counter, which can handle

Table 9.1. Effect of somatic cell count (SCC) on milk composition.

Constituent	Low SCC	High SCC	% of normal
Butterfat	3.90	3.90	100
Total protein	3.35	3.32	99
Casein	2.6	2.1	82
Whey protein	0.75	1.22	162
Lactose	4.6	4.2	90
Calcium	0.12	0.04	33
Sodium	0.057	0.105	184

large numbers of samples per hour. There are other automatic testing machines, including the Bentley. There will be a level of variation in the measurement of milk samples using either of these methods, with a difference of up to $\pm 5\%$. Bulk tank and individual cow samples are all tested using automated methods.

DCC cell count tester

The DCC (DeLaval Cell Counter) is a portable cell count tester that gives a numerical result. This allows accurate testing of individual cows, quarters or bulk milk on the farm. These portable DCC testers are used by dairy farmers, veterinarians and laboratories throughout the world. The test procedure is simple. Milk is loaded into the test cassette (one per sample) and then inserted into the DCC machine. After about 1 minute a numerical result is displayed. This is a very useful tool for managing milk quality at farm and cow level. Independent studies by the University of Wisconsin have shown that the DCC accurately measures cell count.

California Mastitis Test (CMT)

This is a simple test that is useful in detecting subclinical mastitis by crudely estimating the cell count of milk. The CMT test does not give a numerical result, but rather an indication whether the count is high or low. Any result over a trace reaction is regarded as suspicious. The benefits of the CMT are:

- It is a cheap test.
- It can be carried out by the milker during milking.
- Results are available immediately.
- It gives an indication of the level of infection of each quarter, as compared with an individual cow cell count, which only gives an overall udder result.

The test is carried out in the following manner (see Fig. 9.2):

- Foremilk is discarded.
- One or two squirts of milk from each quarter are drawn into the paddle dish.
- The paddle is tilted so that milk is discarded to a fixed volume per sample.
- An equal volume of reagent is added to the milk.
- This solution is then mixed and examined after 30 seconds for the presence of a gel reaction seen on the base of the paddle. The plate must be rinsed before going on to the next cow.

The disadvantages of the CMT include:

- Significant variation in results.
- Potential variation between operators.
- Changes are only seen at cell counts of 400,000 and over.
- No numeric result.
- Does not pick up all infected quarters.

The results can be scored into five categories ranging from negative, where the milk and reagent remain watery, up to the highest cell count, where the milk and reagent mixture almost solidifies. This is determined according to the gel reaction.

Agitation of the bulk tank before sampling

Somatic cells concentrate in butterfat and so the bulk tank must be agitated for at least 2 minutes before the milk is sampled to collect a representative sample. Otherwise, the cell count result may be higher. Edmondson found that the cell count in unagitated milk was 486,000 compared with 119,000 when the tank was agitated for two minutes.

Factors that Affect Somatic Cell Counts

When discussing cell counts, take care with definitions. For example, when referring to 'cell count' it might be:

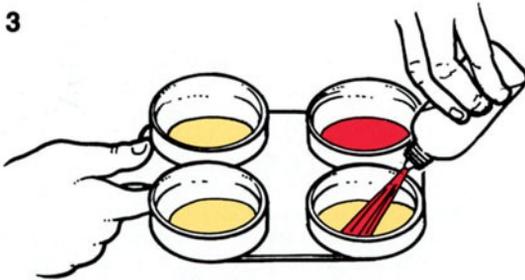
- The cell count of an individual cow or quarter at one sampling.
- The bulk tank cell count for that day.
- The mean bulk cell count over a 3- or 12-month period, etc.



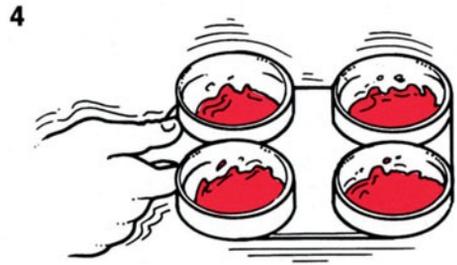
Foremilk is discarded and one or two squirts of milk are drawn from each quarter into a paddle dish.



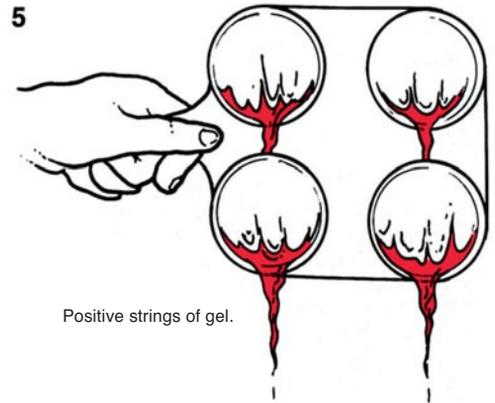
Excess milk is discarded.



An equal volume of CMT reagent is added to the milk.



The milk and the reagent are mixed.



Positive strings of gel.

Solutions are examined for the presence of a 'gel' or 'slime' reaction: gelatinous 'strings' indicate a high cell count quarter.

Fig. 9.2. How to carry out the California Mastitis Test (CMT).

The rest of this section refers to individual cow cell counts. It is then followed by a section on herd cell counts.

Mastitis

Mastitis is the major factor that causes increased cell counts. When mastitis organisms enter the udder, the defence mechanisms of the cow send vast numbers of white cells into the milk to try and kill bacteria (see pages 27–28). If the infection is eliminated, then the cell count will fall back to its normal level. If the white cells are unable to totally remove the organisms, then a subclinical infection is established. White cells are then continually secreted into milk, leading to a raised cell count.

Type of mastitis organism

Contagious bacteria (see pages 38–44) are much more likely to produce subclinical infections and therefore high bulk tank herd cell counts than environmental organisms. The exception to this is *Streptococcus uberis*. Infections caused by environmental organisms tend to be rapidly eliminated and the cell count normally only rises during the period of mastitis.

Different bacteria can produce different immune responses in the body. In addition, the same organism may produce differing responses in the same animal. In the case of acute *E. coli* infections, there is often a huge variation in response, as discussed on page 30. When the immune system responds well, there will be a massive increase in the number of white cells, for example, up to 20,000,000 per ml within 4 hours of *E. coli* invading the udder. In other instances, particularly in early-lactation cows, if the defence mechanism does not react, there is no increase in cell count and the cow will die no matter how she is treated. This is because the organism is free to multiply and produce toxins with no resistance from the cow.

For some organisms, there is a relationship between cell count and the level of

infection in the udder. For example, severe *Streptococcus agalactiae* infections may produce counts of up to 12,000,000 in infected quarters and the count correlates well with the level of infection. Other mastitis bacteria, particularly *Staphylococcus aureus*, which are shed in much lower numbers, produce a much more variable response, as shown in Table 4.5. There is no method of identifying the mastitis organism from the cell count of an individual quarter.

Age

In *S. aureus* problem herds, older cows tend to have higher cell counts. This is simply due to the increased period of exposure of the udder over previous lactations. The teat canals may be damaged, allowing easier access for bacteria to enter the mammary gland. Finally, the immune response from older animals may be effective.

Figure 9.3 shows the distribution of cell counts in a herd infected with *S. aureus*. This herd was divided into three groups: first lactation heifers, cows in lactation numbers two to four, and older cows in their fifth and subsequent lactation. Only 11% of the heifers had cell counts over 1,000,000, compared with 21% of the middle group and 46% of older cows in lactation five and upwards.

Freshly calved heifers tend to have cell counts in the range of 20,000 to 100,000, and in the absence of mastitis counts would remain at this level. When analysing individual cell count data, check the differences between age groups. If the older cows have higher cell counts, this suggests problems with *S. aureus*, which can be confirmed by bacteriology.

Stage of lactation

Cell counts are often high in the first 7 to 10 days after calving, although this may not occur in every cow. Towards the end of lactation, as the amount of milk produced reduces, cell counts may rise in animals that have subclinical mastitis. For example, a

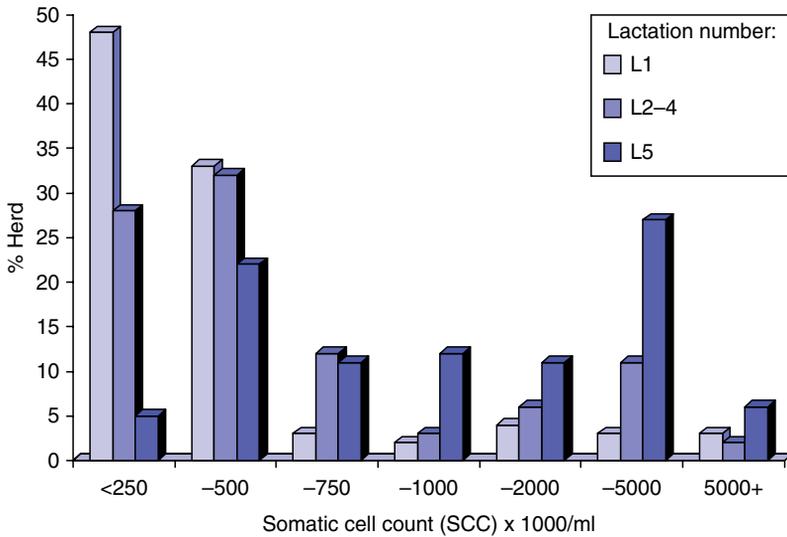


Fig. 9.3. Distribution of cell counts by lactation number in a *Staphylococcus aureus* infected herd.

cow producing 10,000,000 cells per day in 20 litres of milk will have a cell count of 500. If the same cow were only producing 5 litres, the cell count might increase significantly due to a concentration effect. This effect is very marked in animals yielding less than 5 litres of milk per day. Cell counts in cows that are free from subclinical infection do not alter significantly in late lactation.

Diurnal and seasonal variations

In herds that do not have regular milking intervals, cell counts tend to be higher in the afternoon milking than the morning milking. This is partly due to a shorter milking interval and lower milk yield resulting in a concentration effect. This can be seen in a herd that had separate tanks for morning and afternoon milk (see Table 9.2) and where there was a 14:10-hour milking interval. The results for a month were averaged out and show that the afternoon milk had a significantly higher cell count.

In grazing herds, counts can be higher in summer than in the winter, but the reason for this is not clearly understood. In seasonally calving herds with subclinical infection, there may be a rise in cell count when most

cows are towards the end of lactation. Figure 9.4 shows the monthly and the annual average cell count for an autumn-calving herd over a 4-year period. The monthly results fluctuate depending on the time of the year. In the summer, when most cows are in late lactation, the monthly cell counts rise, but they fall back in the winter.

Stress

Any event that causes stress, such as oestrus (bulling), sickness or events like tuberculin testing may affect the cell count of an individual animal. In addition to increasing the number of white cells in the blood, there is frequently a reduction in milk yield and this causes a further concentration effect. Stress will not be responsible for an increase in herd cell count.

Table 9.2. Variation in morning and afternoon cell count in herd with uneven milking interval.

Milking	Average cell count	Variation
Morning	147,000	±60,000
Afternoon	221,000	±70,000

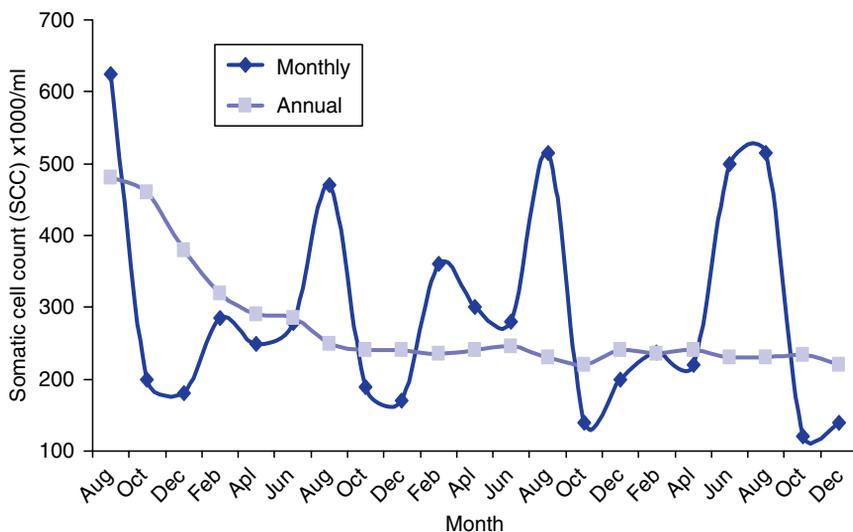


Fig. 9.4. Annual average and monthly bulk somatic tank cell count found in a seasonally calving herd.

Milking frequency

Some farmers reduce the frequency of milking to once daily or even every other day before drying off. Research work shows that cows milked intermittently towards the end of lactation will have dramatically increased cell counts.

In tests, the average cell count for a group of non-infected cows yielding over 5 litres per day was 237,000. When these cows were not milked for 2 days the cell count increased to 540,000. Stopping milking for an extra 4 days increased the cell count to 7,600,000, with some of the cows having counts as high as 15,000,000. These results clearly show that cows should be dried off abruptly.

The reason for this increase is that the milk (and bacteria) are not flushed out and so there is a significant increase in the number of bacteria and the cell count rises. This also explains why cell counts usually fall in herds that are milked three times a day.

Day-to-day variations and management factors

Cell counts vary from day to day. This is due to a variety of all the factors listed previ-

ously, together with management factors such as nutrition, calving patterns, sources of replacements and milking machine function. Research work has established that, as the level of vacuum reserve in a milking plant decreases, the herd cell count will increase. Hence, it is essential to ensure that the machine is well maintained to help keep cell counts low.

Herd Somatic Cell Counts

The factors listed above explain the variation in individual cow cell counts. Within a herd, many of these variations are averaged out. By far the greatest influence on herd cell count is the level of subclinical mastitis. As this rises, so does the cell count. A herd with a cell count under 200,000 will have little contagious mastitis present compared with a herd with a count over 500,000, which has a serious problem.

However, herd cell counts are not necessarily linked to the number of clinical cases, since this could be due to a high level of environmental mastitis, which will have little effect on cell count. In effect, clinical mastitis and subclinical mastitis (high cell count) are two separate conditions.

Farmers may receive a variety of different sorts of herd bulk tank cell count results:

- Individual tank results.
- Monthly figures.
- Three-month geometric rolling mean.
- Annual rolling mean count.

These may all give different results and trends that can be misleading. Specific results will vary greatly, depending on what is happening within the herd on that date. The more samples that are measured, the greater the amount of variation.

In herds with rising cell counts, two or three sets of low bulk tank results may suggest that the problem has disappeared. In some situations this may be the case, as the offending cow or cows may have been dried off or sold. In the majority of cases, however, it is just a temporary fall and will rise again.

Figure 9.5 shows the individual, monthly, 3-monthly and annual rolling mean bulk milk cell counts for a 150-cow herd over a period of 13 months. It can be seen that there is a high individual bulk result marked 'A' at the beginning of May. The next six individual results are significantly lower, and many farmers may consider that the problem has disappeared. However, it can be seen that over the following 3 months, all the cell count parameters increase, indicating that there has been an increase in subclinical infection and that result 'A' was not a one-off incident. It is essential to examine the cell count trend to see what is happening in the herd.

In this herd it can be seen that there was a significant improvement in herd cell count from August until December and that the monthly and 3-monthly average cell counts fell. The annual average hardly changed and reflects very slow changes in this cell count measurement parameter.

High herd cell counts can only be reduced over a short period of time by ruthless culling of the animals responsible for the increase, or by withholding milk from the bulk tank. However, in the long term, this is unlikely to solve the underlying mastitis problem. The dairy farmer who expects that he can reduce a cell count of 350,000 to

150,000 in a matter of a couple of months with minimal effort is likely to be disappointed, as infection is in the udder and can only be removed by culling, drying off cows or treating during lactation. The speed of decline in most cases will depend on:

- The type of infection present.
- The proportion of the herd infected.
- How well control measures have been implemented.
- Culling policy.
- Financial situation of the farmer.
- The willingness to follow recommendations.
- Action taken for individual problem cows.

Very low herd cell counts

Can cell counts get too low? The simple answer to this is no. At one time, it was felt that, if the herd cell count was too low, then cows would lose their ability to fight off infection that entered the udder and would therefore become more susceptible to environmental mastitis.

This is not the case. It is the speed of movement of the white cells into the milk, not the number of white blood cells present before infection occurs, that determines whether or not bacteria will be eliminated.

There are plenty of data available to show that herds with cell counts under 100,000 can have less clinical mastitis than herds with higher counts. Data from 11 herds in a Somerset veterinary practice with cell counts under 70,000 had a low mastitis rate of between seven and 21 clinical cases of mastitis per 100 cows per year. This is well below the target figure of 30 cases (see page 186). However, it is important to remember that some low cell count herds may have more clinical cases of mastitis than a herd with a high cell count. This would be due to a high level of environmental mastitis. Table 9.3 shows some hypothetical examples of this.

Herd A has good control of both contagious and environmental mastitis and so has an overall mastitis rate (cases per 100 cows per year) of 17, well below the target of 30.

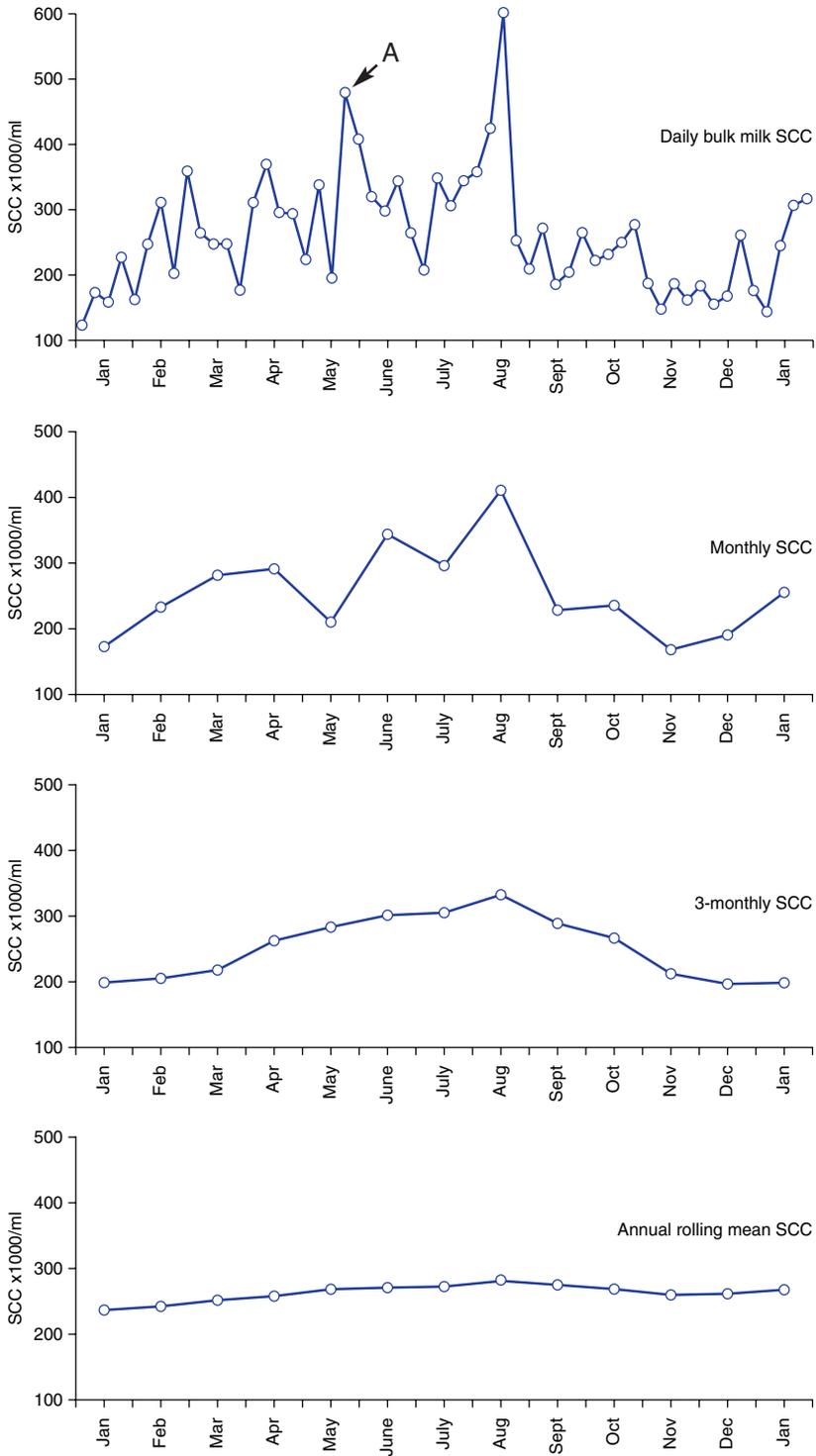


Fig. 9.5. Daily bulk milk, monthly, 3-monthly and annual rolling mean bulk milk cell count (SCC) over a 13-month period.

Table 9.3. The incidence of contagious and environmental clinical mastitis over a 12-month period in four herds with differing mastitis control and environmental management.

Herds ^a	A	B	C	D
Somatic cell count (× 1000/ml)	125	125	300	300
Control of contagious mastitis	Good	Good	Poor	Poor
Environmental mastitis management	Good	Poor	Good	Poor
Contagious cases	7	6	25	24
Environmental cases	10	43	4	42
Total mastitis cases	17	49	29	66

^aAll herds contained 100 cows.

Herd B has major problems with environmental mastitis, which could be due to dirty cows or a poor milking routine, etc., but still has good control of contagious mastitis. Herd B therefore has a high mastitis rate but a low cell count.

Herd C has a high cell count but no problems with environmental mastitis and so the mastitis rate is still below target levels, at 29. Herd D has problems with both types of mastitis and a mastitis rate of 66.

All a herd cell count tells us is that there is good control of contagious mastitis. The above examples show that there is no association between herd cell count and clinical case incidence. Herds can have a high or low cell count, and a high or low level of clinical mastitis. However, as most low cell count herds are well managed, the risk of environmental mastitis is often lower. It all comes down to attention to detail.

Individual Cow Somatic Cell Counts (ICSCCs)

Individual cow cell counts are the best way to identify high cell count cows. A count of over 200,000 indicates subclinical infection. Individual cell counts are calculated from a mixed sample from all four quarters. This may also be called a composite sample. Quarter cell counts refer to results from individual quarters. Samples for cell count testing need

not be collected in a sterile manner. However, lumps of faecal matter may cause problems with electronic testing, and foremilk should be discarded as it may have a higher count.

In herds where the cows are regularly sampled, individual cow cell counts are tested electronically and it can take time from collection to the results getting back, so when the farmer receives them they are historical data. This is to say that the results do not necessarily relate to current udder status.

In order to get the maximum benefit, cows should be sampled monthly so that trends can be studied rather than individual results only. A single high cell count indicates current infection status. However, subsequent tests may be low.

The danger of taking action on a single result has already been discussed earlier in this chapter. Many farmers with a high cell count have culled cows on the basis of a one-off screening of their herd, only to find that the herd count has remained unchanged. Culling should never be considered on the basis of a single cell count.

The big problem with individual cow results is that they do not identify which or how many of the quarters are infected or the level of any infection. This is shown in Table 9.4. From the composite result and interpretation guidelines shown in Table 9.4, we would expect Cow 2 to have no subclinical infection. The quarter results, however, show that there is significant infection present in the left hind quarter. Individual results and their interpretation from Cows 60 and 140 are correct.

Table 9.4. The effect of quarter and individual cow cell counts (× 1000/ml) in three cows.

	Cow 2	Cow 60	Cow 140
Individual cell count	139	314	582
Interpretation	Not infected	Infection present	Infection present
Quarter cell count results:			
LF (left fore)	20	600	425
RF	52	31	673
LH (left hind)	570	573	423
RH	33	51	807

Interpretation and Use of Cell Count Data

Individual cow cell count data need to be carefully analysed. For problem herds, the percentage contribution to the bulk tank is an important figure. In some herds, a small proportion of cows can make up a significant proportion of the bulk tank cells. Some farmers decide to cull these animals without seeing any long-term benefit. This is because these cows are symptoms of a subclinical infection and, if all that is being done is removing the symptom, then the disease will continue to spread throughout the herd.

Many farmers receive monthly cell count data but this information is not always used to maximum benefit. Looking at individual cell count data for the lactation is important. How many tests were over 200,000? Did this cow have problems in the previous lactation, in which case it may suggest a chronic infection such as *Staphylococcus aureus* or *Streptococcus uberis*. If it is the older cows that have the highest cell counts, then this suggests a problem with *Staphylococcus aureus* infection. If the end of lactation cows have the highest cell counts, then these cows can be dried off with antibiotic dry cow therapy to try and remove infection. If more than 15% of the herd have cell counts over 200,000, this suggests widespread subclinical infection.

Having accumulated and studied your cell count results over a period of 3–4 months, you need to know what action can be taken. There are a variety of options.

Culture

By sampling high cell count cows, the contagious mastitis organisms present in the herd can be identified and specific control measures implemented. Sterile samples must be collected carefully and submitted to the laboratory in the correct manner.

As there is no method of knowing how many or which quarters are infected from an individual cow cell count result, it is recommended to carry out a CMT and only col-

lect milk samples from high count quarters to maximize success.

Select a range of cows for sampling. Animals with persistently high counts should be chosen, and use a mix of young and old. It makes no difference if an animal is going to be culled or dried off, all that is needed is to identify the cause of infection.

Having identified the organism present, the control options for the high cell count are:

- Treatment.
- Dry off quarter.
- Dry off early.
- Culling.
- Milk last.

Early dry cow therapy

Early dry cow therapy should be considered if a high count cow is in late lactation. This will remove her milk from the bulk supply, which will have an immediate effect in reducing the herd cell count. It will also remove the risk of spreading infection to clean cows.

Unfortunately, dry cow therapy will not eliminate all infections. *Staphylococcus aureus* is the classic example (see page 210). If the infection is due to *Streptococcus agalactiae*, then dry cow therapy is very effective. However, there is a risk that cows with an extended dry period may become overfat, leading to calving problems and an increase in the number of metabolic problems during the next lactation.

The benefits of dry cow therapy in eliminating subclinical infection can be demonstrated using individual cell counts. In one experiment, 38 cows were sampled in the last 2 months before they were dried off, using dry cow therapy. They were resampled 14 days after calving. The results are shown in Fig. 9.6. Over 60% of cows had a cell count over 500,000 before drying off, compared with only 9% after calving, indicating that the dry cow therapy had removed the bulk of the subclinical infection at the end of lactation.

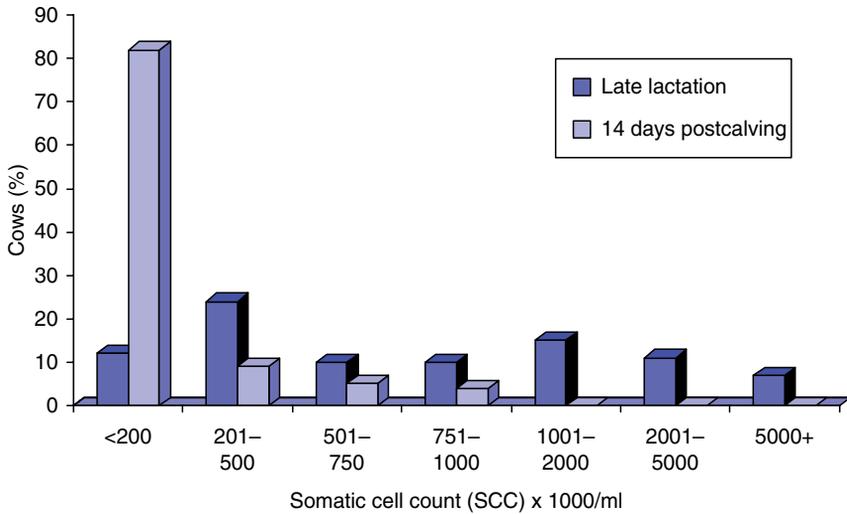


Fig. 9.6. Individual cow cell count found in late lactation and 14 days postcalving showing effect of dry cow therapy.

Drying off an individual quarter

Some farmers just dry off one infected quarter, which they identified by using the CMT test or individual quarter cell counts. It is advisable to carry out a CMT on the cow for three or four milkings to ensure that the correct infected quarters are identified. It is practical to dry off one quarter, but less so if two or more are infected. It is important that no dry cow antibiotic treatment is administered to this quarter, as it may lead to antibiotic failure. More details of drying off quarters and trial results are given in Chapter 12.

Treatment during lactation

Treatment of chronic subclinical *Staphylococcus aureus* mastitis during lactation is generally unsuccessful. This is because it tends to be chronic and well established. It is important to know which bacteria you are attempting to treat. Cure rates are very low with *S. aureus* infections (see pages 197, 205–206), often under 25% during lactation, and, when combined with the high treatment costs (intramammary antibiotics, discarded milk and extra labour), this line of action becomes very expensive. The only

time when treatment of *S. aureus* during lactation may be considered is with an exceptional cow and if the farmer is prepared to accept that this form of treatment may well be unsuccessful.

If the high cell count is due to *Streptococcus agalactiae*, therapy may certainly be worthwhile. Unfortunately, chronic infections due to *Streptococcus uberis* can also be very difficult to treat.

These considerations show the importance of bacteriology results to work out if treatment is a viable option, and what treatment regime should be used.

Some vets and farmers recommend the CMT to decide which quarter should be treated. This is a logical and responsible approach to treatment; however, two problems can be encountered. First, the CMT only gives a positive reaction once cell counts go over 400,000, whereas infection is present once the cell count is over 200,000. This means that not all infected quarters are identified and treated. Second, bacteria such as *Staphylococcus aureus* are shed intermittently and this means that cell counts of quarters can vary from milking to milking. The best success for treating high cell count cows comes from treating all four quarters with intramammary tubes and combining this with parenteral antibiotics. A prolonged

course of treatment will further maximize success.

Milking order

High cell count cows act as a reservoir of infection. Milking these animals last should help to reduce the spread of infection. In some herds, the big problem is the practicality of segregating these cows and keeping them separate. At best, it is difficult, if not impossible, in many herds. Research work shows that this can be a relatively effective method of reducing disease transmission.

In problem high cell count herds, forming a high cell count group that is milked last reduces the transfer of infection. Once a cow enters this group, she should remain there until the end of her lactation or until the cell count drops for two consecutive months. These groups may be used when it is practical, e.g. during housing, or as a short-term measure to assist control of disease. It is possible to disinfect the cluster after milking high cell count cows to reduce transfer. If herds are grouped then the milking order is important.

Culling

This is a method of eliminating problem cows permanently, but it is costly due to the marked difference between the sale value of a cull cow and replacement costs. In addition, if the cull cow is replaced with a cow purchased at market, you may end up back where you started – with another high cell count animal. Few cows are sold through the market because they give too much milk, have persistently low cell counts or have never had a case of mastitis.

Culling should **never** be based on cell count data in isolation. Factors such as the type of infection present should always be considered. For example, if a high cell count is due to *Streptococcus agalactiae*, then treatment will be successful in reducing the count. In this case, culling these cows would be a very expensive way of reducing

the herd cell count. Of course, if infection is due to *Staphylococcus aureus*, and the cow has a chronic infection and is contributing a high percentage of cells to the bulk tank, then culling is a sound course of action.

Cows with persistently high cell counts for three or more consecutive tests might be considered for culling but it is essential to take other factors into account. These include:

- Percent contribution to the bulk tank.
- Bacteriology results.
- The herd cell count and financial penalties.
- The number of cows in this category.
- The number of cases of mastitis that each animal has had.
- Milk yield.
- Fertility status.
- General health.
- Source of replacements.

Before deciding to cull any cow, the other options such as treatment, drying off early or drying off the infected quarter should be carefully considered.

Withholding milk from the bulk supply

The herd cell count can be reduced by withholding milk from high cell count cows from the bulk supply. This will have an immediate effect in improving the situation and allows the farmer time to consider which line of action he wishes to take, but it is a costly option. In herds that are over quota limits, this temporary line of action can prove invaluable.

Some farmers will then feed this milk to calves. This is a controversial course of action – some suggest that mastitic milk may cause infection of the immature udder. Mastitis organisms may gain entry to the udder either by calves sucking the teats of other calves or they may be spread by flies. For this reason, some people recommend only feeding this mastitic milk to bull calves.

Evaluating treatment efficacy

Cows with mastitis will have increased cell counts. These counts will decline after successful treatment. In cases where there has been a bacteriological cure, i.e. all the bacteria have been eliminated from the udder, then cell counts would be expected to decline to below 400,000, but this may take up to 4 to 6 weeks. If infection remains present and becomes subclinical, then counts will remain elevated.

Study Herd

Table 9.5 shows individual cow cell count results for a herd of 140 cows that records with National Milk Records in the UK. These data have been imported into the INTERHERD computer program, which allows in-depth analysis.

These data show that the current month's average cell count was 396,000 and that the rolling annual average for the herd was 305,000. A cell count of 396,000 indicates very high levels of subclinical mastitis, and the farmer is losing 10% of his milk price in cell count penalties. The annual average cell count changes very slowly, but, if it is increasing, it suggests that the problem is deteriorating. The cell count figure of 396,000 is from monthly milk-recording records, which will differ from those of the milk buyer, who will be testing bulk tank more frequently.

The lactation breakdown for August shows that the 29 heifers had an average cell count of 115,000 which suggests that they are free of infection. However, the result for the 14 cows in lactation 4 shows an average cell count of over a million, indicating that this group of cows have severe levels of infection.

Table 9.5. Summary of somatic cell count results.

Rolling annual average of whole herd ^a		305							
This month's herd average ^a		396							
				Average test for:	3 Jun	3 Jul	3 Aug	Total Cows – Aug	
1st lactation				277	137	115	29		
2nd lactation				725	445	377	28		
3rd lactation				191	189	224	21		
4th lactation				140	886	1002	14		
5th lactation				191	763	566	21		

Cow no.	Lact. ^b	Days in milk	Lact. avg.	No.>200 ^c	1 May	2 Jun	3 Jul	3 Aug	% of total
1385	4	262	1012	7	498	90	682	9720	14
0651	2	16	3631	1	795			3631	9
0331	7	201	474	1	60	18	102	3578	9
0678	4	13	3414	1				3414	4
0318	7	153	829	4	485	218	1903	2021	5
0258	6	140	1360	5	384	645	833	1911	4
0016	4	291	452	2		50	4120	1472	3
0117	6	404	92	3	74	59	241	1329	0
0338	6	66	1710	2			2079	1252	3
1247	3	77	557	2	345	145	411	1244	2
0101	4	18	1152	1				1152	3
0477	4	211	480	5	780	5	761	1021	2
0612	2	362	201	7	412	290	423	1013	1
0255	6	308	1054	8	3534	69	1299	875	2
0028	3	334	607	6	74	90	237	836	1

^aSomatic cell count (SCC) × 1000/ml; ^bLactation; ^cNumber of SCCs > 200.

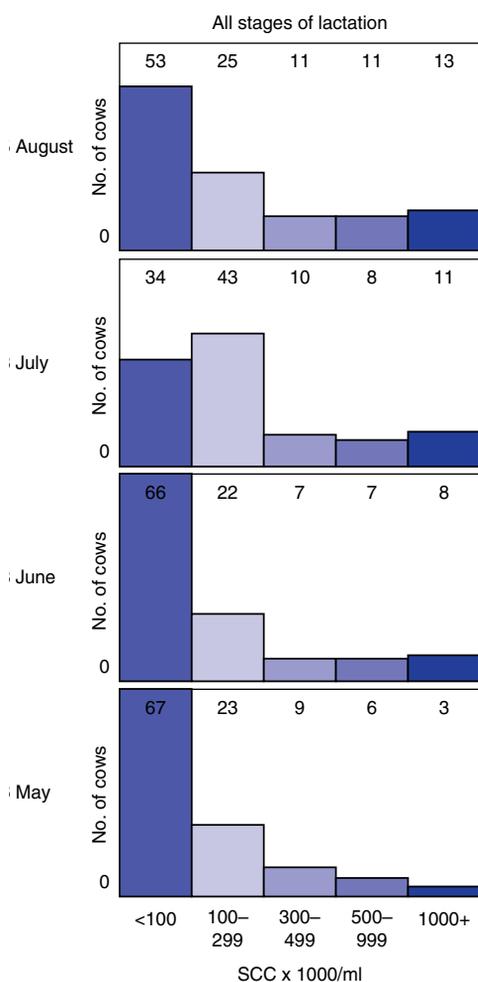


Fig. 9.7. Individual cow somatic cell count (SCC) distribution over past four milk recordings from cows at all stages of lactation.

Below the lactation summary there is a list showing the cows with the highest contribution to the bulk tank for August. Percentage contribution to the bulk tank will be a combination of cell count and yield. On the day of recording, there were 113 cows in milk. The first six cows, or 5% of milkers, accounted for 45% of the somatic cells. There are another three cows in the list that account for a further 9% of somatic cells. This is a prime example of a herd where on the day of sampling a small proportion of cows (8%) accounts for a large percentage (54%) of the bulk tank cell count.

Figure 9.7 shows the distribution of cell count within the herd for the past four milk recordings. This shows that at the last recording there were 13 cows with cell counts over a million, 11 with counts between 500,000 and a million, 11 with counts between 300,000 and 500,000, and 53 with cell counts of under 100,000. Thus, 31% of the herd have cell counts of over 300,000, indicating widespread subclinical infection within the herd.

Table 9.6 shows the cell count by stage of lactation. For August, the average cell count for the first 100 days of lactation is 398,000, from 100 to 199 days it is 324,000, and it is 428,000 for cows calved more than 200 days. If the cell count were high in the end of the lactation group, drying off these cows would be an easy way of helping to reduce the cell count.

This herd has had no samples collected for bacteriology and so ten of the highest contributing cows to the bulk tank should be selected and submitted for culture, along with a sample of bulk milk. This will help to establish the cause of the high cell count.

A farm visit needs to be carried out to assess mastitis management. During this visit, control measures can be tightened up and any measures or products that are used and have no benefit can be stopped. Tackling high cell count cows without addressing the spread of infection in the herd will not result in long-term benefits. Once this step has been carried out, action on individual cows can be taken.

Now let us consider the individual cow results from the cows contributing the highest percentage contribution to the bulk tank and the possible actions that could be taken. These cows will not necessarily have the highest cell counts, but, as we need to reduce the herd cell count, the percentage contribution is the key area to examine.

Cow 1385

This is a lactation 4 cow, contributing 14% of cells to the bulk tank, calved 262 days and giving 15 litres. She is pregnant and so can be dried off. An alternative is to dry off the

Table 9.6. Individual cow cell count average by days in milk.

Days in milk	No.	2 Jun	Cell count	3 Jul	Cell count	Days pp	3 Aug	Cell count
<100	29	37.92 kg	124	34.43 kg	289	49	31.73 kg	398
100–199	27	33.80 kg	511	29.45 kg	516	168	26.47 kg	324
>199	57	30.94 kg	284	24.63 kg	351	281	22.17 kg	428

quarter (DOQ) if only one quarter is CMT-positive. This is discussed in Chapter 12. Drying off the cow or drying off a quarter removes her milk from the bulk tank and helps to protect the rest of the herd.

Cow 651

This is a lactation 2 animal contributing 9% of herd cells and calved only 16 days. Her last cell count in the last lactation was 795 (1 May recording); this suggests that dry cow therapy (if given) was ineffective. It is important to check that this cow does not have clinical mastitis and, if she does, that she is treated. If not, she should be CMT-tested to see if she still has a high cell count. The CMT result for such a high cell count will be very obvious. If the test reading is high, as a short-term measure, this milk should be fed to bull calves and a sterile milk sample taken to identify the cause of the infection, to help decide what action should be taken.

Cow 331

This cow is in lactation 7 and is calved 201 days. She has had one cell count over 200,000 in this lactation. Her last cell count was 3,578,000 and she was contributing 9% to the bulk tank. Her lactation average is 474,000.

Table 9.7 shows her previous cell count history. While this is an old cow, she has only had one high cell count reading over 200,000 in her lifetime. A jump from 102,000 up to almost 4 million suggests that this cow may have been developing or had clinical mastitis at the recording that was missed. On questioning the farmer, this cow

was found to have mastitis the day after recording. If clinical mastitis treatment has been successful, her cell count should reduce at the next recording.

Cow 678

This is a lactation 4 cow, calved 13 days and contributing 4% of all cells. In her previous lactations, the cell count averages were 96,000 and 121,000 with no readings over 200,000. Despite her age, this cow does not have chronic infection and the advice is the same as for cow 651. It is possible that this reading is due to her being freshly calved.

Cow 318

This is a lactation 7 cow, calved 153 days, with a cell count of over 2 million contributing 5% of all somatic cells. This cow has had high cell counts for the previous two lactations, 911,000 and over 2 million, and is clearly a chronic high cell count cow that should be culled from the herd now.

Cow 258

This cow is in lactation 6 and is calved 140 days. She has had five readings over 200,000 this lactation; her last cell count was 1,911,000 and was contributing 4% to the bulk tank. Her lactation average is 1,360,000. It is very helpful to look at the previous history of this cow to make sound management decisions.

Table 9.8 shows her history for the previous lactations. It can be seen that her average cell counts in lactations 4 and 5 were

Table 9.7. Individual cell count history of cow 331.

Identity details		Parity data and events		Origin and movements		Parents and offspring		Performance statistics			Exit details				
Parity record summary															
N	Calving	NS	Last service	Conception	Int.	Lact.	305-d.	Fat	Prot.	Lact.	SCC	Costs	No. Mast	No. Lame	SCC high
0		1	11/12/2000...669	12/12/2000...670	950								0	0	0
1	18/09/2001	1	03/12/2001...76	07/12/2001...80	360	6,510	6,332	4.35	3.54	4.67	27		0	0	0
2	13/09/2002	1	12/12/2002...90	19/12/2002...97	377	9,011	8,802	4.31	3.39	4.50	52		0	0	0
3	25/09/2003	2	24/12/2003...90	31/12/2003...97	377	9,478	9,328	4.28	3.42	4.46	48		0	0	0
4	06/10/2004	2	30/03/2005...175	08/04/2005...184	464	11,732	10,474	4.33	3.41	4.34	75		0	0	3
5	13/01/2006	1	30/03/2006...76	02/04/2006...79	359	10,518	10,518	4.29	3.10	4.35	51		0	0	0
6	07/01/2007	1	09/04/2007...92	12/04/2007...95	375	9,605	9,315	4.78	3.24	4.25	116		0	0	0
7	17/01/2008	4	23/06/2008...158			9,708	9,708	4.32	3.15	4.48	474		0	0	1
Milk recordings for parity 7															
N	Date	Days	Day kg	Fat %	Prot %	Lac %	SCC '000	Conc.							
1	05/02/2008	19	39.8	5.11	2.95	4.40	110								
2	06/03/2008	49	42.6	4.70	3.22	4.51	158								
3	04/04/2008	78	35.0	3.80	3.20	4.53	82								
4	06/05/2008	110	38.1	3.87	3.12	4.50	60								
5	03/06/2008	138	36.9	3.11	2.99	4.90	18								
6	03/07/2008	168	31.7	5.84	3.17	4.22	102								
7	05/08/2008	201	27.7	3.71	3.49	4.19	3578								

531,000 and 723,000. In addition, she has 14 results over 200,000 in lactation 4 and 13 in lactation 5. This cow had received antibiotic dry cow therapy at the end of every lactation. This cell count history indicates that the cow most probably has a chronic sub-clinical infection, such as *Staphylococcus aureus* or *Streptococcus uberis*, which is not responding to treatment. This cow should be culled from the herd. As this herd has a very

high cell count, culling this cow and cow no. 318 should take place as soon as possible as they account for 9% of all somatic cells.

If this cow were in a herd where the cell count was low and the farmer was not being penalized, the cow should still be culled. However, the timing of culling can change. She may not be culled until the end of lactation as it is clear that herd mastitis control measures are effective due to the low herd

Table 9.8. Individual cell count history of cow 258.

Identity details		Parity data and events		Origin and movements		Parents and offspring		Performance statistics			Exit details				
Parity record summary															
N	Calving	NS	Last service	Conception	Int.	Lact.	305-d.	Fat	Prot.	Lact.	SCC	Costs	No. Mast	No. Lame	SCC high
0		1	27/11/2000...886	27/11/2000...886	166								0	0	0
1	03/09/2001	1	25/11/2001...83	21/11/2001...79	359	7,107	6,807	4.68	3.91	4.82	32		0	0	0
2	28/08/2002	2	03/01/2003...128	10/01/2003...135	415	9,345	8,851	4.38	3.67	4.60	66		0	0	0
3	17/10/2003	2	19/02/2004...125	15/02/2004...121	401	9,302	8,840	4.40	3.70	4.67	57		0	0	0
4	21/11/2004	3	06/01/2006...411	15/01/2006...420	700	15,102	11,304	4.46	3.71	4.35	531		0	0	14
5	22/10/2006	1	24/06/2007...245	12/06/2007...233	513	12,707	10,313	4.63	3.44	4.33	723		0	0	13
6	18/03/2008					7,830	7,830	4.40	3.16	4.33	1,360		0	0	5
Milk recordings for parity 6															
N	Date	Days	Day kg	Fat %	Prot %	Lac %	SCC '000	Conc.							
1	04/04/2008	17	25.5	4.86	3.44	4.15	3812								
2	06/05/2008	49	34.3	4.76	2.70	4.59	384								
3	03/06/2008	77	36.9	3.52	3.16	4.22	645								
4	03/07/2008	107	32.7	4.62	3.39	4.37	833								
5	05/08/2008	140	26.5	4.45	3.23	4.26	1911								

cell count. She would pose a lower risk to other cows because hygiene is good. An alternative would be to dry off the quarter.

These results show that the interpretation of individual cell counts requires careful consideration, and action can only be taken on a series of results, not on the basis of a one-off screening.

10 Bactoscan and Total Bacterial Count (TBC)

The Three Sources of Bacteria in Milk	172
Mastitis organisms	172
Environmental contamination	173
Dirty milking equipment	174
Failure of Refrigeration	174
Bulk Tank Analysis (BTA)	176
Common tests carried out on bulk milk	176
When to use bulk tank analysis	178
Interpretation of bulk samples	179
Examples of the use of BTA in problem herds	180

This chapter describes the sources of bacteria in milk, compares TBC testing with Bactoscan and shows how bulk tank analysis of milk can help to identify the cause of high Bactoscan counts.

A high Bactoscan or TBC can affect the dairy farmer in two ways: directly in the form of financial penalties and the possibility of increased levels of mastitis, and indirectly through the production of a poor-quality, short shelf life milk that is less acceptable to the consumer and manufacturer. Some bacteria can cause 'off' flavours, due to increased levels of the enzymes plasmin and lipase, which break down casein and butterfat. Some producers believe that pasteurization will not only kill all the bacteria present in milk, but will also put right any milk quality problems. This is not true.

The total bacterial count (TBC) of milk is a measure of the number of bacteria grown

using a specific culture medium and a specified temperature, and over a fixed period of time. It is sometimes referred to as the total viable count (TVC).

The Bactoscan test measures the total number of bacteria using an electronic method. The test takes 10 minutes compared with 72 hours for the TBC. The Bactoscan is far more accurate and measures all bacteria (dead and live) rather than counting living colony-forming units (cfu). It counts all bacteria, irrespective of their culture medium and temperature requirements and, as such, measures psychrotrophs (bacteria that grow under refrigerated conditions) which are not picked up by the TBC test. Bactoscan testing is a far more accurate and reliable measure of the bacterial count in milk.

For both testing methods, the results are given as the total number of bacteria per ml of milk. For simplification, the results are commonly reported back to the farmer in

thousands, and so a count of 9 refers to a TBC or Bactoscan of 9000 per ml.

The majority of milk buyers have replaced the TBC with the Bactoscan test. A comparison of the two tests is shown in Table 10.1. There is no precise numerical correlation between the two tests. An added advantage of the Bactoscan is that the farmer can be notified of a high result quickly so that he can take action.

The 1992 EC Council Directive 92/46/EEC for dairy products required bacterial levels to be under 100,000/ml. It must be remembered that most dairy companies are only interested in purchasing 'quality' milk. A herd with good hygiene will have a Bactoscan of 20,000 or less. There are many herds that have counts of around 10,000 and these are often herds that predip and have meticulous parlour hygiene, as well as excellent wash-up routines, as seen in Plate 10.1. Dairy companies measure Bactoscans regularly.

However, the benefits of Bactoscan testing to the farmer are limited as results do not identify the bacteria present or the source of the organisms. Despite this, the Bactoscan remains an accurate test for measuring the number of bacteria in milk. With further testing, bulk tank samples can yield valuable information in relation to mastitis management, as described later in this chapter.

Bacterial contamination of milk may occur in two ways: directly from the cow when mastitis organisms are shed into the milk, or indirectly from the environment or milking equipment.

The Three Sources of Bacteria in Milk

There are three main causes of high Bactoscans. These are:



Plate 10.1. A good wash-up routine will ensure that farms maintain a low Bactoscan.

- Mastitis organisms.
- Environmental contaminants.
- Dirty milking equipment.

Mastitis organisms

Mastitis organisms should be suspected if the Bactoscan fluctuates dramatically. Milk from a healthy quarter will have low numbers of bacteria present, usually under 1000 per ml. When quarters become infected with clinical or subclinical mastitis, the numbers of bacteria can increase substantially. *Streptococcus agalactiae* and *Streptococcus uberis* are shed in extremely high numbers, for example, up to 100,000,000 per ml, in clinically infected quarters. Large numbers of coliforms may also be shed with *E. coli* mastitis. In herds infected with these organisms, it is easy to understand why Bactoscan levels can fluctuate.

Take a herd producing 1500 litres of milk per day with an average Bactoscan of

Table 10.1. Comparison of TBC and Bactoscan.

	Bactoscan	TBC
Measures	All bacteria	Colony-forming units
Time	10 minutes	72 hours
Accuracy	± 10%	± 30–50%
Psychrotroph measurement	Yes	No
Correlation	Approx. 4–5 × TBC	

5000. The addition of as little as 2 litres of mastitic milk from a clinical *S. uberis* cow (shedding 100 million bacteria per ml) can increase the bulk tank Bactoscan to 138,000. For this reason, it is important to detect clinical infections early so that mastitic milk does not enter the bulk supply.

Figure 10.1 shows the typical fluctuating effect of an *S. agalactiae* infection on the Bactoscan in a herd of 50 cows. Other mastitis organisms, for example, staphylococci, tend not to shed bacteria in large enough numbers to significantly affect the bulk tank Bactoscan (see Table 4.5).

Unfortunately, the milker cannot detect subclinical mastitis and so it is inevitable that some mastitis bacteria will enter the bulk supply. The best way to reduce this effect is through a mastitis control programme that will reduce the level of infection in the herd, and ultimately the number of mastitic organisms entering the milk.

Environmental contamination

The main cause of environmental contamination of milk is keeping cows in poor environmental conditions, combined with inadequate teat preparation. The importance

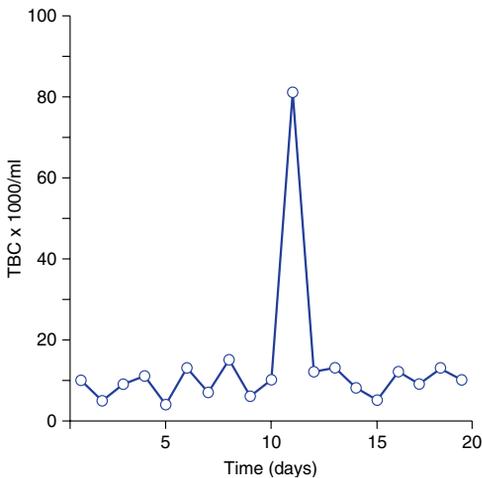


Fig. 10.1. The typical effect that a single clinical case of *Streptococcus agalactiae* mastitis can have on the bulk TBC in a herd of 50 cows.

of good udder preparation has been referred to in Chapter 6. It is essential that the milking unit is attached to clean dry teats. Milking dirty teats will not only contaminate the bulk milk but must also increase the likelihood of environmental mastitis.

The coliform count measures the number of coliform organisms in milk and gives an indication of the level of environmental contamination and the standard of premilking preparation. Coliforms are only one group of environmental organisms, of which the most important is *E. coli*, but there are many others, such as *Streptococcus uberis*, *Streptococcus faecalis*, *Bacillus* species, etc.

The technique for carrying out a coliform count is described on page 59. The target figure with good milking hygiene is to have a coliform count under 10 per ml, but levels under 20 per ml are acceptable. High levels of environmental bacteria will reduce the shelf life of milk and increase the risk of off flavours and hence its acceptability to processors. There are many herds with excellent premilking preparation that regularly have coliform counts of 5 per ml or less.

Table 10.2 shows a comparison of the TBC and coliform count before and after a change in the milking routine in a herd of 1000 dairy cows in Arizona. Initially, teats were washed but not dried, which resulted in high TBCs and coliform counts. Once the milking routine was modified and teats were washed and then dried before milking, the counts reduced significantly. Remember the coliform count will not measure all environmental organisms. It just gives an indication of whether the level of environmental contamination in milk is high or low.

Table 10.2. The effect of different types of teat preparation on the TBC and coliform count/ml of milk. (From T. Fuhrmann, unpublished data, personal communication.)

Test	Washing but not drying: 3 months before change in routine	Washing and drying: 3 months after change in routine
TBC	50,000	10,000
Coliform count	120	20

The type of winter bedding used is also important: sawdust and wood shavings become rapidly contaminated with bacteria just 24 hours after bedding down – this is due to their large surface area and their ability to absorb moisture. Sand is inert and does not support bacterial growth.

In well-run dairy herds where there is plenty of clean, dry and well-bedded accommodation, teats should remain clean. In poorly managed herds where there is insufficient or dirty, damp accommodation, for example, where there are uncomfortable cubicles resulting in cows lying outside, the condition of the teats of the cows entering the parlour will be poor.

A practical way to assess premilking teat preparation on farm is to check the milk socks or filters after milking. If they are dirty, this is likely to contribute to high coliform counts. Cows should come in to be milked with clean udders and teats. Hairy udders and long tails can increase the risk of dirty cows. Check inside the liners during milking; they should be free of faecal contamination.

Dirty milking equipment

Inadequately cleaned milking equipment can lead to raised Bactoscans, as seen in Plate 10.2. A laboratory assessment of plant cleaning can be made using the laboratory pasteurized count (LPC) or thermoduric (TD) count, where levels over 175 cfu/ml suggest a wash-up problem.

Milkers should look out for the following, which can cause contamination of the bulk milk:

- Wash-up problems, see pages 88–89.
- Dirty bulk tank (see Plate 10.3): it should be inspected after every wash.

Failure of Refrigeration

Milk should be cooled to 4°C or less as soon as possible after milking to limit the growth of bacteria. This helps to maintain milk quality. In the UK, milk cannot legally be collected off farm if it is over 6°C. When there



Plate 10.2. A dirty receiver vessel will increase the Bactoscan.

is a refrigeration problem and milk is not kept cool or not cooled rapidly, bacterial multiplication will take place.

The importance of efficient refrigeration is becoming greater with a decreasing frequency of milk collection from some farms. In some countries, milk for liquid milk consumption is collected every second day, while milk for manufacturing can be collected every third day, without any significant effect on Bactoscan or milk quality provided the refrigeration is efficient and there are good hygiene and management practices on farm.



Plate 10.3. Soil, as shown in the dark line just above the milk, allows psychrotrophic bacteria to grow.

The effect of multiplication will depend on the number and type of bacteria, together with the temperature of the milk. Warm milk is an excellent medium for bacterial growth. Some bacteria, such as coliforms, may double in number every 20 minutes under optimum conditions. The increase in bacterial numbers in raw milk stored at different temperatures over a 12-hour period is shown in Fig. 10.2. These data refer to ‘clean’ milk, i.e. milk without excess environmental contamination. As temperature increases above 4.5°C, the rate of bacterial growth increases exponentially.

Plate coolers are commonly used to cool milk quickly and slow down bacterial multiplication, while saving energy. They operate by using a heat exchange mechanism. Large volumes of cold running water (as much as seven times that of the milk) flow in the opposite direction to milk with the heat from the milk passing through to warm the water, as shown in Fig. 10.3. Some plate coolers circulate chilled water from the bulk tank, which drops the temperature further. The resultant effect of the heat exchange mechanism (with the most efficient systems) is to have milk leaving the cooler at a temperature as low as 6°C. Tube coolers (tubes surrounding the



Plate 10.4. Plate coolers drop the temperature of milk quickly and slow down bacterial multiplication, while saving energy.

milk line through which cold water flows in the opposite direction to the milk) have the same effect.

The warm water from the plate coolers may be used to wash dirty teats before milking. Others divert this water to a drinking trough so that cows can have a warm drink after milking. Cooling milk before it enters the bulk tank saves energy as the tank has less work to do. It also helps to protect milk quality, as the milk reaches 4°C more rapidly, thus reducing bacterial multiplication.

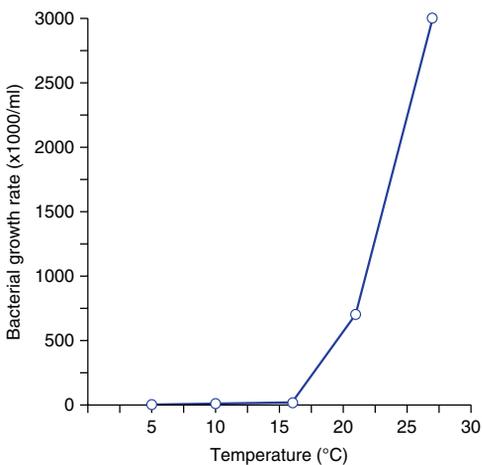


Fig. 10.2. Effect of storage temperature on bacterial growth in raw milk over a 12-hour period. At 21°C there is a 700-fold increase in bacterial numbers over a 12-hour period. (From Philpot and Nickerson, 1991.)

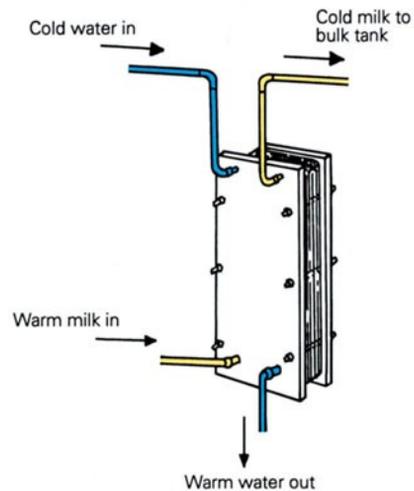


Fig. 10.3. Plate cooler. Large volumes of cold water run through the plate cooler in the opposite direction to the milk, resulting in warm water, but cool milk leaving the plate cooler.

The shelf life of pasteurized milk is considerably affected by storage temperature, as shown in Fig. 10.4. This is of great importance to retailers, the milkman and, of course, the consumer. It can be seen that when pasteurized milk is stored at 16°C its shelf life is only 1 day, compared with 10 days when stored at 5°C.

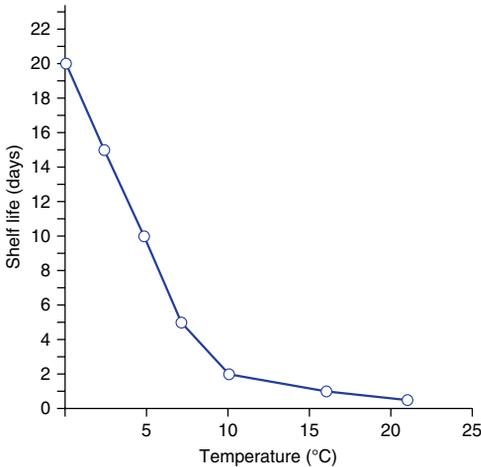


Fig. 10.4. Shelf life of pasteurized milk in days according to the storage temperature. (From Philpot and Nickerson, 1991.)

Bulk Tank Analysis (BTA)

Bulk tank analysis can offer a variety of tests that can be carried out, along with a differential bacterial screen. All bacteria in milk have originated from either the udder, the environment or a dirty plant, and common tests for these are described in the next section.

Common tests carried out on bulk milk

The somatic cell count (SCC) gives an indication of the level of subclinical mastitis.

The TBC (total bacterial count) gives a quantitative indication of bacteria in milk and, while it is not as accurate as a Bactoscan test, it gives an indication of the total numbers of bacteria in milk.

The LPC (laboratory pasteurized count), often called a (TD) thermoduric count, measures thermoduric bacteria, which withstand high temperatures of pasteurization. High counts indicate problems with plant washing. Because thermoduric bacteria can withstand the temperatures of pasteurization, they can continue to grow in the milking system if the wash-up routine does not remove them.

The coliform count (CC) gives an indication of faecal and environmental contamination, which is due to poor teat preparation or poor hygiene. Coliforms act as a marker for all the other environmental organisms, such as faecal streptococci, yeasts and fungi. Increased coliforms can also arise from mastitis.

The *Pseudomonads* count gives another indication of environmental contamination, although the source of these organisms is very different. Some *Pseudomonads* bacteria are psychrotrophs and so multiply in cold conditions.

Streptococcus uberis, *Staphylococcus aureus* and the total staphylococci counts give a measure of these individual bacteria. The total staphylococci count measures all staphylococci, including *S. aureus*. These counts are useful in herds with contagious mastitis problems. *Streptococcus uberis* is predominantly an environmental bacterium associated with the use of straw bedding and is a common cause of clinical and sub-clinical mastitis in the UK and Europe.

A differential bacteria screen identifies all bacteria in milk, no matter what their origin. Some laboratories quantify these as + for small numbers, up to +++ for high levels. In the UK, screening for *Mycoplasma* is not routinely carried out, as this is a rare mastitis pathogen. In parts of the world where this is a problem, it should be a regular part of the bulk tank screening.

The presence of each type of bacterium in milk each has its own individual significance.

- *Streptococcus agalactiae* is a highly contagious mastitis bacterium that can result in very high cell counts.

- *Streptococcus dysgalactiae* is associated with poor teat skin condition and possible milking machine problems linked to teat damage and/or injury.
- *Corynebacterium bovis* is associated with poor postmilking teat disinfection.
- *Pseudomonas aeruginosa* is associated with contaminated water sources and can cause a severe mastitis.
- *Klebsiella* is associated with wood products, such as sawdust, and can cause clinical mastitis.
- Yeasts and fungi are associated with poor hygiene, as is the presence of *S. faecalis*.

Table 10.3 shows the target levels for the different test types for bulk tank analysis from one laboratory. There are many herds that are consistently under these targets.

Table 10.3. Targets for bulk tank analysis. Dairy farmers who have a good mastitis management regularly achieve these targets.

Somatic cell count	<150,000
Total bacterial count	<5,000
Laboratory pasteurized count	<175
Coliform count	<20
<i>Pseudomonads</i> count	<500
<i>Streptococcus uberis</i> count	<200
Total <i>staphylococci</i> count	<200
<i>Staphylococcus aureus</i> count	<50

There is another group of bacteria called psychrotrophs. These are bacteria that grow well under cold conditions, as low as 2 to 9°C, and so these bacteria continue to multiply in the bulk tank. Some organisms are thermotrophic psychrotrophs; an example is *Bacillus cereus*, which is commonly found in the environment. If these enter the bulk supply, as may occur if teats are not properly prepared before milking, then they are not killed by pasteurization and continue to multiply in refrigerated pasteurized milk. *B. cereus* can cause food poisoning in man. Psychrotrophs are not routinely measured in bulk milk, as, provided milk meets the standards for the above tests, they should not cause any problem.

Bulk tank analysis is a useful way to identify herd and management factors associated with Bactoscan problems. It may be possible to eliminate a dirty milking plant or environmental contamination as the cause of the high Bactoscan. However, care is needed with interpretation. For example, if an organism has been isolated from a bulk sample, then we know that this organism is present in the herd. However, if a suspected organism has not been identified, it does not mean that it is absent from the herd, but rather that it just has not been identified from that particular sample. It may be the case that it may be identified, if present, in future samples.

It is essential that the sample milk for testing is transported from the farm to the laboratory within 24 hours, and at no more than 6°C to minimize any bacterial growth. Milk should be fresh and not frozen, although collection of daily samples over a week, freezing, and then processing the whole batch can help to eliminate laboratory variation associated with a series of samples. However, freezing will alter the number of organisms present; for example, there will be a reduction in the number of coliforms. If two bulk tanks are used, both should be sampled and tested individually, and any differences between milkers and wash-up routines should be recorded.

Bulk tank samples should be collected in the following way:

- Agitate the bulk tank for at least 2 minutes to ensure that the milk is well mixed, see Plate 10.5.
- Scoop at least 30 ml of milk into a sterile sample pot using a sterile scoop, or wearing a clean disposable glove and dipping the pot into the bulk tank. This ensures that the remainder of the milk does not become contaminated during sampling.
- Seal the container and label with the date and farm name, and tank identity (if samples are taken from more than one tank).
- Store at 4°C from collection until transporting to the lab; the sample can be kept in a fridge and then transported in an ice-box to the laboratory.



Plate 10.5. Milk samples must be collected from a well-agitated bulk tank and kept cold until they reach the lab.

Some milk quality labs that test the cell count and Bactoscan for dairy companies can also offer bulk tank analysis. The advantage is that they can test a sample with a known high Bactoscan reading, which will help pinpoint the origin of the contamination.

When to use bulk tank analysis

Raised Bactoscan (TBC) counts

It is essential to know the source of bacterial contamination in problem herds to be able to resolve the problem efficiently. Bulk testing can assist with this. In an ideal world, you would only test samples with known high counts, but this is not always practical. In herds with fluctuating Bactoscans, bulk samples can be frozen and then known high Bactoscan samples can be sent to the lab for testing. Freezing can affect the coliform count but, when the levels are high enough to influence the Bactoscan, they are well above the target levels and this has little effect on diagnosing the problem.

Raised somatic cell counts

Some herds do not have any individual cow cell counts or bacteriology results. The BTA can give a good indication of problem areas through the *Streptococcus uberis*,

Staphylococcus aureus and total staphylococci counts, along with the differential bacteriological screen. A BTA screen should always be carried out when investigating problem herds.

Problems with clinical mastitis

The majority of cases of clinical mastitis in low cell count herds will be due to environmental mastitis caused by *E. coli* and maybe some *Streptococcus uberis*. In some countries with high somatic cell count herds, there may also be problems from the contagious bacteria, such as *Staphylococcus aureus*, *Streptococcus agalactiae* and *S. uberis*. BTA provides helpful insight into hygiene from the coliform counts. A low count indicates excellent pre-milking teat preparation. But the presence of high coliform counts, along with the presence of faecal streptococci, yeasts and fungi, indicates gross contamination and will increase the risk of environmental mastitis. High levels of streptococci and staphylococci may also be contributing to the problems.

Screening tool

Many producers want to have a regular check on milk quality so that they can pass these results back to the milking team to provide motivation and encouragement. BTA can act as an early warning system for impending problems and allows early intervention.

Individual BTA tests

In some herds there may be a problem with teat preparation. Using the coliform count alone as a monitoring tool for individual milkings can have quite an impact on getting people to improve performance. This may be used as a separate test to monitor milkers whose hygiene may be marginal. Milkers can argue with your interpretation of their hygiene, but it becomes much more difficult to argue with lab tests. These results support the decision-making process.

Milk quality enhancement programmes

When farmers have Bactoscan results below the target levels set by their milk buyer, there may still be further room for improvement. A Bactoscan enhancement programme was set up for a dairy company where BTA was carried out for every milk producer over a 2-year period. The milk buyer wanted to have much lower Bactoscan results for commercial advantage and this was a novel way of persuading farmers to improve. Results and interpretation were returned to the farm (see Fig. 10.5).

While many of the farmers may have considered they had no areas for improvement, it was surprising to find that only 6% had levels below all the target figures for LPC, coliform and *Staphylococcus aureus* counts; 78% had wash-up problems; 35% had high coliform counts, indicating poor premilking teat preparation; 53% had above target levels of *S. aureus*; and 8% of herds were found to have *Streptococcus agalactiae* infections. Over the 2-year period, the Bactoscan averages for all purchased milk fell from 38,000 to 20,000/ml. There was also a reduction in cell counts by identifying and addressing contagious mastitis problems that were previously not identified.

Interpretation of bulk samples

The interpretation of bulk samples requires knowledge of the various tests and how they relate to mastitis management. Frequently, the problems identified may involve more than one area of mastitis. For the four problem study herds discussed below, the results are given of bulk tank sample analyses, together with their interpretations, in conjunction with other findings on the farm.

When investigating problems, remember that analysis of one bulk tank sample is a snapshot of the milk on that day only. A variety of factors need to be considered, including: who was milking, were the normal milking and wash-up routines followed, how good was mastitis detection on that day compared with others, and was the sample correctly taken, stored and dispatched to the laboratory?

Some advisers may read too much from one sample. A lab result should never be considered in isolation from the rest of the herd history. It must be remembered, however, that bulk tank analysis is only an aid to identifying the possible causes of high Bactoscans.

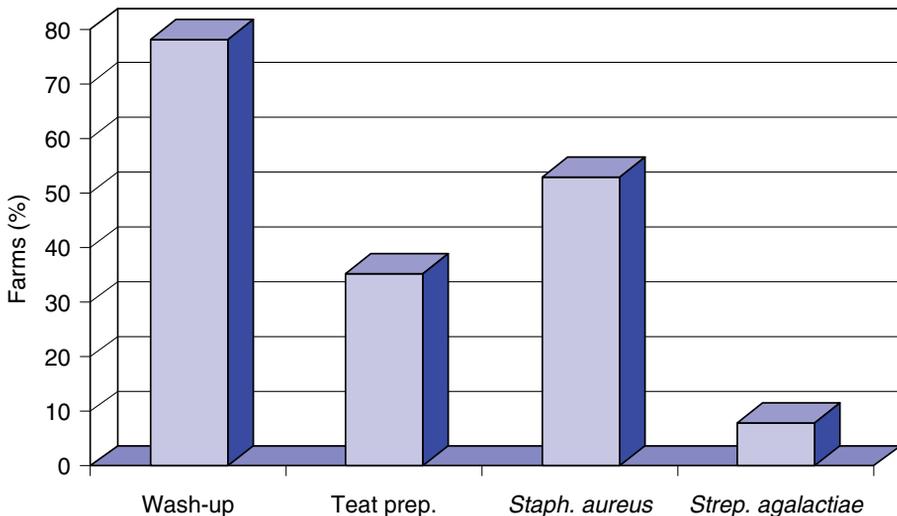


Fig. 10.5. Percentage of farms with problems with wash-up, teat preparation, presence of *Streptococcus agalactiae* or above target levels of *Staphylococcus aureus*.

Examples of the use of BTA in problem herds

The following examples are actual results taken from real-life herd problems investigated by the authors.

Herd A: somatic cell and clinical mastitis problems, and the occasional high Bactoscan result

The presenting problem in herd A is a rising cell count (see Table 10.4) and a high level of clinical mastitis. Milking cows are housed both in cubicles and on straw yards. The owner has a herdsman who is convinced that he is doing an excellent job. The herd has expanded by 50% over the past 3 years. Various recommendations have been made but were rejected due to shortage of time.

The TBC is above target and the high coliform count result shows major problems with teat preparation. The presence of yeasts and *Streptococcus faecalis* further confirm poor hygiene. There are also high *S. uberis*, staphylococci and *Staphylococcus aureus* counts and high cell count. The presence of *Corynebacterium bovis* suggests problems with postmilking teat disinfection. The wash-up routine was adequate, as shown from the low thermoduric count, or LPC, of 50, well below the target of 175.

The following were found during the farm visit. The coliform count of 312 shows that teat preparation was poor, as the milker was finding it difficult to milk on his own; he had too big a parlour for one milker and was trying to cut out tasks to ensure he had a fast throughput in the parlour.

Units were applied to dirty teats. There was no foremilk and mastitic milk frequently entered the bulk tank. As many of the cows were bedded on straw yards, a common cause of clinical mastitis will have been *Streptococcus uberis* which is shed at very high levels and can increase bulk tank Bactoscan results. Straw yards were cleaned out every 6 to 8 weeks instead of every 3 to 4 weeks, due to shortage of labour.

There were no separate clusters for milking cows with mastitis and, when a regular cluster was used to milk a cow with clinical mastitis, it was not disinfected before milking the next cow. There were a lot of old cows with high cell counts and these cultured positive to *Staphylococcus aureus*. The farmer was teat dipping but, due to reduced profitability, diluted the teat dip 1:5 rather than 1:4 to try and save money. This will have contributed to the high levels of *C. bovis*.

The bacteriology results helped to persuade the owner and his milker to make major changes to mastitis management and the problems were slowly resolved.

Herd B: somatic cell and Bactoscan problems

The request for herd B was to investigate a high somatic cell count of over 400,000/ml (Table 10.5). The owner had another business, which has been far more profitable than the dairy, and he had left control to the dairy staff for many years. Four months earlier, the old staff left and two replacements with little experience of milking were recruited. They were given minimal training.

Table 10.4. Bulk tank analysis for Herd A.

Test	TBC	LPC	Coliform count	<i>Pseudomonads</i> count	<i>Streptococcus uberis</i> count	Total staphylococci count	<i>Staphylococcus aureus</i> count	SCC × 1000/ml
Target	<5000	<175	<20	<500	<200	<200	<50	<150
Result	9000	50	312	250	6500	650	216	338
Differential bacterial screen				Yeasts ++ <i>Streptococcus faecalis</i> +++ <i>Corynebacterium bovis</i> ++				

Table 10.5. Bulk tank analysis for Herd B.

Test	TBC	LPC	Coliform count	<i>Pseudomonads</i> count	<i>Streptococcus uberis</i> count	Total staphylococci count	<i>Staphylococcus aureus</i> count × 1000/ml	SCC
Target	<5,000	<175	<20	<500	<200	<200	<50	<150
Result	22,000	950	87	590	150	1,600	330	421
Differential bacterial screen				<i>Streptococcus faecalis</i> +++ <i>Streptococcus dysgalactiae</i> + <i>Corynebacterium bovis</i> ++ Yeasts ++, Fungi +				

The herd had a new parlour fitted 2 years ago and since then had never had a low Bactoscan count, with levels always above 60,000/ml.

The TBC is well above target and the high LPC (950) result shows that there is a plant wash-up problem. The high coliform count (87) and the presence of *Streptococcus faecalis*, yeasts and fungi suggest poor environmental conditions and/or premilking teat preparation. The total staphylococci and *Staphylococcus aureus* counts are very high, indicating that these are likely to be contributing to the high cell count of 421,000.

The presence of *C. bovis* suggests that there are problems with postmilking teat disinfection. That of *Streptococcus dysgalactiae* suggests teat damage or poor teat skin condition.

From further investigation, the following were identified. Postmilking teat disinfection had stopped completely a few months previously. Teat skin condition was very poor, with quite a few teat lesions. Teats were dirty on entering the parlour and were washed but not dried before units were attached. This effectively suspends environmental organisms into a 'soup' on the teat, which subsequently passes into the bulk tank.

A hot wash after milking was carried out once daily with inadequate amounts of hot water, insufficient chemicals and blocked air injectors, and this resulted in deposits in the milk transfer line, as shown in Plate 10.6. The milker also failed to turn off the plate cooler, which cooled the circu-

lating wash solutions. This accounted for the high LPC.

Once the wash cycle was modified, the Bactoscan results fell to under 30,000/ml immediately. The predominant cause of the high cell count was *Staphylococcus aureus*. This was identified from bacteriology of the high cell count cows and the fact that it was the older cows that had the high cell counts. Over a period of a year the cell count was reduced to below 150,000 and many chronic high cell count *S. aureus* cows were culled.

Herd C: high Bactoscan counts

The presenting problem in herd C was consistently high Bactoscan results of over 70,000 in a herd that normally had excellent milk quality and low levels of clinical mastitis. Prior to being asked to investigate, the



Plate 10.6. Soil can be seen half way up the milk transfer line, caused by lack of turbulence during the wash cycle.

owner had stripped the parlour and plate cooler and cleaned all areas thoroughly as he thought it might have been due to a fault with the wash-up routine after milking. He had also replaced all the rubberware in the plant.

The initial results failed to identify any specific problem that could be responsible for the high Bactoscan result, apart from a slightly raised LPC (190) and raised *Pseudomonads*, total staphylococci and *S. aureus* counts (Table 10.6). Also, the fact that the LPC was slightly raised did not seem to support a failure of washing sufficient to cause such a high Bactoscan. This sample did not seem to support such high readings as were found on the Bactoscan results.

A farm visit was arranged to check the acid boiling wash procedure; this showed that the plant temperature was only 71°C, 6°C lower than required for effective washing. The boiler was serviced and wash temperatures adjusted, but the high Bactoscan counts persisted. Another bulk sample was collected and showed low thermoduric counts but a *Pseudomonads* count of over 15,000/ml.

Water samples were collected from all the water sources in the parlour and dairy but all tested negative. The only possible source of high levels of *Pseudomonads* was from the chilled water from the bulk tank that circulated around the plate cooler to cool milk rapidly. The plate cooler was bypassed and the Bactoscan results immediately fell back to their normal level of under 10,000/ml.

It transpired that there was a pinpoint leak in the plate cooler that allowed small

amounts of the chilled water contaminated with *Pseudomonads* to enter the milk as it passed through the plate cooler. *Pseudomonads* are psychrotrophs and continued to multiply in the bulk tank, and were responsible for the high Bactoscan. This highlights the advantages of the differential tests in diagnosing causes of the problem, but also demonstrates the need to visit the farm to see what's happening.

While the herd cell count is 98,000/ml and indicates good control of contagious mastitis, the slightly raised counts for *S. aureus* and other staphylococci need to be carefully monitored.

Herd D: problems with teat preparation due to large numbers of milkers

The 600 cows of herd D were milked three times daily through two separate parlours. Both groups had a very high mastitis rate and used up to seven different milkers. Poor teat preparation was one of the main problems contributing to the high levels of clinical mastitis. Bacteriology results showed that most clinical cases were environmental in origin. Milkers blamed each other for failure to prepare teats properly.

Milker schools were carried out during week 2 to provide a uniform agreed milking routine and to explain why changes were necessary. Coliform counts were carried out in week 1 without the milkers' knowledge. This showed that all the milkers, with the exception of Sean, had poor teat preparation. A league table was organized to rank milkers according to coliform counts (Table 10.7). This had an impact on most milkers, and

Table 10.6. Bulk tank analysis for Herd C.

Test	TBC	LPC	Coliform count	<i>Pseudomonas</i> count	<i>Streptococcus uberis</i> count	Total staphylococci count	<i>Streptococcus aureus</i> count × 1000/ml	SCC
Target	<5,000	<175	<20	<500	<200	<200	<50	<150
Result	11,000	190	3	995	70	325	65	98
Differential bacterial screen				<i>Streptococcus faecalis</i> +				

Table 10.7. Coliform counts for Herd D.

	John	Sean	Martin	Mike	James
Week 1	35	8	944	28	142
Week 2	24	20	254	22	95
Week 3	15	12	165	25	18
Week 4	18	16		18	14
Week 5	12	9		12	17
Comment	Improved	Always a clean milker	Stopped milking Week 3	Improved	Significant improvement

any who remained consistently high were assigned other duties outside the parlour.

This approach stopped milkers blaming each other, highlighted Sean as a consistently good milker and removed Martin from

the milking team. The other three milkers have been shown that their improvements following the milking school have paid dividends in their performance, with a concurrent reduction in clinical mastitis cases.

11 Targets and Monitoring

Record Keeping	185
Mastitis Targets	185
Mastitis rate	185
Percentage of herd affected	185
Recurrence rate	187
Milking cow tube usage	188
Seasonal variation	188
Stage of lactation	188
What Does Mastitis Cost in My Herd?	189
Herd Examples	190
Herd A	190
Herd B	191

This chapter examines the different ways in which mastitis can be recorded and how records can be used to help identify the cause of mastitis. Target figures and the economics of the disease are discussed and some examples are given of how herd records can be used.

Records are an important part in monitoring the incidence of any disease. Mastitis is no exception. In fact, mastitis is one of the few diseases where a detailed analysis of the data can be used to help in the control of infection.

Many farmers rely on their cell count results to give an indication of their mastitis situation, as their milk buyer provides this information monthly. Many farmers keep mastitis records, but these are not analysed and so the incidence of clinical mastitis is often underestimated. Cell counts do give useful information but have limitations. High counts indicate the pres-

ence of subclinical mastitis, especially that due to *Staphylococcus aureus*, *Streptococcus dysgalactiae* and *Streptococcus agalactiae*. Unfortunately, cell counts do not necessarily bear any relation to the clinical incidence of mastitis. Therefore, it is important to have and make use of accurate clinical records; otherwise there is little benefit to be gained from keeping the data.

Mastitis records will enable the farmer to do the following:

- Identify cows whose milk needs to be withheld from the bulk supply.
- Identify problem cows that should be considered for culling.
- Allow detailed monitoring of the herd mastitis performance to check that it is within acceptable limits and to see how the herd compares with others being monitored.

- Gain valuable information that can point towards the possible cause of mastitis outbreaks and other problems.

Record Keeping

The following should be recorded for each case of mastitis:

- Cow number.
- Date.
- Quarter/s infected.
- Treatments given and number of tubes of antibiotic used.
- Bacteriology results (if available).

One case of mastitis is defined as one quarter infected once. A cow that calves down with mastitis in all four quarters therefore counts as four cases of mastitis. If a treated quarter clears up but mastitis recurs 7 or more days after the remission of clinical signs, then this is defined as a new case of mastitis. If mastitis recurs in the same quarter less than 7 days after the remission of clinical signs, then this is defined as a continuing case of mastitis.

Mastitis cases can be recorded in a variety of ways (see Fig. 11.1). Ideally, subsequent cases of mastitis should be recorded adjacent to the first case so that problem cows are readily identified. In the first method of data layout, shown in Fig. 11.1(a), it can easily be seen that cow 32 has had repeat cases of mastitis. Although exactly the same information is given using the second method, shown in Fig. 11.1(b) it is not immediately apparent that cow 32 is such a problem.

Mastitis records should be checked regularly and cows with four or more cases should be considered either for culling from the herd or for having the offending quarter dried off.

An alternative, more visual system, can be used, as shown in Fig. 11.2. Here the cows are recorded on a bar chart on a monthly basis. The cow number and quarter are entered on the chart and a monthly target can be added. In this example, it can be seen that the incidence is above target from

November to April, which coincides with housing.

Record analysis will allow the most appropriate control measures to be put into place. It is important that these data are analysed regularly. Every 6 months is ideal, as it will help to identify possible problems and trends. Economic data can also be included to cost the benefits or losses from mastitis, together with Bactoscan and cell count penalties.

Mastitis Targets

Table 11.1 gives a range of figures that should be achievable within a herd, that is the targets to be aimed for and the level at which some action or interference should be taken.

Mastitis rate

The mastitis rate is the number of cases of mastitis per 100 cows per annum. It is an invaluable measure of the mastitis incidence as it allows comparison between herds irrespective of size. A mastitis rate below target means that the herd has good control of clinical mastitis. Of course, this is assuming that all cases of mastitis have been accurately recorded.

The mastitis rate can be worked out using the formula below:

$$\text{Mastitis rate} = \frac{\text{No. of cases of mastitis per year} \times 100}{\text{Total no. of cows in herd (milking and dry)}}$$

A high mastitis rate indicates a high number of mastitis cases in the herd but does not identify what type of infection is present, i.e. contagious or environmental.

Percentage of herd affected

The percentage of cows affected per year represents the proportion of the herd that have had one or more cases of mastitis over a 12-month period. This helps to give some

PERIOD	TO	FARM PRACTICE							
		KEY LEFT FORE LEFT HIND	<input type="checkbox"/> <input type="checkbox"/>	RIGHT FORE RIGHT HIND	<input type="checkbox"/> <input type="checkbox"/>	10/9 *	<input type="checkbox"/> <input type="checkbox"/>	DIED OFF	<input type="checkbox"/> <input type="checkbox"/>
COW NUMBER	TOTAL THIS LACTATION	NOTE: FIRST TREATMENT ONLY							
69	<input type="checkbox"/>	22.5	<input checked="" type="checkbox"/>						
32	<input type="checkbox"/>	25.5	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	3.7	<input checked="" type="checkbox"/>	15.8	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>
73	<input type="checkbox"/>	2.6	<input checked="" type="checkbox"/>						
4	<input type="checkbox"/>	15.6	<input checked="" type="checkbox"/>		17.9	<input checked="" type="checkbox"/>			
17	<input type="checkbox"/>	12.8	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	21.9	<input checked="" type="checkbox"/>			
166	<input type="checkbox"/>	25.9	<input checked="" type="checkbox"/>						
99	<input type="checkbox"/>	3.10	<input checked="" type="checkbox"/>						
37	<input type="checkbox"/>	14.11	<input checked="" type="checkbox"/>						
91	<input type="checkbox"/>	21.11	<input checked="" type="checkbox"/>						

(a)

Cow	Date	Quarter
69	22.5	LH
32	25.5	LH + RH
73	2.6	LF
32	3.7	LH
4	15.6	LF
17	12.8	LH + RH
32	15.8	LH + RH
4	17.9	RF
17	21.9	RH + RF
166	25.9	RH
99	3.10	LF
32	23.10	LH
37	14.11	LH
91	21.11	RH

(b)

indication of the type of mastitis present. On the one hand, chronic recurring mastitis, caused by *Staphylococcus aureus*, could affect a small percentage of the herd, but there may still be a high mastitis rate. This may occur because the same cows keep getting repeat cases of mastitis in the same quarter. On the other hand, an outbreak of coliform environmental mastitis is likely to

Table 11.1. Target and interference levels for different mastitis and milk quality parameters.

Parameter	Targets	Interference
SCC	150,000	200,000
Bactoscan	20,000	30,000
Mastitis rate (cases per 100 cows per year)	30	40
Percentage herd affected	20	25
Recurrence rate	10	20
Milking cow tubes per cow per year	1.4	2.5
Milking cow tubes per case	4.5	6.0
Percentage dry cow mastitis	1.0	2.5

Fig. 11.1. Two forms of mastitis recording. (a) The top system more readily identifies the problem cows as all information relating to the same cow is recorded on one line. (b) In the chart below separate cases of mastitis in the same cow are not related back to each other as they are in the top chart

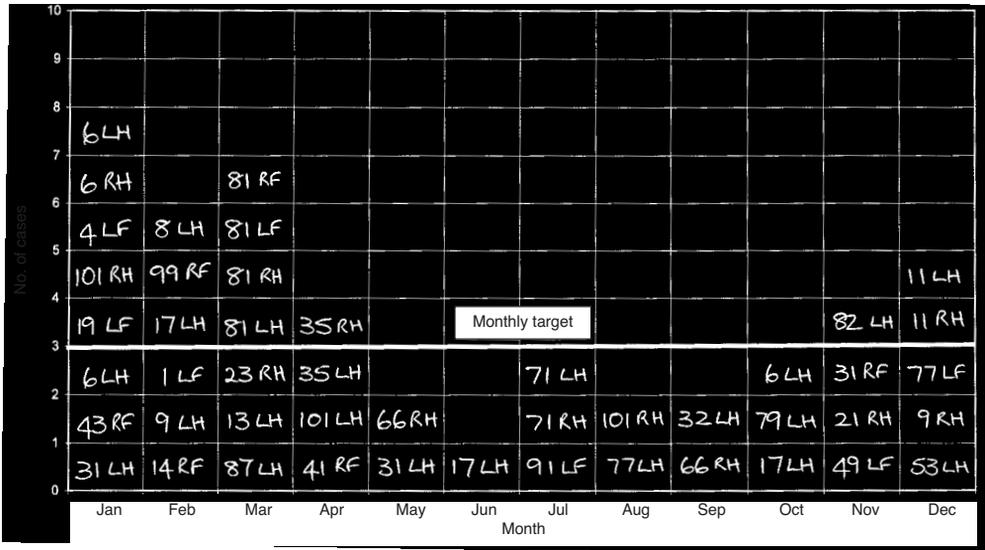


Fig.11.2. Monthly mastitis records.

result in a larger percentage of the herd affected but relatively few repeat treatments in the same quarter. Herds with severe problems with environmental mastitis can have 35% or more of the herd affected.

The percentage of cows affected per year can be worked out using the following formula:

$$\text{Percentage cows affected} = \frac{\text{No. of cows that have had mastitis over a 12-month period} \times 100}{\text{Total no. of cows in the herd (milking and dry)}}$$

Recurrence rate

The recurrence rate is the percentage of quarters requiring one or more repeat treatments over a 12-month period. A repeat treatment refers to one or more cases of mastitis recurring in the same quarter. The recurrence rate can be worked out using the following formula:

$$\text{Recurrence rate} = \frac{\text{No. of mastitis quarters requiring repeat treatment} \times 100}{\text{Total no. of quarters affected}}$$

For example, in Fig 11.1 cows 32 and 17 have both had quarters requiring one or more repeat treatments. Cow 32 had two recurring quarters (the left and right hind (LH and RH)) and cow 17 had one (the right hind (RH)). The total number of quarters needing one or more repeat treatments is therefore three – two (LH and RH) for cow 32 and one for cow 17 (RH).

So, looking at the cows in Fig. 11.1, there have been 18 cases of mastitis in a total of 13 quarters. Three of these quarters have had one or more repeat treatments and so the recurrence rate is $3/13 \times 100 = 23\%$.

A high recurrence rate may be due to problems with *Staphylococcus aureus* or *Streptococcus uberis* infections, which are often difficult to treat. High rates can also be due to poor mastitis detection, where infections are not picked up early and so the response to treatment is poor. Likewise, if the treatment regime is ineffective, such as too short a duration of treatment or the incorrect selection of antibiotic, then cases may also recur.

Milking cow tube usage

The number of milking cow tubes used in the herd will depend on the number of cases of mastitis, the number of tubes used to treat each case and whether any high cell count cows were treated with intramammary tubes during lactation. Although most manufacturers recommend three tubes per case, the average usage is closer to five to six tubes per case. A high number of tubes used per cow per year, e.g. over 6, could indicate one or more of the following:

- A high incidence of clinical mastitis.
- Infection that responds poorly to treatment.
- Not all cases of mastitis have been recorded.
- A high number of high cell count cows treated in lactation.
- Mastitis tubes used for reasons other than for treating mastitis.

As the total tube usage is not collected from farm data (it comes from your vet and/or pharmacist), the tube usage figure gives a useful indication of the accuracy of farm data.

Many veterinary practices now have computerized accounting systems that can give the number and type of intramammary tubes supplied over a given period of time. The number of milking cow tubes used per case of mastitis and per cow is worked out using the following formulae:

$$\text{No. milking cow tubes per case} = \frac{\text{No. tubes used}}{\text{No. cases mastitis}}$$

$$\text{No. tubes per cow} = \frac{\text{No. tubes used per year}}{\text{Total no. of cows in herd}}$$

Seasonal variation

It is useful to examine mastitis incidence by month of the year. A high number of cases in the housed period suggests a problem due to environmental mastitis, while year-round incidence may suggest a problem with contagious mastitis. It is helpful to work out the percentage of cases that occur during the housed period, as this is the period of greatest risk of environmental mastitis. Figure 11.2 shows the seasonal trends of clinical mastitis in one herd, clearly with the majority of mastitis occurring during the housed period.

Stage of lactation

Analysis of mastitis according to stage of lactation can be very helpful. This information is only likely to be able to be extracted from herds that record on a computer system which can analyse mastitis data.

If there is a peak of mastitis around the time of calving, this suggests problems with organisms such as *Streptococcus uberis* and *E. coli*, as shown in Fig. 11.3. In this herd, it can be seen that 15% of all cases occur within a week of calving. These relate to dry period infections and the conditions in which the cows are kept during the dry period and around calving. Also, 25% occur between 61 and 100 days, which coincides with peak lactation. These figures suggest that there are many issues with cows in early lactation and further investigation is required.

More than half of all cases of mastitis are expected to occur within the first 100 days of calving as this relates to the effect of dry period infections, calving, peak yield and the highest production stress on the cow. If mastitis occurs all the way through lactation, then this suggests that there may be other factors that are influencing mastitis, such as defective milking machine function or poor hygiene.

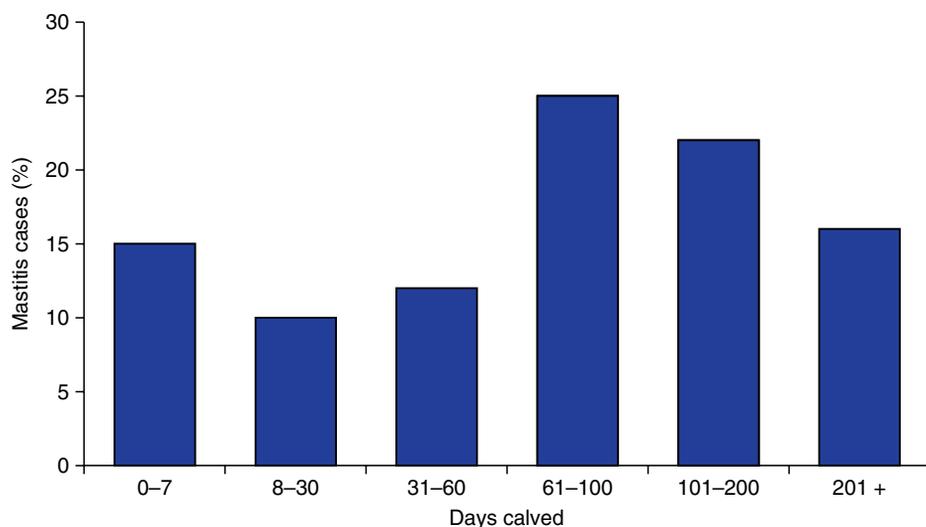


Fig.11.3. Incidence of mastitis by days calved.

What Does Mastitis Cost in my Herd?

Mastitis and milk quality costs are easily quantified. They include:

- Penalties from cell count.
- Penalties from Bactoscan.
- Increased clinical mastitis.
- Reduced yields.
- Any costs relating to a bulk tank antibiotic failure.

Mastitis is one of the few diseases where these losses can easily be calculated, and two herd examples are discussed at the end of this chapter. Penalties from the cell count and Bactoscan can easily be added up from milk statements. Alternatively, you multiply the penalty by the average yield per cow and the number of cows in the herd. So a herd of 150 cows with an average yield of 8000 litres losing 0.3 p.p.l. (pence per litre) from the cell count and 0.2 p.p.l. from the Bactoscan is losing a total of 0.5 ppl of milk. If you then multiply this by 8000 litres it comes to a loss of £40 per cow per year. The herd cost is £40 times 150 cows, which is £6000 per year.

Of course, this does not take into account any effect of reduction of milk yield from the high cell count herds, or the

culling, the treatment of problem cows or the time spent trying to manage the problem.

For clinical mastitis, the costs are more difficult to work out. They include:

- Discarded milk.
- Medicine costs.
- Labour.
- Veterinary fees.
- Any deaths.
- Reduction in yield for the rest of lactation.
- Risk of spread to other cows.
- Culling and loss of genetic potential.

The milk discarded is easy to work out by adding the duration of treatment to the withdrawal period and multiplying by the average yield of the cow. For example, if a cow with clinical mastitis giving 40 litres per day is treated for 5 days, and then has a milk withdrawal period of 4 days, then her milk will be out of the tank for 9 days and the total milk loss will be 360 litres. This can then be multiplied by the milk price. Some farmers discount these costs, as this milk may be fed to calves. However, many regard it as inadvisable to feed mastitic milk to replacement heifer calves, as this might contribute to problems with antibiotic

resistance, although there are no firm data to support the existence of this risk.

Medicine costs and labour should be easy to quantify. Many farmers estimate that a simple case of mastitis slows down milking. Time is spent milking the cow separately, administering any medication, completing the farm records and ensuring that the milk does not enter the bulk supply.

There is a reduction in milk yield following mastitis. Mild cases can result in a 10% reduction in yield and more severe cases 25%, and toxic cases may result in cows not producing any milk whatsoever. The cost of this reduction in yield is always underestimated. If you take a cow with a mild case of mastitis in one quarter, in a cow yielding 8000 litres, that quarter will have a reduction in yield of 200 litres.

Often average figures are quoted for clinical mastitis, which may be too high or too low for some herds. It is always worth working out the costs of discarded milk, treatment and labour for an average case as a benchmark figure. An allowance then needs to be added to cover the costs of reduced yield for the remainder of lactation, and also the culling, death and veterinary fees.

Herd Examples

Herd A

The mastitis data for herd A are shown below and relate to a 12-month period. The farmer requested help in dealing with his cell count problem, from which he was losing 1 p.p.l. He has kept accurate records and has used his milking cow tubes only for treating clinical mastitis.

Herd size: 150 cows
 Number of cases of mastitis: 188
 Number of cows affected with mastitis: 68
 Total number of quarters with mastitis: 125
 Number of quarters with one or more repeat treatments: 42
 Number of intramammary tubes purchased: 750
 Average yield per cow: 8000

$$\begin{aligned} \text{Mastitis rate} &= \frac{188 \text{ (cases of mastitis)} \times 100}{(150 \text{ cows in herd})} \\ &= 125 \text{ cases}/100 \text{ cows/year} \end{aligned}$$

$$\begin{aligned} \text{Percentage of herd affected} &= \frac{68 \text{ (no of cows affected)} \times 100}{150 \text{ (cows in herd)}} = 45\% \end{aligned}$$

$$\begin{aligned} \text{Recurrence rate} &= \frac{42 \text{ (quarters repeating)} \times 100}{125 \text{ (quarters with mastitis)}} = 34\% \end{aligned}$$

$$\begin{aligned} \text{No. of milking cow tubes per cow per year} &= \frac{750 \text{ (tubes)}}{150 \text{ (cows)}} = 5.0 \end{aligned}$$

$$\begin{aligned} \text{No. of tubes per case per year} &= \frac{750 \text{ (tubes)}}{188 \text{ (cases)}} = 4.0 \end{aligned}$$

Mastitis rate	125
Percentage of herd affected	45%
Recurrence rate	34%
No. of tubes/cow/year	5.0
No. of tubes/clinical case	4.0
Herd cell count	280,000

The mastitis rate of 125 shown above indicates major problems with clinical mastitis and is more than four times the target level of 30. The farmer had no idea of the extent of his clinical mastitis problem. He did keep records, but these were never analysed. He was very surprised at the level of infection. This is a common finding in herds in which there is no regular analysis of their mastitis records.

As 45% of the herd has had one or more cases, this suggests that there are issues with environmental mastitis. Further record analysis shows that 60% of all cases occurred during the housed period – 5 months of the year – again suggesting that clinical mastitis is due more to environmental bacteria.

The recurrence rate of 34% is very high and suggests that there is a problem either with *Staphylococcus aureus* or *Streptococcus uberis* infections, poor mastitis detection and/or a poor treatment regime. The herd has a high cell count and so we

know that there are problems with subclinical mastitis, suggesting that *Staphylococcus aureus* or *Streptococcus uberis* would be prevalent in this herd. The cause of clinical cases (and, of course, of high cell count) will need to be confirmed by bacteriology.

Economics of mastitis for herd A

The milk buyer is deducting 1 p.p.l. or 4% of the milk price due to the high herd cell count. If you multiply milk yield (8000 litres) times financial penalty (1 p.p.l.) this works out at £80 per cow per year. Multiply this by the number of cows in the herd (150) you get a total loss of £12,000. This is more than the cost of the farmer's veterinary bills and medicine costs.

The farmer estimates that each case of mastitis costs £125. He treats cows for an average of 3 days, with a 4-day milk withdrawal period, and so milk is discarded for a total of 7 days. The average yield of cows with clinical mastitis is 40 litres and so for each clinical case he is discarding 280 litres at an average cost of 25 p.p.l., which is £70.

Medicine costs for a case average £25 (as he injects his cows as well as using intramammary tubes) and he has allowed £10 for labour. These three costs alone come to £105 and he has not made any allowance for any reduction in yield for the remainder of lactation, or for toxic cases or any culling that may result from persistent cases. It is likely that he has underestimated the cost of clinical mastitis.

Clinical mastitis cannot be eradicated but, over a period of time, we may be able to attain the target mastitis rate of 30 cases per 100 cows per year, or 45 cases in the herd per year (there are 150 cows, so you multiply the target figure by 1.5). This means that we would save a total of 143 cases at a saving of £17,875 (143 cases × £125/case). While it may take some time to achieve this, as there may be issues with accommodation or the milking parlour, the savings do help the farmer to focus on the true losses and also help him make any decisions on capital expenditure.

So, for this herd, if the cell count and clinical mastitis issues are resolved, there is

a potential to increase profit by £29,875 per year (£12,000 plus £17,875), which is equivalent to almost £200 per cow, or to a milk price rise of 2.5 p.p.l. (10% of the current price). So, while the farmer initially called for help for his cell count problem, as this was easily identified from his milk statement, he was totally unaware that the greatest economic loss came from the high levels of clinical mastitis in his herd.

Herd B

This is a 200-cow herd yielding close to 9000 litres which is housed from November through to the end of April. The mastitis data have been analysed and the results are as follows:

Mastitis rate	73
Percentage of herd affected	54%
Recurrence rate	12%
No. of tubes/cow/year	2.6
No. tubes/clinical case	3.5
Herd cell count	120,000

Figure 11.4 shows the seasonality of clinical mastitis, along with a baseline showing the average monthly target of five clinical cases: the target is for a mastitis rate of 30 cases per 100 cows per year, and so, as this herd has 200 cows, this would be 60 cases per year or five per month. Figure 11.5 shows the distribution of clinical cases according to days calved.

The mastitis rate of 73 shows significant problems with clinical mastitis. Over 50% of the herd has had clinical mastitis, which suggests problems with environmental mastitis. The herd cell count is 120,000, which suggests little problem with contagious mastitis.

Figure 11.4 confirms environmental infections, with 69% of all cases occurring during the 6-month housed period. Figure 11.5 shows that 27% of all clinical cases occur within the first week of calving, suggesting problems with dry period infections, such as *Streptococcus uberis* and *E. coli*, and possibly poor management around calving. However, if the herd

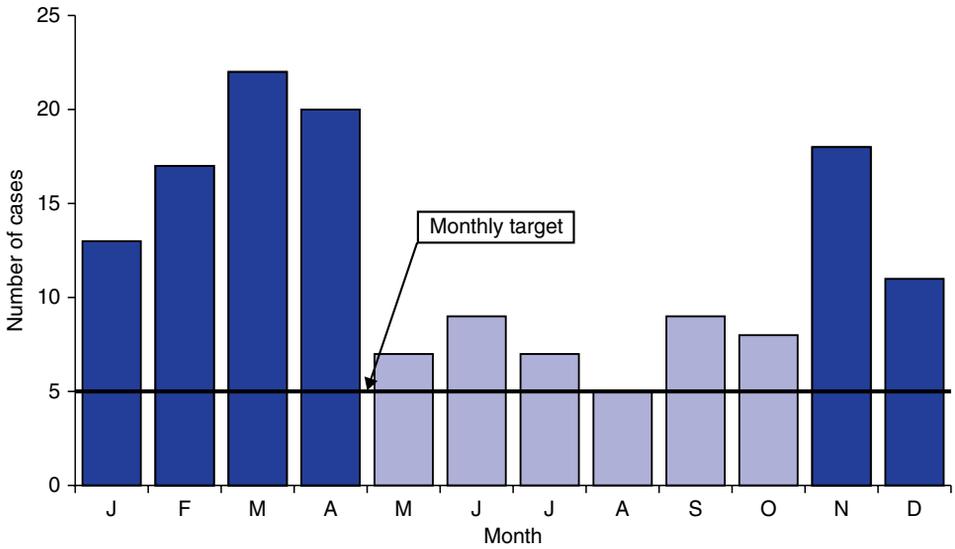


Fig.11.4. Monthly mastitis cases in herd B. Blue bars indicate housed period, light blue bars non-housed period.

had problems with *S. uberis*, the herd cell count would be higher and so it is most likely that the problem is due to *E. coli* and other coliform infections. The farmer had two cows die with postcalving *E. coli* mastitis, which is the most severe form of mastitis.

The recurrence rate of 12% is slightly above target; however, the herd cell count is low, at 120,000, indicating very good control

of subclinical mastitis. There are likely to be few problems with *Staphylococcus aureus* or *Streptococcus uberis*. But there could be some cows in the herd that are infected with either of these bacteria. An average of 3.5 tubes per case are used and this suggests that the response to treatment is adequate. Bacteriology is required to confirm that *E. coli* is the predominant cause of clinical mastitis.

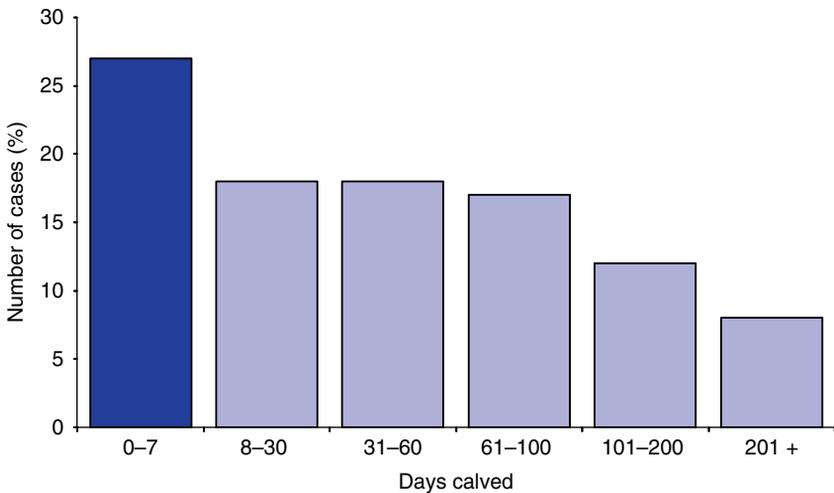


Fig.11.5. Clinical cases by stage of lactation in herd B.

Economics of mastitis for herd B

There are no cell count or Bactoscan penalties for this herd. The herd owner estimates that each case of mastitis is costing him £200 due to the fact that two cows have died with clinical mastitis in the past year, and four others have either dried up or lost quarters. He also realizes that there is a significant pro-

duction loss for the remainder of lactation.

Mastitis cannot be eradicated but, in time, there is no reason why the mastitis rate cannot drop from 73 down to 30. This is a saving of 43 cases per 100 cows. The owner has 200 cows and so will save 86 cases of mastitis at £200 each, which is £17,200 each year, or £86 per cow per year, equivalent to a milk price increase of almost a penny per litre.

12 Treatment and Dry Cow Therapy

Treatment Overview	195
Reasons for Treatment	195
Treatment During Lactation	195
Separation of the mastitic cow	195
Technique for the administration of intramammary antibiotics	196
Antibiotic Therapy	197
Is antibiotic treatment worthwhile?	197
Choice of Antibiotic	199
Antibiotic sensitivity and udder penetration	199
Response of coliforms to antibiotics	201
Effectiveness in milk	201
Bactericidal and bacteriostatic antibiotics	201
Acidity and lipid solubility	202
Intracellular effects	202
Withdrawal period	202
Benefits of Early Treatment	202
Combination Antibiotic Therapy (Concurrent Injection and Tubing, Aggressive Therapy)	203
Drying Off Quarters	204
Resistance of <i>Staphylococcus aureus</i> to Treatment	205
Blitz Therapy Against <i>Streptococcus agalactiae</i>	206
Supportive Therapy	206
Fluid therapy	207
Anti-inflammatory drugs	208
Administration of calcium	208
Administration of glucose	208
Continual stripping and oxytocin	208
Non-antibiotic intramammary infusions	209
Topical preparations	209
Homoeopathy	209
Dry Cow Therapy	209
Long-acting antibiotics	210
Treat all quarters	211
Teat sealants	212
Administration of dry cow tubes	212
Infusion technique	213

While much of this book focuses on the prevention and control of mastitis, the text would not be complete without some reference to treatment. The main objective of treatment is to reduce or eliminate infection from the udder.

Treatment Overview

Mastitis treatments can be administered at two different stages in the cow's lactation cycle:

- **Lactating cow therapy** is administered to cows while they are in milk.
- **Dry cow therapy**, administered the day the cow is dried off, attempts to: (i) remove those infections accumulated during the lactation, i.e. to prevent carry-over to the next lactation; and (ii) to reduce the number of new infections contracted during the dry period.

Mastitis treatment can be administered by different routes:

- **Intramammary treatment** is infused into the udder through the teat canal.
- **Parenteral treatment** is given by injection.

Reasons for Treatment

Irrespective of the cause of mastitis, there are several reasons why some form of treatment (not necessarily antibiotic) should be instigated as soon as a cow is clinically affected. These are:

- To prevent the spread of infection to other cows.
- To restore the productivity of the cow, thereby allowing her milk to be sold.
- To prevent the mastitis from getting any worse.
- To reduce the probability of recurrent cases.
- To avoid long-term and possibly irreversible udder damage, which would have a deleterious effect on yield and milk quality (i.e. cell count and TBC/Bactoscan).

- To improve overall cow health and welfare.

Treatment During Lactation

It is the milker who will first recognize a case of mastitis and it is usually the milker who will make many of the decisions relating to treatment. This chapter is therefore written with this person very much in mind. Foremilking and other procedures to assist in the prompt recognition of clinical mastitis are described in Chapter 6, which should be read in conjunction with this section.

Separation of the mastitic cow

Ideally, as soon as a clinical case has been identified, the mastitic cow should be separated from others to prevent the spread of infection. In large herds, this may consist of physically removing the cow to a mastitic or 'hospital' group, which is then milked last, and where treatment is administered. In smaller herds, the affected cow should be carefully identified, e.g. by a leg or tail band or udder spray, and then milked through a separate cluster and into a dump bucket (Plate 12.1) or dump line.

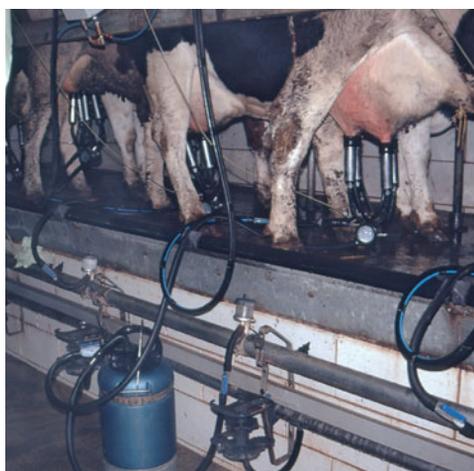


Plate 12.1. A dump bucket with separate claw.

The advantages of separating the mastitic cow into a separate group are:

- Treatment can be administered more carefully and recorded.
- It reduces the risk of transfer of infection to other cows.
- It reduces the risk of antibiotic contamination of the bulk tank.

If the cow is in a separate group, then the milker has sufficient time to administer intramammary antibiotics carefully, record which cow has been treated and possibly take her temperature to see if additional parenteral therapy (by injection) is needed. During milking, there is much more pressure on the milker and a greater risk that mistakes will be made.

An infected cow (and especially one infected with *Staphylococcus aureus*) will contaminate teat liners and can transmit infection to the next six to eight cows milked. Milking her last, or through a separate cluster, avoids this, provided that the mastitic cluster is disinfected, e.g. by soaking it in hypochlorite solution, before milking the next mastitic cow; otherwise infection can still be spread. This is often overlooked and is particularly important if the same cluster is also used to milk freshly calved cows, whose milk is being discarded because of colostrum or dry cow antibiotic.

Avoiding antibiotic residues is discussed in Chapter 15. When milking mastitic cows last or using a separate bucket and cluster, there is a lesser (or zero) risk of contaminating bulk milk with antibiotic from treated quarters. Some parlours have a dump line, as already described, through which colostrum and mastitic milk pass into a separate collection vessel. However, they may still have no separate cluster. This is very dangerous – you would need to avoid only one extra case of mastitis to pay for an additional cluster.

Technique for the administration of intramammary antibiotics

This should be done as carefully and as cleanly as possible. Rough handling can lead to teat canal damage, which in turn predisposes to mastitis. Administration of antibiotic through a contaminated teat end might introduce a yeast infection, which is particularly difficult to cure. The following procedure is suggested:

1. Carefully mark the cow to show that she has been treated with antibiotic. Most farms would put this as the final step, but, having known of cases where the wrong cow has been treated (and then not known which cow this is), it is recommended that this is done first. A variety of marker sprays, leg tapes and tail bands are used.
2. Ensure that both the milker's hands and the affected teat are clean and dry. Wash, if necessary, and then wipe dry with a clean paper towel.
3. Swab the end of the teat with methylated spirits or alcohol, until it is clean, i.e. until the swab can be rubbed across the teat end without becoming soiled. This may take more than one swab (Plate 12.2).



Plate 12.2. Swab the teat end until it is clean. Ideally gloves should be worn.



Plate 12.3. Inserting an intramammary antibiotic tube.

4. Remove the cap of the antibiotic tube and, without touching its tip with your hand, gently insert it into the teat canal (Plate 12.3). It is not necessary to insert the nozzle to its full depth: in fact, to do so could dilate the teat canal excessively, thereby cracking its protective keratin and lipid lining (see page 22) and predisposing the cow to mastitis. Partial insertion is also recommended (particularly for dry cow therapy) because it enables some antibiotic to be left in the canal itself. Some manufacturers are now producing antibiotic tubes with very small nozzles to achieve partial penetration and reduce teat-end damage (see Fig. 12.1). However, if the cow is nervous or difficult to handle, full penetration may be unavoidable.
5. Hold the tip of the teat between the finger and thumb of one hand and then use the other hand to massage the antibiotic up into the teat and udder cistern.
6. After administration, dip all four teats. This is important for both lactating and dry cow therapy. Tubing the cow, however carefully, dilates the teat canal and hence the extra protection of a dip is very valuable. In addition, it is probable that, even if only one quarter has mastitis, during the milking process infection may have spread to the teat orifice of the other

Fig. 12.1. Long and short nozzle tubes: the short nozzle (right) is preferable, as it will prevent unnecessary damage of the teat canal.

- three quarters. This infection can be removed by thorough teat disinfection.
7. Record the treatment in the medicines book (a legal requirement in the UK) and elsewhere as necessary (see pages 186 and 242).

Antibiotic Therapy

Is antibiotic treatment worthwhile?

This question has to be answered before selecting the antibiotic to be used. There is a body of opinion that considers that antibiotic treatment of some types of mastitis during lactation is simply not worthwhile. The reasons given for this are:

1. The response of *Staphylococcus aureus* infections to treatment is very disappointing (see Table 4.4). Although the clots and other clinical signs may disappear, there may be only a 20–35% bacteriological success rate.
2. Many cases of mastitis undergo self-cure, i.e. the infection is naturally eliminated

by the cow without treatment. This is particularly likely with coliform infections, where the response by the cow can be so dramatic that in some cases all bacteria may have been eliminated within 4–6 hours (see page 29). However, even coliforms occasionally establish themselves as chronic persistent udder infections.

3. The cost of the discarded antibiotic milk and the risk of antibiotic contamination of the bulk milk are both so high that they render treatment uneconomic.

Many papers have been written on this subject, some in favour of treatment, others against. It is the opinion of the authors that treatment is worthwhile. The main reasons for this are:

1. For certain infections the response to antibiotics is good.
2. Even for *S. aureus* the response rate is acceptable if the infection is detected early.
3. Response to treatment generally gives higher bacteriological elimination rates than self-cure.

Cure rates for streptococci and Staphylococcus aureus

The response of *Streptococcus agalactiae* and *Streptococcus dysgalactiae* infections to

treatment is generally good, although it is accepted that, as was shown in Table 4.4, complete bacteriological cure rates for *Staphylococcus aureus* can be poor. However, if it is a first-time infection, then cure rates even for *S. aureus* may be reasonable. Table 12.1 is taken from NIRD work in the 1960s and shows the bacteriological cure rates for clinical *S. aureus* mastitis identified by the milker and treated with the antibiotic cloxacillin. Note that, although overall bacteriological cure rates were disappointing at 38%, when cows were infected for the first time the cure rate was as high as 50%. This certainly makes treatment worthwhile. It was only those cows that had had previous unsuccessful treatments both during lactation and at drying off where the response rate was so poor, at only 6%. The 'response rate to all infections' includes all cows infected at the start of the trial plus those infected during the trial, and hence the overall lower response rate.

Self-cure rates versus antibiotics

The data in Table 12.2 are a summary of a range of clinical trial reports. They show that, for streptococci, treatment gives a considerably better bacterial elimination than self-cure. Even for *S. aureus* and coliforms, the cure rate following therapy was higher than for self-cure. The highest rate of antibiotic cure (35%) quoted in the table for *S. aureus* would relate to an average infection

Table 12.1. Response rate of clinical cases of *Staphylococcus aureus* mastitis to treatment with cloxacillin.

Previous unsuccessful treatments		New infections		All infections	
During lactation	At drying off	No. treated	% eliminated	No. treated	% eliminated
0	0	283	50	452	41
1	0	63	22	131	18
>1	0	48	10	102	8
0	One or more	12	25	52	17
1	One or more	8	12	49	12
>1	One or more	17	6	72	5
Total infections		431	38	858	28

Table 12.2. Spontaneous versus antibiotic cure rates for mastitis.

	Spontaneous cures	Antibiotic cures
<i>Staphylococcus aureus</i>	20%	20–35%
<i>Streptococcus agalactiae</i> and <i>S. dysgalactiae</i>	19%	36–95%
Coliforms	70%	71–90%

(see Table 12.1), and it is likely that, if all first-time clinical cases were treated aggressively, response to treatment would improve.

Overall benefits of antibiotic therapy

In summary, it is considered beneficial to treat clinical mastitis using antibiotic therapy for the following reasons:

1. It produces a more rapid and higher bacteriological elimination than 'self-cure'.
2. It reduces the probability of chronic recurrent infections.
3. It reduces the extent of milk yield depression.
4. It results in a more rapid return to an acceptable cell count, and hence to saleable milk.

If therapy saves one mastitis death, this pays for many treatments.

Mastitis control is a 'numbers game'. It involves reducing bacterial challenge at the teat end, rather than totally eliminating it. Although antibiotic treatment may not eliminate infection totally, it may reduce bacterial numbers such that the risk returns to manageable proportions.

Choice of Antibiotic

This is a huge subject and in itself consists of sufficient material to fill a whole book. This section gives simple guidelines only. It does not lay down specific rules for treatment, but rather points to the complexity of the sub-

ject and gives examples of a few of the factors involved.

As is the case with the purchase of a car, there are numerous manufacturers, each with their own range of products and each with its own unique claims of effectiveness. Also, like cars, there are many products on the market between which there is little to choose in terms of value for money.

The following criteria should be considered when making a choice of antibiotic for treatment:

- Antibiotic sensitivity of the bacteria involved.
- Ability to penetrate the udder.
- Ability to persist in the udder at a concentration sufficient to kill bacteria following single or multiple infusions.
- Effectiveness in the presence of milk.
- Whether it is bactericidal (killing) or bacteriostatic (arresting growth).
- Lipid solubility, plasma protein binding properties and pH level in solution.
- Withdrawal periods.
- Cost.

There is an excellent article by MacKellar (1991) that gives much more detailed information.

Antibiotic sensitivity and udder penetration

The following section discusses some of the properties of commonly used antibiotics and their spectrum of action. These are summarized in Table 12.3.

Penicillins

As a general rule, penicillins are effective against Gram-positive bacteria (staphylococci and streptococci, but not against Gram-negatives (coliforms, etc.). Most penicillins penetrate the udder reasonably well. Examples include:

- Penicillin G.
- Penethamate.
- Cloxacillin.
- Nafcillin.

Table 12.3. The spectrum of action of common antibiotics.

Antibiotic	Gram-positive bacteria ^a	Beta-lactamase Gram-positive bacteria ^{a,b}	Gram-negative bacteria ^c	Fastidious Gram-negative bacteria ^d
Penicillin	+	–	–	+
Penethamate	+	–	–	+
Cloxacillin	+	+	–	+
Amoxycillin	+	–	+	+
Amoxycillin + clavulanic acid	+	+	+	+
Streptomycin	–	–	+	+
Erythromycin	+	+	–	+
Cephalosporins (3rd generation +) ^e	+	+	+	+
Tetracyclines	+	+	+	+
Tylosin	+	+	–	+

^aGram-positive bacteria include staphylococci, streptococci, *Bacillus* species.

^bBeta-lactamase producers include *Staphylococcus aureus*.

^cGram-negative bacteria include *E. coli*, *Pseudomonas*, *Klebsiella*.

^dFastidious Gram-negative bacteria include *Pasteurella*, *Moraxella*, *Bordetella*, *Actinobacillus*.

^eCephalosporins include cephaloperazone and cefquinome. Third- and fourth-generation cephalosporins have a greater effect against coliforms.

Penethamate has better udder penetration than the rest of the group, and is marketed as a specific treatment against *Streptococcus uberis*. When in the udder, it undergoes a change to penicillin G before it starts its bacterial killing. As it is not effective against coliforms or beta-lactamase strains of *Staphylococcus aureus*, it needs to be used selectively.

Unfortunately, many (approximately 70%) mastitic strains of *S. aureus* are now penicillin-resistant, because they have adapted to produce the enzyme beta-lactamase. This enzyme breaks down the beta-lactam ring structure of penicillin. Cloxacillin and nafcillin are effective in the presence of beta-lactamase and, despite widespread use in dry cow therapy preparations over the past 40 years, to date, no staphylococci have been found that are resistant to these drugs. Cloxacillin and nafcillin are therefore suitable treatments for dry cow infections caused by staphylococci (see Table 12.6). However, they are not effective against coliforms, which are Gram-negative.

Some penicillins have been synthetically modified so that they have some effect against coliforms, namely:

- Ampicillin.
- Amoxycillin.

However, these two drugs are still not effective against beta-lactamase producing staphylococci. Clavulanic acid is an irreversible inhibitor of beta-lactamase, and by combining clavulanic acid with amoxycillin, provides a product which should be effective against the vast majority of mastitic bacteria. Other combination products that should, theoretically, achieve good udder penetration and be effective against all organisms are a mixture of cloxacillin (kills Gram-positives and beta-lactamase producing staphylococci) plus amoxycillin (kills Gram-positives and Gram-negatives) or cloxacillin plus ampicillin.

Aminoglycosides

The aminoglycoside group of antibiotics, are:

- Streptomycin.
- Neomycin.
- Framycetin.

They are active against coliforms and effective against beta-lactamase producing

staphylococci. They have poor penetration of the udder tissue. One of their strengths is that they are relatively inexpensive. As penicillins achieve good penetration of the udder, products containing penicillin and streptomycin are often used in combination.

Cephalosporins

Cephalosporins are active against Gram-negative and Gram-positive bacteria, including beta-lactamase producing staphylococci, although penetration of the udder is not as good as with the penicillins. 'Second generation' cephalosporins, for example cefuroxime, have improved activity against Gram-negatives, whilst 'third-generation' products, for example, cefquinome, have the added advantage of some effect against *Pseudomonads*.

Tetracyclines

Tetracyclines are broad-spectrum, that is, they are effective against Gram-negative and Gram-positive bacteria, with some activity against beta-lactamase producing staphylococci. However, penetration of the udder tissue is limited (although this can be overcome by using very high dosages) and resistance may occur with coliforms.

Response of coliforms to antibiotics

Coliforms have a variable sensitivity to antibiotics, and standard texts (Tyler and Baggot, 1992) show how wide this can be. For example, in two reports the range of sensitivity to tetracycline varied from 23 to 68%. For ampicillin the range was 35–64%. As a herd outbreak of *E. coli* mastitis (the most common of the coliform types) always involves a range of different strains of *E. coli*, precise guidelines regarding the most effective antibiotic to use on the basis of the sensitivity of a single bacterial isolate cannot be given. Gentamycin is effective against both *E. coli* and *Klebsiella*, but the cost of treatment is usually prohibitive, with a very long withdrawal period.

Whichever preparation is decided upon for routine use (and this must be a joint decision between the farmer and his vet), the following factors are important when selecting a preparation for routine 'first line' treatments:

- Due to the increasing incidence of coliform mastitis (see Table 4.2), lactation treatments should always involve a broad-spectrum antibiotic, e.g. one that is effective against Gram-positive (i.e. staphylococci and streptococci), Gram-negative (e.g. coliforms) and beta-lactamase producing organisms.
- Although preparations used in dry cow therapy were originally aimed primarily at staphylococci and streptococci, cover against coliforms is an advantage.

Effectiveness in milk

Although the antibiotic sensitivity plate test is used to assess response, many antibiotics are less effective in the presence of milk than the test suggests. For example, the ratio for oxytetracycline is 4:1. This means that oxytetracycline is four times less effective in the presence of milk than it is in the plate test. Other examples include streptomycin (5:1), erythromycin (7:1) and trimethoprim/sulfadiazine (500:1). However, these figures were obtained in experiments using whole milk and results may not apply to mastitic milk, which has a higher pH than uninfected milk.

Bactericidal and bacteriostatic antibiotics

Antibiotics vary in the way they act. Some, for example, the penicillins, specifically kill bacteria (they are bactericidal). However, others, for example, the tetracyclines, simply prevent bacterial growth and multiplication (they are bacteriostatic) and rely on the cow's own defence mechanisms to overcome the infection. If the cow is freshly calved or if she is very sick, the activity of her defence mechanisms may be compromised, and bacteriostatic antibiotics may not be appropriate. In such cases, the use of

bactericidal antibiotics may be preferable, as the innate immune response is then less important. The counter-argument to this is that bactericidal antibiotics may lead to sudden bacterial death and the release of endotoxins (especially with coliform infection; see page 45), making the clinical condition more severe.

Acidity and lipid solubility

Antibiotics are either acidic or basic, depending on their pH when in solution. Because the pH of milk (6.7) is lower than the pH of blood (7.4), drugs such as tylosin, erythromycin, trimethoprim and tilmicosin, which are naturally more alkaline, will be drawn into the mammary gland and penetrate the udder. They are likely to be most effective in the active udder, because during the dry period the pH difference is less. It is for this reason that the use of these products is recommended more during lactation, at drying off or at the start of the next lactation, rather than in the dry, inactive udder.

When the udder becomes inflamed, as in severe mastitis, the pH of milk increases towards the pH of blood, and this pH trap becomes less important.

The lipid solubility and the degree to which antibiotics bind to proteins in blood will also affect their ability to penetrate the udder, particularly following intravenous or intramuscular injection.

Intracellular effects

Certain bacteria, for example, *Staphylococcus aureus* and *Streptococcus uberis*, are able to penetrate and exist inside neutrophils and macrophages, where they are protected against the action of many antibiotics. They remain in a quiescent intracellular state until they become active at a later date to produce a repeat case of mastitis. Some manufacturers claim that certain antibiotics can penetrate cells and reach quite high intracellular concentrations, thereby eliminating the carrier state. Examples include tylosin, which is said to

reach an intracellular concentration ten times higher than that of the surrounding tissue fluid.

Withdrawal period

Post-treatment milk- and meat-withholding periods are stated on the product and must always be observed. Although the majority of the antibiotic remains in the treated quarter, some will diffuse into the bloodstream, pass around the body and be deposited back into the untreated quarters. This is because there is a very high blood flow through the udder (400–500 litres of blood for each litre of milk produced). When the affected quarter is inflamed, flow rates may be even higher. Milk must therefore be discarded from all four quarters, even if only one quarter is being treated.

The withdrawal period given on the tube relates to the use of that tube as stated in the instructions. If the herdsman decides to use an increased frequency of tubing, administers two tubes at the first treatment or injects the cow with antibiotic in addition to tubing her, then this could affect the required withdrawal period. Further detail is given in Chapter 15. If in doubt, ask your vet. In the UK a few products, e.g. ceftiofur, have a licence for combination therapy, and milk-withholding periods are specified. Further details are given on antibiotic residues in Chapter 15.

The ability of an antibiotic to persist in the udder at bacteria-killing concentrations depends partly on the chemical nature of the antibiotic and partly on its formulation. For example, products with a long persistency, as would be required for dry cow therapy, are formulated in slow-release oils or waxes, or manufactured with a much smaller particle size. Conversely, aqueous preparations are generally shorter-acting, with a low persistency but a short milk-withholding period.

Benefits of Early Treatment

If an initial infection can be treated early post-infection, then response to therapy is

likely to be improved. This was demonstrated for the field cases of *Staphylococcus aureus* described in Table 12.1. Early treatment has also been shown to be more effective under experimental conditions (Milner 1997). Teats were dipped in a culture of *Staphylococcus aureus* or *Streptococcus uberis*, and various methods were used to detect signs of infection. The first evidence of infection was bacteria cultured from the milk (approximately 1 day after exposure), the second an increase in milk cell count (2 days), the third a rise in milk conductivity (3 days) and the fourth as clinical signs (4–5 days after infection). It was found that, if treatment was instigated early, i.e. when changes in milk conductivity were detected, then both clinical and bacteriological response rates improved. Results are shown in Table 12.4. Fewer tubes were used for treatment, and milk yield depression was less if treatment was instigated early.

This is good evidence to promote:

- Foremilking, as this enables the early detection of clinical cases and hence more effective treatment.
- Treatment of cows with rising cell counts, i.e. before they become clinical.

The same experiment also showed that it took around 2 weeks for the cell count of a conventionally detected and treated infected quarter to fall below 400,000, despite the fact

that clinical and bacteriological cure had been achieved well prior to this. The significance of a prolonged milk discard from such cows is obvious.

Combination Antibiotic Therapy (Concurrent Injection and Tubing, Aggressive Therapy)

In some countries, mastitis is treated only by parenteral therapy (i.e. by injection), and in these countries treatment is considered to be equally effective whether antibiotic is administered as an intramammary tube or by injection.

In the EU, injections are increasingly administered at the same time as intramammary tubes. This is known as ‘combination therapy’ and, if continued for a longer period of time, or at a higher dose level, it is referred to as ‘aggressive therapy’. It has been estimated that the surface area of the udder is 25 sq.m per quarter and, if this is correct, then it is perhaps not surprising that intramammary therapy does not reach all parts of the udder.

Results of a trial demonstrating the advantages of injecting penicillin at the same time as using amoxycillin intramammary tubes in the treatment of *Staphylococcus aureus* mastitis is shown in Table 12.5), where cure rates increased from

Table 12.4. Experimentally it has been shown that early treatment (evidenced by an increase in milk conductivity) of mastitis produces a more effective response than conventional treatment (evidenced by clinical changes in the milk) (Milner, 1997).

	Conventional treatment (clots seen)		Early treatment (conductivity)	
Experimental infection with	<i>Streptococcus uberis</i>	<i>Staphylococcus aureus</i>	<i>Streptococcus uberis</i>	<i>Staphylococcus aureus</i>
Clinical cases seen	8/8	6/6	0/8	0/8
No. of tubes used to resolve clinical signs or reduce conductivity	8	10	6	6.5
Number of cows with low yields at 14 days post-treatment	7/8	1/6	3/8	1/8
Somatic cell count at treatment	12 million	4 million	2 million	2 million
Number of milkings (days) before cell count of affected quarter fell to <400,000	31 (14.5 days)	35 (17.5 days)	17 (8.5 days)	14 (7 days)

Table 12.5. Advantages of injecting penicillin intramuscularly concurrently with amoxicillin intramammary tubes. (From Owens *et al.*, 1988.)

	3 days amoxicillin intramammary	3 days amoxicillin intramammary + penicillin IM
No. of quarters	40	35
% cured	25	51

25 to 51% when additional parenteral therapy was used. This treatment would not be effective against beta-lactamase producing staphylococci.

Aggressive primary therapy certainly reduces the incidence of recurrent and chronic infections, and will be especially used in animals that are pyrexia (have a high temperature) or sick. Aggressive therapy, e.g. 5–7 days of concurrent intramammary tubes and injectable antibiotic, has also been suggested for the treatment of chronically infected cows or cows with a high cell count. Others have suggested 5 days of ‘milking cow’ intramammary treatment, followed by drying off with dry cow antibiotic treatment.

Drying Off Quarters

It is known that in some 5% of quarters existing infections ‘self-cure’ during the dry period, and there is evidence that the longer the dry period the greater is the probability of self-cure. From this, a technique for the treatment of chronic recurrent cases of mastitis has developed (Blowey and Deyes, 2005). Quarters that have had four or more cases of clinical mastitis in a lactation are best dried off, so that the cow can continue to be milked on three quarters. While there are obvious disadvantages of having three-quartered cows in the herd, if the alternative to drying off a quarter is to cull the cow (a very expensive option), then clearly a three-quartered cow is acceptable.

The main advantages of drying off quarters are as follows:

- The cow continues in production.
- Infected milk no longer increases the SCC and TBC/Bactoscan of the bulk milk.
- It reduces the risk of spreading infection to other cows.
- Following a prolonged dry period, plus dry cow antibiotic therapy, the quarter often returns to normal production in the next lactation.
- It does not require the cost of antibiotics used in aggressive therapy.

The technique used by most herdsmen is to give the cow one final intramammary treatment using lactating tubes, e.g. for her fourth clinical case, then simply stop milking the affected quarter. Do not use dry cow therapy at this stage because of the risk of antibiotic residues.

When the remaining three quarters are then dried off at the end of lactation, all four quarters are given dry cow therapy plus an internal teat sealant. Some herdsmen also use an internal teat sealant when the quarter is dried off. In a survey of 4326 cows in 16 dairy herds using a variety of techniques to dry off the quarter, 125 cows had had quarters dried off, and the overall success rate, defined as cows returning to normal production in the next lactation, was 66% (Blowey and Deyes, 2005). However, if only those cows that were treated with antibiotic at drying off the quarter and then again at drying off the cow were included, the success rate rose to 92%. The majority of these cows also had a low cell count and no major pathogens in the next lactation.

Partial drying off of quarters has been attempted as a treatment. A chronic recurring clinical case is treated for 3–5 days and then not milked for 3–4 weeks. These cows will come back into production again in the same lactation, although milk needs to be discarded for the first few days because the initial cell count will be very high. This technique is less effective than the longer dry period.

Temporary cessation of milking is a good technique for cows with teat damage, and is discussed later in this chapter.

Drying off the affected quarter or the whole cow, or culling, is still the only certain way of removing chronic carriers.

Resistance of *Staphylococcus aureus* to Treatment

Staphylococcus aureus (also known as coagulase-positive staphylococci) gives a notoriously poor response to treatment. This was demonstrated in Table 4.4 and again in Table 12.2. Even with dry cow therapy, response is poor (Table 12.7).

This is particularly the case in older cows, where the infection has been present in the udder for some time. (Tables 12.1 and 12.8).

There are several reasons for the disappointing response of staphylococci to treatment. These include:

- *S. aureus* forms abscesses within the udder. A typical example is shown in Plate 4.2. These abscesses are often surrounded by a thick fibrous capsule. This prevents the antibiotic from reaching the bacteria, or insufficient antibiotic concentration is achieved within the abscess to kill bacteria effectively.
- Some strains of *S. aureus* can live within cells such as macrophages. Most antibiotics are only able to circulate in the body fluids surrounding cells and are not able to penetrate the cell itself. Those staphylococci that live inside the cells are hence protected from the majority of antibiotics. (A few antibiotics can penetrate cells – see previous section – but in the UK these are not yet available as intramammary preparations.)
- Many strains of *S. aureus* produce beta-lactamase, making them resistant to certain types of penicillin. Even when effective antibiotics are used, however, response to treatment is still very poor.
- Some strains of *S. aureus* can persist in a state of bacterial dormancy with a mucoid capsule and completely cease multiplying. In this state they are not killed by antibiotics, although they can reactivate at a later date.
- L forms of *S. aureus* may occur. These are bacteria that do not have a proper cell wall and therefore most antibiotics will not kill them. This includes

cloxacillin and other antibiotics that are effective against beta-lactamase producing strains. (The antibiotic novobiocin is effective.) However, there is some doubt whether L forms of *S. aureus* are produced under the conditions present in the udder.

One of the difficulties with assessing response to treatment for *S. aureus* is that, following a course of antibiotic therapy, many quarters initially appear to have responded and no bacteria are isolated from a milk sample. However, this is simply because no *S. aureus* are present in that particular sample. If the same cow is sampled at a later date, bacteria may have been released from an abscess or an intracellular site, or they may have been revived from their dormant state, and it is then found that the cow is still infected, i.e. treatment was not effective.

This is clearly demonstrated in Table 12.6. When cows infected with *S. aureus* were sampled 16 days after treatment, it was found that bacteria were still present in only 43% of treated quarters, so the success rate was 57%. However, if sampled again at 30 days, bacteria were isolated from 56% of the treated cows, and this increased to 62% of cows (only a 38% response) if sampled at 60 days post-treatment.

These results were obtained in a trial using combined intramammary and injectable antibiotics. If only intramammary treatment was used, then the response at 60 days was even lower (27%).

Table 12.6. The results obtained in trials assessing the response rate of *Staphylococcus aureus* to treatment with antibiotics.

No. of days after treatment	% cows with <i>Staphylococcus aureus</i>	% response to treatment
16	43	57
30	56	44
60	62	38

Blitz Therapy Against *Streptococcus agalactiae*

The response of *Streptococcus agalactiae* to treatment is totally different from that of *Staphylococcus aureus*. *Streptococcus agalactiae* is very sensitive to many antibiotics and response to treatment, even during lactation, is generally very good (Tables 12.2 and 12.4). This allows a system known as 'blitz therapy' to be used in the elimination of *S. agalactiae* from milking herds. Control of *S. agalactiae* is also discussed on pages 42–43.

Blitz therapy involves the use of intramammary antibiotics infused into all four quarters of every milking cow in the herd. Two forms are used: total – where the entire milking herd is treated – and partial – where only selected animals are treated, e.g. those that have a high cell count and are culture-positive. Bacteriology of high cell count cows is essential to confirm that *S. agalactiae* is the primary cause of the high cell counts. It is not acceptable to simply rely on positive bulk tank samples, as these do not give sufficient detail on the extent of the infection.

In order for this technique to be successful, *S. agalactiae* must be the major organism responsible for the mastitis problem, and all aspects of milking hygiene must be reviewed. As discussed earlier in this book, the presence of *S. agalactiae* in a herd is an indication that there is a fault in basic hygiene, e.g. inappropriate postdipping and/or dry cow therapy, as these measures will normally eliminate the infection from a herd.

Future replacement cows may be a possible cause of reinfection, and many recommend that newly purchased cows are treated with intramammary antibiotics before they join the milking herd. Any dry cows not being milked at the time of the 'blitz' should be given dry cow therapy if it has not been administered previously.

Scrupulous hygiene is needed to 'blitz' a herd. Extra help is needed in the parlour, and it is essential that teat ends are thoroughly disinfected before any tubes are administered. Do not remove the caps of the

intramammary tubes in advance (e.g. for speed of administration in the parlour) as this increases the risk of contamination prior to infusion. There have been several documented cases where severe outbreaks of mastitis have followed blitz therapy, and these can often be traced back to suboptimal hygiene. If the introduced organisms are yeasts or fungi that do not respond well to treatment, the overall herd situation can be made much worse.

Blitz therapy is not always successful. There are many reasons for this, for example:

- Infected cows may be reintroduced into the herd.
- The milkers become careless with basic parlour hygiene and allow residual infection to spread within the herd.
- Dry cows may not have received dry cow therapy and so may reintroduce infection into the milking herd.
- When using partial or selective blitz therapy, some infected cows are not selected for treatment and so a reservoir of infection remains within the herd.
- Other organisms, in addition to *S. agalactiae*, were the cause of mastitis, and the therapy chosen was not effective against these other organisms, or they showed a poor response rate
- A total error in diagnosis, in that *S. agalactiae* was not the main pathogen in the herd.
- Infection was introduced during the mass tubing, and this led to increased mastitis.

Although blitz therapy is a useful technique, therefore, it should only be undertaken following a thorough investigation of the herd problem, and even then with strict attention to aseptic techniques.

Supportive Therapy

In addition to antibiotics, a wide range of other treatments have been suggested for different types of mastitis. These include fluid therapy and supportive therapy, such as the

use of anti-inflammatory agents and oxytocin.

Fluid therapy

Toxins, particularly those produced by coliform and gangrenous staphylococcal mastitis, can cause a state of shock and affect many body organs. Blood vessels dilate and as a result blood pressure starts to fall. Falling blood pressure leads to poor circulation and consequently poor tissue perfusion with blood. The animal appears dehydrated, as its body fluids are in the tissues and not in its circulation. Dehydration may frequently reach 7–10% of body weight. This means that, for an average 600 kg cow, 40–60 litres of fluid need to be replaced to restore the circulation to normality. Dehydration further increases the feeling of malaise and general ill health of the cow. Administration of fluids, especially in animals that will not drink, can be of considerable benefit.

Fluids may be administered in a variety of ways.

Intravenous administration

As the rate of intravenous administration is slow, this can be a time-consuming and therefore expensive exercise. Firm fixation of the intravenous catheter is required, and as such should only be undertaken by vets. Sometimes a small garden pump is used. An effective intravenous solution can be prepared by mixing a proprietary packet of oral electrolyte powder with warm tap water. This should help to restore normal metabolic activity.

Some people recommend the intravenous administration of 2.0 litres hypertonic (concentrated) salt solution (70 g per litre NaCl). This stimulates extreme thirst in the cow and encourages her to drink. However, if the technique is used in recumbent cows, it is obviously vital that water is fully accessible. Extreme care is needed during the infusion to monitor for shock.

Oral administration

Provided that the cow will drink, one of the simplest ways of administering oral fluids is via a watering can, as in Plate 12.4. If electrolyte solutions (that is, calf scour formulations) containing bicarbonate are given, they may stimulate closure of the oesophageal groove, transferring the fluid directly into the abomasum, where its absorption is more effective.

A faster way of administering large volumes of fluid (e.g. 10–20 litres) is by using an oral pump, as shown in Plate 12.5. The tube is protected from the cow's teeth by flexible metal rings and held in place by nose clips ('bulldogs'). Fluids are pumped into the rumen via a stirrup pump from a bucket.



Plate 12.4. Oral fluids are easily administered using a watering can.

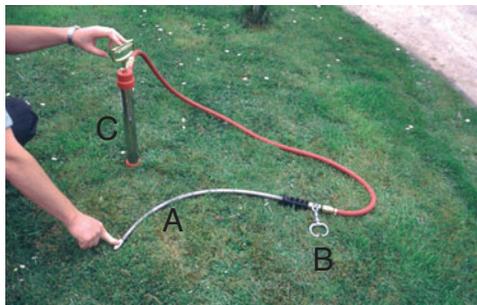


Plate 12.5. Oral pump fluid apparatus: (A) flexible metal tube to insert through mouth and into oesophagus, (B) nose clips hold the tube in position, (C) pump, which is placed into the bucket of oral fluids.

Provided that cows will drink voluntarily, it is difficult to see how additional benefit is obtained from forcible fluid administration. However, one point is important and that is that water (preferably warm) should always be readily available to a sick cow. If she is recumbent, this means offering her warm water five to six times daily, within easy reach. Cows will also readily drink electrolyte solutions.

Anti-inflammatory drugs

In addition to the use of fluids, shock can also be counteracted by the use of anti-inflammatory drugs, such as flunixin, meloxicam, aspirin or cortisone. These drugs are not licensed for use in dairy cows in all countries and therefore the specific instances for their use should be confirmed with the regulatory authority. Cortisone given locally or parenterally reduces swelling and the inflammatory response, but it may also allow greater bacterial multiplication. However, there are no experimental data to support this.

Many commercial intramammary products contain 10 mg prednisolone (a form of cortisone), which is aimed at reducing the hardness and swelling in the affected quarter. This perhaps permits better antibiotic penetration. Some cows with a typical hard quarter 4 to 5 days after a coliform infection respond well to larger doses of cortisone, either by injection or by infusion into the udder. Intramammary infusion of cortisone may not be legal in some countries and abortion may be induced.

Administration of calcium

Some sick cows are naturally hypocalcaemic (have low blood calcium) and, if dealing with a case of acute mastitis at calving, it is not always possible to be sure if a concurrent hypocalcaemia is present. Calcium is also said to aid detoxification processes in the liver. For this reason, 400 ml of calcium borogluconate, possibly mixed with glucose

(as dextrose), is sometimes given to sick mastitic cows.

Administration of glucose

Cows with acute *E. coli* mastitis can be hypoglycaemic (have low blood glucose) and may benefit from intravenous dextrose infusions (oral glucose is of no value, as it is destroyed in the rumen). In addition, the phagocytic activity of macrophages in the udder, i.e. the way in which white cells engulf bacteria is relatively poor, due to low oxygen concentrations in milk.

It has been suggested that the infusion of dextrose into the udder promotes phagocytic activity. Evidence supporting this is limited. Great care needs to be taken to ensure that the teat canal is not damaged and that yeasts and other organisms are not accidentally infused, making the mastitis worse.

Continual stripping and oxytocin

The toxins produced by mastitic bacteria either are absorbed by the cow (possibly making her ill) or can be stripped out of the udder manually. Clearly the latter is preferable; hence the importance of regular stripping of the affected quarter, maybe six or eight times daily, or more for very sick cows. Some people suggest stripping every 30–60 minutes until the cow is better.

The efficiency of stripping can be improved by giving oxytocin injections, which help to eject milk from the deeper parts of the gland. Natural let-down is highly unlikely to occur in a sick cow, and even if the affected cow is very ill, there is likely to be a considerable amount of residual milk in the alveoli and small ducts and this, plus its toxins, could be removed using oxytocin. Used at a higher dose rate, oxytocin is also said to promote neutrophil movement out of the capillaries, and hence aids in the inflammatory response.

Others have suggested leaving a strong suckler calf with the cow to do the stripping for you. However, if the quarter is painful the

cow might resent the calf sucking and only allow the normal unaffected quarters to be stripped out. In addition, the calf is less likely to suck a quarter containing bitter milk. The value of this technique is therefore a matter of conjecture.

Non-antibiotic intramammary infusions

The use of iodine preparations against yeast and fungal mastitis was described on page 55. Others have suggested infusing infected quarters with 20 ml of natural live yogurt at 12-hourly intervals for 2 to 3 days. The objectives are to decrease the raised pH of mastitic milk and to eliminate residual mastitis organisms by the probiotic effect of natural lactobacilli in yogurt. The procedure has apparently been used successfully in treating yeast and coliform infections.

Topical preparations

Products such as Cai-Pan Japanese peppermint oil ('Uddermint') have been recommended for topical application (that is, to areas of the udder skin). They certainly stimulate warmth in the skin, leading to increased blood flow, but whether this can be translated into increased blood flow through the udder is difficult to assess. If such products improve the feeling of well-being in the affected cow (udder massage can be soothing) and lead to attention being paid to (and stripping of) the affected quarter, then they are worth using. As with so many mastitis preparations, clinical trials are difficult to carry out because a proportion of cases self-cure.

Homeopathy

Homeopathic medicine states that a substance that produces the symptoms of an illness can also be used in the treatment of any illness that causes similar symptoms. Homeopathic remedies are all obtained from natural sources. The system relies on a series of dilutions being made, one part

mother tincture to 99 parts of water and alcohol mixture. The mother tincture is an extract of natural substances, e.g. of a culture of the bacteria that originally caused the symptoms. Dilutions are repeated, and it is said that all impurities are filtered out, leaving only a more potent preparation with more energy. It is apparently this 'energy' and not the material dose of the initial preparation that is critical and many 'remedies' have been diluted down to well below submolecular levels.

The value of homeopathic mastitis therapy remains questionable. At present it has the attraction of offering treatment without a milk withdrawal period. Lay homeopathic advisers stress the concurrent need to prevent infections and promote the basic principle of sealing the teat canal between lactations, which is also the principle on which mainstream dry cow preparations are based.

Homeopathic nosodes (remedies) used to 'prevent' mastitis are administered in drinking water, by drenching and even spraying them into the vulva. Although there are plenty of anecdotal reports showing a benefit, specific trial work is lacking. The one controlled trial carried out (Egan, 1995) showed no benefits. Perhaps, if the herdsman adds a 'remedy' to the drinking water each day, he 'thinks' mastitis each day, and this also results in improved control.

Dry Cow Therapy

The physiology of the udder at drying off and the importance of new infections during the dry period were discussed in Chapter 4, which needs to be read in conjunction with the following. This section deals with the control options available.

Although opinions may vary concerning the value of treatment during lactation, there are few who would doubt the wisdom of dry cow therapy. Dry cow therapy consists of two parts:

1. The administration of a long-acting antibiotic into each quarter at drying off

to remove existing infections and prevent some new infections.

2. The infusion of an internal wax sealant into the canal and base of the teat to prevent new infections.

Long-acting antibiotics

The aim of using long acting antibiotics at drying off is twofold, namely: (i) it reduces the reservoir of contagious organisms that have accumulated over the previous lactation; and (ii) it reduces the number of new infections likely to be contracted during the ensuing dry period.

In Fig. 4.3, it was shown that most new infections occur during the first and the last 2 weeks of the dry period. Dry cow therapy will help to reduce the number of new infections at drying off, but by the time of the next calving, antibiotic levels will be quite low and probably ineffective (the exception to this is a product containing the antibiotic framycetin, which it is claimed, persists at bacteria-killing levels throughout).

In a comparison of treated and non-treated cows, Berry and Hillerton (2002) showed that cows given dry cow therapy:

- Produced 179 kg more milk during the first 120 days of the next lactation.
- Had a tenfold reduction in clinical mastitis in the dry period.
- Showed a threefold reduction in infections at calving.
- Had a threefold reduction in clinical cases in the first 21 days after calving.

The benefits of dry cow therapy are therefore obvious:

- No milk is discarded.
- Response to treatment is much more effective during the dry period than during lactation (Table 4.4). This is partly because much higher doses of antibiotic can be infused into the dry quarter without concerns over withholding milk. The difference is particularly apparent for *Staphylococcus aureus*, where response to lactation treatment may be especially poor.

- A concurrent 'self-cure' takes place during the dry period, which is why many quarters dried off early return to normal milk production in the next lactation.
- Longer-acting antibiotic preparations can be used to improve the efficacy of action. Slow-release dry cow products are prepared by incorporating the antibiotics into waxes, by using benzathine or aluminium salts, or by manufacturing a product with a much smaller particle size.
- It provides some protection against summer mastitis (see Chapter 13).

Dry cow preparations should be effective against *S. aureus* (including beta-lactamase producers), as these are carried in the udder from one lactation to the next, and against coliforms and *Streptococcus uberis*, which may be contracted as new dry period infections. Table 12.7 shows the results of a 1990s Irish survey of 294 cows (1176 quarters) found to be infected at drying off. It is likely that *Staphylococcus aureus* levels would be lower than this in most herds under current UK conditions.

Table 12.7. Intramammary pathogen types and response to treatment in cows at drying off. (From Meany, 1992.)

Bacterium	No. of quarters	% of total	% response
<i>Staphylococcus aureus</i>	259	61	48
Other staphylococci types	27	6	78
Streptococci	118	28	78
Combined staphylococci and streptococci	12	3	42
<i>E. coli</i>	5	1	–
Non-specific	4	1	–
Total	425	100	

S. aureus was once by far the most common organism isolated at drying off, although the incidence of *Streptococcus uberis* is now increasing in some countries. The drugs most commonly used in dry cow preparations are therefore those that are

most effective against *Staphylococcus aureus*. These are:

- Cloxacillin.
- Cephalosporins.
- Nafcillin.
- Combination penicillin/streptomycin products.

Some people suggest that dry cow preparations should be changed occasionally, to avoid the development of antibiotic resistance. As no strains of *S. aureus* have ever been found resistant to cloxacillin or cephalosporins, no benefit is likely to be obtained from changing the antibiotic, although additional antibiotic cover to prevent new coliform infections during the dry period would be logical.

Frequently, failure of dry cow therapy is not the fault of the drug, but rather of the way in which it is used and administered. Despite the fact that dry cow tubes contain large amounts of antibiotic, they will not necessarily eliminate bacteria accidentally infused into the udder at the time of administration as a result of poor hygiene.

Treat all quarters

All quarters should be treated at drying off (blanket dry cow therapy) and not just those which had clinical mastitis during the previous lactation, or those with high cell counts (selective dry cow therapy). This is because:

- Many cows infected during lactation never show clinical signs.
- Some cows may become infected but do not show a particularly high or consistently elevated cell count.
- Even in cows with quite low cell counts, e.g. 200,000, this may be the result of three quarters with a cell count of close to zero, but one quarter obviously infected with a cell count of close to 800,000. Chronic forms of *Streptococcus uberis* are one example of this. Even *Staphylococcus aureus* does not consistently produce high cell counts (see Table 4.5).
- Every attempt should be made to prevent the establishment of chronic *S. aureus*

infections. Table 12.8 shows that response to treatment declines with age. The practical conclusion to be drawn from this is to ensure that even first lactation heifers are given dry cow therapy. The longer their udders can be kept free of *S. aureus*, the better.

- Cows are 15–20 times more likely to contract new infections in the first 2 weeks (and last 2 weeks) of the dry period. Antibiotic cover of all cows during as much of the dry period as possible is therefore likely to be highly beneficial. Because of the risk of antibiotic contamination of the milk after calving, full protection clearly cannot be given during the last 2 weeks of the dry period.

In one NIRD trial, in which dry cow therapy was not used, it was shown that:

- 25% of all quarters were infected at drying off.
- 5% of these quarters shed their infection naturally, i.e. underwent self-cure.
- Another 10% contracted new infections during the dry period. Hence 30% of quarters were infected at the start of the next lactation ($25 - 5 + 10 = 30$).

In another trial, carried out in the Netherlands (Schukken *et al.*, 1993), 68 cows had only two quarters infused with dry cow therapy at drying off, while their other two quarters were left untreated. During the dry period, there were ten cases

Table 12.8. Response of *Staphylococcus aureus* infections to treatment during the dry period. First and second lactation animals respond much better than older cows. (From Meany, 1992.)

Lactation number	No. of cows treated	% response to treatment
1–2	51	63
3–5	99	37
>5	40	33
	Total 190	Average 43

of clinical mastitis in the untreated quarters, seven of which occurred in the first 2 weeks after drying off. Only one case of mastitis occurred in the treated quarters. This is further evidence of the benefit obtained from giving dry cow therapy to all animals.

Some say that continued use of dry cow therapy will produce such low cell counts that cows become excessively prone to *E. coli* mastitis. This is unlikely to be correct. The reasons for this are given on page 31.

Teat sealants

One of the major advances in mastitis control over the past 10 years is undoubtedly the introduction of commercially available internal teat sealants. This has dramatically reduced the incidence of mastitis in early lactation.

Two types of teat seal are available to reduce dry period infections: the external film sealant, which provides a flexible barrier film over the teat end for up to 7 days, and the internal teat canal wax plug. The external seal is no longer commonly used and will not be discussed in detail, although it can be used in the late dry period if no internal seal has been administered.

The internal seal is by far the most effective and the most commonly used. It is a bismuth salt in a wax base that is infused into the teat canal at drying off. It has no antibacterial properties and hence strict hygiene during administration, as described above, is essential. Whereas antibiotic is massaged up into the teat and gland cistern, when administering a teat sealant the base of the teat should be constricted between finger and thumb so that the sealant is confined to the teat itself and 'sits' in the base of the teat just above the canal. Failure to do this may lead to prolonged excretion of sealant in the milk during the next lactation and possibly the formation of 'black spot' in cheese, small foci of bismuth leading to harmless discolorations.

Teat sealants are commonly used in addition to dry cow antibiotic tubes, in which case the antibiotic should be administered first. The effects of teat sealants and

dry cow antibiotics on the incidence of mastitis in the next lactation are additive, i.e. it is not a question of dry cow therapy or teat sealant, but rather of dry cow therapy and teat sealant. In a New Zealand study of 1200 cows in seven herds, all with bulk milk cell counts of less than 200,000, Woolford *et al.* (1998) divided the cows into four groups. The first was a negative control, i.e. no treatment at drying off. The second and third were treated with either dry cow antibiotic (cephalonium) or a teat seal, and the fourth group was given a combined antibiotic (cloxacillin) and teat seal. Results confirmed that:

- All treatments reduced clinical mastitis in the next lactation by 50% compared with the untreated controls.
- Incidence of new intramammary infections decreased by tenfold.
- The combination of antibiotic and teat seal gave the best protection.

A similar trial in the UK, comparing dry cow therapy plus teat seal with teat seal alone, showed a 30% reduction in the incidence of new infections in the first week of the subsequent lactation.

Administration of dry cow tubes

Cows should be dried off abruptly and removed from the milking herd immediately, even if they are still giving 20–25 litres of milk a day. If they continue to go through the parlour, milk let-down will be stimulated, i.e. the alveoli will contract, expelling milk and inhibitor protein (see page 18), thus synthesizing more milk. In addition, if dry cows are left running with the main herd, there is a risk that one of them will be milked inadvertently, leading to antibiotic contamination of the bulk tank. It should not be necessary (or advisable) to limit food and water severely, although for higher-yielding animals it is logical to stop feeding concentrates 4–5 days before drying off, and possibly change groups.

Cows with lower yields at drying off are more likely to produce an effective teat seal.

Gradual drying off is contraindicated for two reasons. Incomplete milking allows bacterial proliferation in the teat canal before therapy and hence predisposes to mastitis. In addition, cows left unmilked for 1–2 days develop large increases in cell count. In one trial (Meany, 1992), a group of late lactation cows, with an average cell count of 237,000 cells per ml, were monitored. When they were left unmilked for 2 days, the cell count increased to 540,000. After they had been unmilked for 6 days, the average cell count increased to 5,600,000 and in one individual cow it rose to almost 17 million. Abrupt drying off is therefore important to maintain low cell counts. Similarly, if a damaged teat is left unmilked for 7–14 days and allowed to heal, the first few milkings should be discarded.

Infusion technique

Dry cow tubes should be administered gently and hygienically. Ideally, drying off should be carried out as a separate job, e.g. split off the cows during morning milking and then bring them back into the parlour after breakfast for tubing. If cows are tubed during milking there is too great a risk that it will be done rapidly or unhygienically, or perhaps, even worse, the wrong cow will be tubed. This has happened on several occasions. Cows have been dried off, the bulk tank fails the antibiotic test the next day, and no one knows which of the 100 or 200 milking cows was treated in error.

The technique for inserting an intramammary tube was described earlier in this chapter. Points specific to the administration of dry cow therapy and internal teat sealant are as follows:

- Dry off cows in batches, making it a specific task carried out after milking. Cows need to be split off during milking and then infused after milking has finished. If done during milking, the herdsman cannot concentrate properly on what he is doing and hygiene may be compromised.
- Dry cow therapy should not be administered at the same time as routine foot-trim-

ming, as the operator's hands are likely to be badly soiled and the teats will probably be splashed with faeces. Administer antibiotic to the whole group first, and then do the foot-trimming as a separate job later.

- Strict hygiene is essential, especially if teat sealant is being administered alone, as this has no antibacterial properties. There have been several reports of sick and dying cows following the unhygienic administration of dry cow preparations.
- Hands should be clean and ideally gloves worn. It is essential to scrub the teat end with surgical spirit or commercial wipes (e.g. Mediwipes) before administration. One cause of failure of dry cow therapy is that bacteria are introduced as it is being administered.
- Swab the two teats furthest from you first and then the two near teats. When tubing, infuse the two near teats first and then the two far teats. In this way contamination of teat ends will be reduced.
- An alternative is to swab the two far teats and administer the tubes and then swab and tube the two near teats.
- Only insert the nozzle a very short way into the teat or, even better, use a tube with a small, short nozzle (see Fig. 12.1). Excess dilation of the teat canal produces cracks in its lipid and keratin layers, thereby compromising its defence mechanisms. In addition, by squeezing the antibiotic through the teat canal, rather than fully inserting the nozzle, bacteria colonizing the canal may also be killed. This may not occur if the nozzle is inserted directly into the teat sinus.
- Infuse the dry cow antibiotic first and work this up the teat and into the udder. When infusing the teat sealant, hold the base of the teat between your finger and thumb so that all the sealant is retained in the teat.
- Ensure that teats are dipped immediately after tubing, thereby removing any bacteria that might be able to colonize the teat end and produce a new infection. A few farmers regularly dip cows throughout the dry period, or at least for the final 3 weeks. This is excellent practice. Others suggest

- dipping teats after cleaning and then administering dry cow therapy through a film of teat dip.
- Record dates of drying off and details of the tubes used. In addition to being a legal requirement, it is important to know when a cow has calved early, as an extended milk-discarding period may be required.
 - Cows should be particularly carefully checked for mastitis in the 5 days after drying off and, if possible, teat-dipped daily.

13 Summer Mastitis

The Bacteria Involved	215
Mode of Transmission	216
Clinical Signs	216
Treatment	217
Control	218
Reduce exposure	218
Fly control	218
Dry cow management	218

Summer mastitis has a very different aetiology and epidemiology from other forms of mastitis and does not fit either the contagious or environmental categories listed on page 36. It is essentially a disease of dry cows and heifers, although very occasionally steers, or even bulls, may be affected. The disease is common in temperate areas of the northern hemisphere, although the incidence varies enormously from one year to the next.

One survey estimated that 35–60% of herds in the UK are likely to experience the condition each year, with approximately 20,000 animals (or 1.5% of the national herd) affected. In some other countries in northern Europe, the incidence is even higher; for example, in Denmark, it is 5.0%. It is therefore a significant problem.

The Bacteria Involved

At least six organisms have been isolated. These are:

- *Arcanobacter (Corynebacterium) pyogenes*: this is the most frequent isolate and is the organism responsible for the severe necrosis and destruction of the quarter.
- *Peptococcus indolicus*: ferments milk and damaged tissue into organic acids and indole and is responsible for the characteristic foul smell.
- *Streptococcus dysgalactiae*: this may be the primary infection, allowing *A. pyogenes* to enter and/or proliferate in the mammary gland. It is commonly found on flies and on damaged teat skin.
- Microaerophilic cocci: sometimes known as Stewart–Schwann cocci.
- *Bacteroides melaninogenicus*.
- *Fusobacterium necrophorum*.

However, by no means are all six organisms isolated from every case of summer mastitis. Table 13.1 shows the percentage of occasions on which each organism is isolated. *A. pyogenes* and *P. indolicus* are the most commonly isolated in the UK, whereas in

Denmark the Stewart–Schwann coccus is more common.

Table 13.1. The percentage of occasions on which different bacteria are isolated in summer mastitis cases, in the UK, Denmark and the Netherlands. (From Hillerton, 1988.)

Bacterium	% isolated		
	UK	Denmark	Netherlands
<i>A. pyogenes</i>	85	72	37
<i>P. indolicus</i>	62	87	33
<i>S. dysgalactiae</i>	24	37	8
Stewart–Schwann coccus	22	83	–
<i>F. necrophorum</i>	1	51	22
<i>B. melaninogenicus</i>	<1	35	8

Mode of Transmission

The major means of transmission of infection in the UK is thought to be by the sheep head fly, *Hydrotoea irritans*, which lives by sucking the blood of cattle. The fly prefers woods, copses and damp ground that is sheltered from the wind. Larvae overwinter in light, sandy soils and emerge as adults in July. They are present primarily during July, August and September, and these are therefore the most common months for summer mastitis. Cases may also occur in June and October, if the weather is exceptionally hot and humid. Eggs are laid into the soil in October, and there is only one generation of adults each year.

The flies live in bushes and trees and only fly out to feed on cattle when wind speeds are low (less than 20 km per hour) and in the absence of rain. Their favoured landing areas are the legs, abdomen and udder. The fore teats are more commonly affected than the rear teats, possibly because the swishing tail removes flies from the hind teats.

Although there is considerable evidence that *H. irritans* is a vector for summer mastitis (and hence fly control is a major part of prevention), there are still doubts about it being the only factor involved. This is because:

- *Hydrotoea* flies are often found in association with cattle, but without causing summer mastitis. Possibly some other factor simultaneous with the presence of the fly is required to damage the teat end: for example, thorns, nettles, thistles or long grass, another type of fly, or even cattle licking themselves excessively.
- Summer mastitis can occur in parts of the world where *H. irritans* is not present.
- Disease can occur in winter (usually associated with teat-end damage), when there are no flies.
- Although many of the bacteria causing summer mastitis can be found in the intestine of the fly and are regurgitated during feeding, experiments that attempt to transmit summer mastitis from infected flies to cows have been unsuccessful. Experimentally, it is possible to induce summer mastitis by infusing *A. pyogenes* and *P. indolicus* through the teat canal.

One theory, therefore, is that the first case of summer mastitis occurs spontaneously, possibly by infection tracking in from infected teat sores, and subsequent cases are caused by flies spreading the infection. Outbreaks of disease do occur and hence there must be some vector, perhaps in association with a reduction in the immune status of the animal.

Clinical Signs

The classic symptoms of summer mastitis are a hot, hard and swollen quarter, usually with a tense and enlarged teat, as in Plate 13.1. The quarter is painful and the secretion is thick and clotted, with a characteristic foul smell. More severely affected cows have a raised temperature, are often lame because of the painful quarter and may develop swollen hocks. Some animals may abort (summer mastitis primarily affects pregnant cattle), and others give birth to a full-term but retarded and weakly calf. Neglected cases may die, especially if dry cows and pregnant heifers do not receive as much attention as they should. Prompt treatment is certainly important.



Plate 13.1. A teat and quarter swollen with summer mastitis. Sometimes the legs also swell.



Plate 3.2. Summer mastitis. When it is neither practical nor possible to strip the teat, the udder may burst and discharge.

In some cows and heifers, the disease is very mild and is not seen during the dry period. It is only after calving, when a blind (i.e. non-functional) quarter is detected, that previous infection becomes apparent. These animals have a thickened teat, with a fibrous core running down the centre, replacing the teat cistern. This can be detected by rolling the teat between your finger and thumb. It is worth comparing the feel with an adjacent non-affected teat to emphasize the difference. Attempts to infuse antibiotics often demonstrate how small the cistern has become – much of the antibiotic will run back out under pressure.

A further syndrome, which has increased in frequency over the past few years, is seen in cows calving down with what appears to be a low-grade mastitis, with just a few clots at each milking. On culture, this proves to be summer mastitis caused by *A. pyogenes*. Presumably only a very small part of the mammary gland is affected and it is only when the gland becomes active, as at calving, that clinical signs appear. However, these cows often do not recover, even with high and prolonged doses of antibiotic.

Treatment

The two main organisms causing summer mastitis (*A. pyogenes* and *S. dysgalactiae*)

are both highly sensitive to penicillin, and hence penicillin and its derivatives are the antibiotics of choice for treatment. Even so, very few quarters ever recover. Intramammary tubes are of very doubtful value, but prompt parenteral (injectable) antibiotic therapy is essential, and can be combined with anti-inflammatory agents such as flunixin if the cow is sick. This will reduce the chances of abortion and death. Antibiotics need to be continued for 4 to 5 days, or until the animal's temperature has returned to normal. If at all possible, the infected teat should be stripped very regularly, especially during the first 2 to 3 days. This may then reduce the chances of an abscess bursting through the side of the udder, as seen in Plate 13.2. Pus discharge through the side of the udder is a normal part of the healing process in many cows, however. If it does occur, simply flush the affected area with an antiseptic solution, keeping the wound as open as possible to allow it to drain. Most cases will eventually heal and the animal is not adversely affected. Once the temperature has returned to normal, further antibiotic therapy is likely to be of limited benefit. Some animals resent manual stripping and an alternative is to drain the udder by making a longitudinal cut through the teat, as in Plate 13.3 (anaesthetic is required and the venous plexus at the base of the teat must be avoided.). Infection and pus then discharge from the teat. The environment is already



Plate 13.3. When manual stripping is not possible, the teat can be drained by making a longitudinal slit. However, the cow will still be discharging infection and needs to be removed from other dry cows.

highly contaminated with *A. pyogenes* (which is a normal environmental organism) and so, provided the cow is removed from the group, to avoid fly-borne transmission, the risk is minimal. Even if the teat is not opened, the affected animal should always be removed from the rest of the group to avoid spreading infection to other cows.

Control

Prevention of summer mastitis is primarily based on fly control, long-acting intramammary antibiotics and internal teat sealants. The most common measures are as follows.

Reduce exposure

Keep susceptible cattle away from known summer mastitis pastures. Open fields on high ground exposed to wind and with a clay soil are ideal, as the sheep head fly dislikes these conditions. Avoiding high-risk areas is probably the best control measure.

Fly control

There are a variety of methods available, but most rely on the flow of sebum over the body surface. However, the udder has no sebaceous glands and so there is no flow of sebum over teat skin.

- Pour-on preparations are applied along the animal's back, but also give poor teat protection. In addition, during wet periods, when flies are most active, their persistence is reduced.
- Fly tags give good protection of the head and back, particularly if two are used, one in each ear. The abdomen and udder are still not well protected, however, and these are the favourite landing places for *H. irritans*.
- Sprays: one needs to be very conscientious to achieve a thorough covering of each animal, including the udder.
- Micropore tape. Sealing the teat ends with tape has been used successfully on the continent, particularly in Denmark, but is not popular in the UK. The tape is not easy to apply and has to be replaced every 3 weeks.
- By far the best approach is to apply fly repellent directly on to the udder and teats every week in high-risk areas. Although this incurs a huge labour cost, only one animal has to be saved to make the effort worthwhile. In very high-risk areas, some farms successfully use weekly applications of a mixture of pour-on fly repellent and Stockholm tar, although such preparations are, of course, unlicensed. Stockholm tar alone, regularly applied to teats, is also effective. Chlorhexidine teat dips combined with a fly repellent are available, although to be effective they must be applied daily.
- Segregate and house affected animals; this removes an important source of infection.

Dry cow management

Dry cow antibiotic therapy undoubtedly helps, as most cases of summer mastitis

occur 4 or more weeks after drying off, in other words, when the antibiotic concentrations are declining. Measures include the following:

- A combination of long-acting intramammary antibiotic and internal teat sealant would be ideal.
- In high-risk areas, some farmers repeat infusions of dry cow antibiotic every 3 to 4 weeks. However, this breaks the teat seal and hence may not be ideal. Careful attention to expected calving dates is also needed to avoid antibiotic contamination of milk after calving.
- Heifers can be tubed, but the tip of the tube must be abutted against the teat orifice and the antibiotic squeezed through the teat canal under pressure, rather than inserting the tube into the teat canal itself. Some farms have used internal teat sealant on heifers, with a New Zealand trial showing a 50% reduction in postcalving

mastitis. Care is needed to avoid teat canal damage.

- House the late pregnant dry cows: *H. irritans* will not enter buildings and therefore there is less irritation and nuisance inside. The cows can go out later at night, i.e. after dark, when the fly is not active.
- Move the calving pattern to earlier in the summer, so that there are fewer dry cows in July, August and September.
- On some small farms the dry cows are run with the milkers so that they can be teat-dipped and a watchful eye kept on the udder. Provided the dry cows are clearly identified (to avoid being milked), this form of control can prove to be very effective.

As approximately 20,000 animals are affected in the UK each year, summer mastitis continues to represent a major cost to the dairy industry. It seems extraordinary that there is no adequate technology to control flies.

14 Disorders of the Udder and Teats

Disorders Caused by Metabolic and Toxic Conditions	221
Blood in milk	221
Anterior udder sore/intertrigo/UMD	221
‘Pea’ in teat	222
Photosensitization	222
Teat sunburn	223
Udder oedema	223
Single-quarter oedema	224
Wet eczema/necrotic dermatitis/udder skin slough	224
Ischaemic necrosis of the teat	224
Single-quarter agalactia	226
Chemical teat damage	226
Diseases with Infectious Causes	226
Bacterial eczema	226
Bovine herpes mammillitis	227
Pseudocowpox	227
Staphylococcal impetigo	228
Summer sores (licking eczema)	228
Teat warts	228
Black spot	229
Disorders Caused by Physical Trauma	230
Injuries from crushing of teats	231
Cut teats	231
Cuts penetrating the canal	232
Total amputation	232
Teat Damage from Machine Milking	232
Hyperkeratosis	232
Teat scoring	233
A parlour audit	234
Teat oedema and teat-end wedging	235
Teat ringing	236
Teat chaps	236
Teat-end haemorrhage and pressure necrosis	237
Machine Milking Factors Associated with Teat Damage	237
Machine factors	237
Milker factors	238

Mastitis is clearly the most common disorder of the udder and teats, but the herdsman will encounter a wide range of other conditions. These may or may not be directly related to mastitis, but any problem involving the mammary gland has the potential of increasing susceptibility to mastitis. In addition, a few of these disorders may be confused with mastitis. Some of the more common conditions are described in the following section.

Disorders Caused by Metabolic and Toxic Conditions

Blood in Milk

This is seen in freshly calved cows and can vary from a few clots of blood in the milk from one quarter (Plate 14.1) to almost pure blood coming from all four quarters (Plate 14.2). Some herds may experience a very high incidence of this problem in freshly calved cows, resulting in large quantities of milk being discarded. Although extensively investigated, often no cause is found. In individual animals, blood in milk may be the result of trauma at calving (the legs bruising the udder during abdominal contractions), excessive udder oedema, cows with unusual gaits, or pendulous udders that are knocked by the legs when walking. In occasional cases, rupture of the anterior



Plate 14.2. Blood in the milk: a severe case, with almost neat blood drawn from the udder.

udder ligaments (see pages 8–9 and Plate 2.3) can produce severe blood in the milk, and the blood may even discharge through the ruptured skin at the front of the udder. For treatment, most people recommend only light milking (sufficient to flush bacteria from the teat canal), thus producing increased pressure within the udder, in an attempt to stop the bleeding.

Anterior udder sore/intertrigo/UMD

This condition, also referred to as ulcerative mammary disease (UMD), is often first noticed because of its purulent smell. A foul, moist, discharging area is seen at the front of the udder (Plate 14.3). The condition occurs

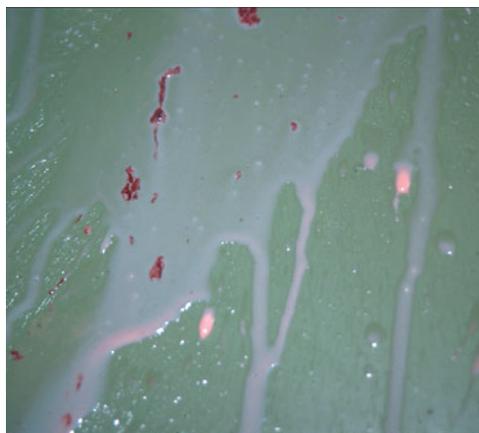


Plate 14.1. Blood in the milk: a mild case.



Plate 14.3. Necrotic dermatitis, also known as intertrigo and ulcerative mammary disease, UMD, is often first noticed as a foul-smelling sore at the front of the udder.

primarily in freshly calved cows, especially older animals, with large deep folds of skin around the front of the udder. The condition is thought to be caused by an ischaemic necrosis (death of tissue due to lack of blood) of the skin, resulting from severe congestion of the udder around the time of calving. Outbreaks have been seen in herds with digital dermatitis, with typical spirochaetes seen on microscopic examination, but digital dermatitis is not the only cause. No treatment is particularly effective, but thoroughly washing the area with antiseptic, removing dead tissue and applying glycerine and an antiseptic or antibiotic ointment will help.

'Pea' in teat

The first sign of a 'pea' in the teat will be when one quarter is found to be full of milk after the cluster has been removed. Hand-stripping will initially produce a few good draws, but flow suddenly stops. At this stage, the 'pea', a thick pad of fibrous material, has become lodged in the teat canal (Plate 14.4). A variety of shapes, sizes and colours of 'pea' are seen (Plate 14.5). They occur most commonly in freshly calved cows, usually up to peak yield, and, from their red colour, must originate from blood clots.

If possible, the 'pea' should be extruded from the teat under pressure. Local anaesthetic, infused into the teat canal, may be



Plate 14.4. 'Pea' protruding from teat.



Plate 14.5. 'Peas' are found in a variety of shapes and colours.

needed. One method of extruding the 'pea' is to pull two 30 ml syringes down over the teat under pressure. Lubricate the teat well and then, with a syringe on each side of the teat barrel, hold the two syringes together at the base of the teat and slowly draw them down to the sphincter. This will build up considerable pressure within the teat, which is often enough to express the 'pea'. If not, then dilate the canal with a semicircular McClean's teat knife and try again. Some vets prefer to use a spiral metal coil inserted through the teat canal. If the 'pea' is attached to the side of the teat wall, then it may have to be removed by using crocodile forceps to cut the tissue away from the inner teat wall.

Photosensitization

Occasionally, photoreactive chemicals accumulate under the skin of individual cows. These are chemicals that react with sunlight and ultraviolet rays. When exposed to ultraviolet light, the chemicals produce thermal energy, which in turn causes intense inflammation, very similar to a burn. Only white or lightly pigmented skin is affected, since black skin prevents absorption of ultraviolet light.

The initial photoreactive agents may have been eaten (e.g. St John's wort in the UK, or lantana poisoning in New Zealand), or may be produced as a result of liver damage. The teat skin is initially thickened and often very painful. It later becomes dry and peels off, leaving a raw surface beneath (Plate 14.6), before eventually healing.



Plate 14.6. Photosensitization: the udder skin thickens, becomes dry and, in the later stages, peels off.



Plate 14.7. Teat sunburn: note that it affects one side of the teat only.

Teat sunburn

Occasionally, cows with non-pigmented teats and large udders develop sunburn along one side of the teat (Plate 14.7). This can be an irritant and, if flies are attracted, may develop into a summer sore. The use of emollients and fly repellents is effective.

Udder oedema

'Oedema' is the name given to an accumulation of fluid in and under the skin. The classic test for oedema is to press the surface of the udder with your finger for 4 to 5 seconds: a pit remaining at the point where you applied pressure (Plate 14.8) is characteristic of oedema. It can also occur on the lower abdomen, running from the front of the udder towards the forelegs (Plate 14.9). Note how in the heifer shown the teat skin is dry and cracking, again due to poor circulation. This is the early stage of necrotic dermatitis.

Excess udder oedema can become a problem, particularly in heifers. In its most severe form it can lead to such extensive necrotic dermatitis that the teat and udder skin is eventually sloughed (i.e. falls off). These animals are impossible to milk and have to be culled. Many develop mastitis. Even in those which can be milked, milking is such a painful process that let-down is poor and yields suffer. Because of the turgidity of the teats, there is an increased



Plate 14.8. Udder oedema characteristically leaves a 'pit' after finger pressure.



Plate 14.9. Oedema under the belly (A). Note also the dry, cracked teat skin (teat necrosis/necrotic dermatitis).

incidence of liner slip and teat-end impacts, which will increase the risk of mastitis. Finally, gross congestion of the udder puts an enormous strain on the suspensory ligaments, which may then rupture (see pages 8–9), seriously reducing the longevity of the heifer.

Possible causes of excess udder oedema at calving include:

- Excessively old and/or overfat heifers.
- Excessive feeding immediately prior to calving.
- Overzealous precalving mineral supplementation, leading to fluid retention. There are anecdotal reports that udder oedema problems have resolved coincident with the removal of ad lib mineral supplementation. Feeding caustic-treated straw or wheat has also been suggested as a causal factor, because it leads to excessive sodium intakes.
- Inadequate exercise. Natural flow of fluid from the udder is via the lymphatics (the body fluid drainage system), moving upwards towards the pelvis. The flow of lymphatic fluid is promoted by limb movement during exercise. Lack of exercise at calving increases fluid stasis, leading to oedema.
- Rupture of udder ligaments can also disrupt flow into the lymphatic ducts, and this may result in a fluid accumulation and oedema.

Single-quarter oedema

Over the past few years a new form of udder oedema has begun to increase in incidence. This is sudden in onset, may affect one or perhaps two quarters only and is most commonly seen in cows in mid-lactation, well after the periparturient oedema (close to calving) has disappeared. Skin sloughing does not occur. The cause is unknown. Affected animals respond only very slowly to diuretics, i.e. drugs that remove excess fluid from the body. Cows may be difficult to milk while the condition is present. This is because the teat may almost disappear into the hard, swollen and

oedematous quarter. At first sight, the herdsmen is highly likely to suspect mastitis, but there are no changes in the milk, there is no increase in body temperature and the cow is not off-colour in any way. The finger-pressure test shows that the swelling is a typical oedema.

Wet eczema/necrotic dermatitis/udder skin slough

This is thought to be a degeneration of skin, often in association with excessive udder oedema, and is most commonly seen in heifers between the legs and udder (Plate 14.10). More advanced cases may develop into a necrotic dermatitis affecting the whole udder (Plate 14.11). The skin is initially swollen and thickened, later becoming dry with a flaking surface. Occasionally heifers are so badly affected that they become impossible to milk. In other cases, damage to the teat end leads to mastitis. In some cows, it is only the udder skin that is affected, and the teats remain soft and pliable, as in Plate 14.12. A heavy growth of *Streptococcus uberis* was isolated from beneath the scab in this cow, so topical antibiotics were applied to the affected area and a successful resolution resulted.

Ischaemic necrosis of the teat

This condition starts as a small, insignificant-looking area of dry skin (A) at the base of the teat (Plate 14.13) and, if identified and treated at this stage, the disorder may not progress. Almost always on the medial aspect, the area of dry skin commonly erodes more deeply into the base of the teat, and may eventually spread over the whole teat barrel. It can become intensely irritant, leading to extensive licking, and self-inflicted injury may totally remove the teat, as in Plate 14.14. It has been proposed that the intense irritation may be caused by a 'pins and needles' effect as the lesion penetrates the erectile venous plexus (Fig. 2.7) at the base of the teat.



Plate 14.10. Wet eczema between the legs and udder, seen mainly in heifers.



Plate 14.11. Advanced wet eczema, which has developed into necrotic dermatitis: some heifers are so badly affected that they are impossible to milk.



Plate 14.12. Udder skin slough. In this cow the teats were only slightly affected.

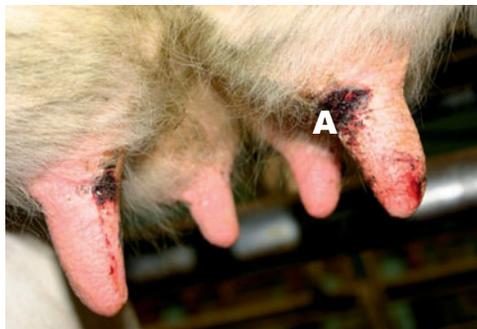


Plate 14.13. The early stage of ischaemic teat necrosis is seen as an area of dry skin (A) at the base of the teat.



Plate 14.14. Severe ischaemic teat necrosis. This heifer eventually removed her teats by excess licking due to the intense irritation.

In the early stages of the condition, use of emollients and anti-inflammatory agents such as flunixin will help, and vasodilators have also been suggested. It may be preferable to discontinue milking. The cause of the condition is unknown, but suggestions include reaction to rubber liners (but why only at one site?), the liner pulling on the teat, e.g. due to poor cluster alignment, or an inherent poor blood flow within the erectile plexus at the teat base.

Single-quarter agalactia

This condition occurs primarily in heifers during the first 4 months of lactation. One quarter starts to become 'light', i.e. milk output is reduced, and this progresses until the quarter is completely non-functional. There are no visible changes in the milk, no increase in cell count and no significant organisms are obtained on culture. Affected animals are non-pyrexemic and continue to eat and to produce milk in the remaining three quarters. If retained, the majority of heifers return to production in the next lactation, although in occasional animals the quarter becomes agalactic (= no milk) for a second time. The cause is unknown.



Plate 14.15. Chemical teat and udder burn. These cows became almost impossible to milk.

Chemical teat damage

The most common mistake is to accidentally use an iodophor/phosphoric acid bulk tank cleaner as a teat dip. Others have used peracetic acid, which is used for cluster flushing when diluted. This has happened on numerous occasions and can lead to severe problems, with chapped teats, sores, skin slough and subsequent mastitis. A typical example is shown in Plate 14.15. Note how both the teat and udder skin are affected. The teat ends are raw, which will predispose to mastitis. These chemicals can also affect the milker's skin. Drums of chemicals must be carefully labelled.



Plate 14.16. Bacterial eczema: this case was caused by contact with an infected sore on the cow's lower lip. Note that eczema appears only on one side of the teats.

Diseases with Infectious Causes

Bacterial eczema

A relatively uncommon form of teat eczema is shown in Plate 14.16. Note how only one side of the teats is affected. This was caused by an open sore on the lower lip of this beef suckler cow (Plate 14.17) and hence the teats were only affected on the side of the sore. A good response was obtained to parenteral (injectable) antibiotics and topical antiseptic teat ointment. The most probable cause of the lesion was *Fusobacterium necrophorum*, although a culture was not carried out



Plate 14.17. Open sore suckler on cow's lip, acting as the source of infection for the teat lesion in Plate 14.16.

to confirm this. The same organism is associated with 'black spot' on the teat end (see page 229), and is indicated in summer mastitis (see page 215).

Bovine herpes mammillitis

This is a much more serious viral infection of the teats than pseudocowpox (see next section), and in some cases can lead to such severe and painful teat skin damage that the animal becomes impossible to milk. In appearance it is very similar to necrotic dermatitis (seen in Plate 14.11), but usually with the teats more affected than the udder. Treatment with emollient dips helps, but teats are slow to heal. In painful cases, predipping with glycerine (which should be wiped off prior to the application of the cluster) will soften the teats and assist milking. During the active phase of the disease, the vesicles (fluid blisters) that appear on the teat skin contain large numbers of virus particles. Affected cows should therefore either be milked last or the milking unit should be thoroughly cleaned and disinfected between cows. Fortunately, once a cow has been infected and recovered, she is left with lifelong immunity. The condition is virtually never seen in dry cows and some people consider that the herpes mammillitis virus may remain dormant on carrier dry cows to become active and cause disease after calving.

Pseudocowpox

This is a viral infection of teat skin and produces characteristic horseshoe-shaped lesions. The teat shown in Plate 14.18 is quite extensively affected. More commonly, a smaller area of teat skin with a smaller and less well-defined lesion is seen, as in Plate 14.19. In the initial stages, there is commonly redness of the skin, which develops into pustules and finally forms scabby areas, which when removed expose the horseshoe-shaped lesions. The condition is not particularly painful and milking can continue. Most animals heal in 3–4 weeks,

resolution being assisted by the use of teat dips containing an emollient. Provided that the weather conditions are mild, hypochlorite dips are thought to be particularly effective. Iodine dips may also be used. Dips are probably better than sprays, as they achieve a more thorough cover. They also reduce the growth of mastitis bacteria, such as *Staphylococcus aureus* and *Streptococcus dysgalactiae*, which could otherwise proliferate in the pseudocowpox scars. Greasy



Plate 14.18. Pseudocowpox: characteristic spreading area of superficial, non-painful haemorrhage.

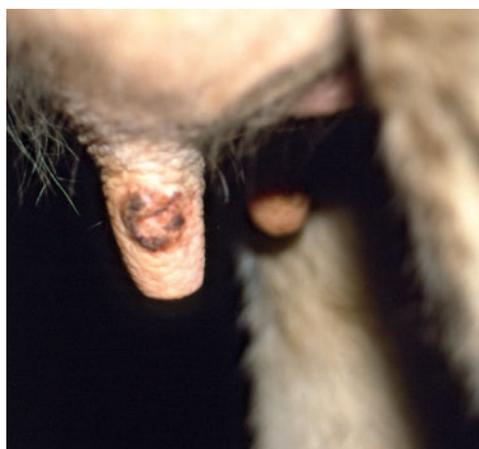


Plate 14.19. Pseudocowpox: single circular lesion – this is the most commonly seen form.

ointments are not recommended, as they attract dirt, may spread bacteria and do not kill viruses.

Immunity to pseudocowpox is short-lived and further infections can occur 6–12 months later. The same virus may also produce small warts, sometimes called milker's nodules, on the herdsman's hands. It has been suggested that pseudocowpox is related to orf, because it is often seen in herds that have contact with sheep. However, the milker's nodules seen in man are very different from human orf lesions.

Staphylococcal impetigo

Not a common condition in dairy cattle but frequently seen in lactating goats, staphylococcal impetigo is a red, raw rash, appearing on the surface of the udder (Plate 14.20). The lesions are not particularly painful, but they produce moist, pimply areas on the udder skin and could represent a reservoir of mastitic bacteria. Washing the skin and treatment with topical antiseptics are usually effective.

Summer sores (licking eczema)

This is thought to be caused by fly irritation. Some cows lick their teats and abdomen excessively, causing surface erosions and sores. A typical example is seen in Plate 14.21. Much worse cases can occur and may lead to summer mastitis. Treatment with fly repellents allows rapid healing. Teat dips may also help.

Teat warts

Warts are caused by papovaviruses. There are five different strains of virus, which possibly explains the big variation in the type of wart seen. The most common are fleshy nodules (Plate 14.22) and feathery warts (Plate 14.23). The latter can be pulled off quite easily, as their roots readily detach. Nodular warts are more difficult to remove.



Plate 14.20. Staphylococcal impetigo. Note the red rash on the udder.

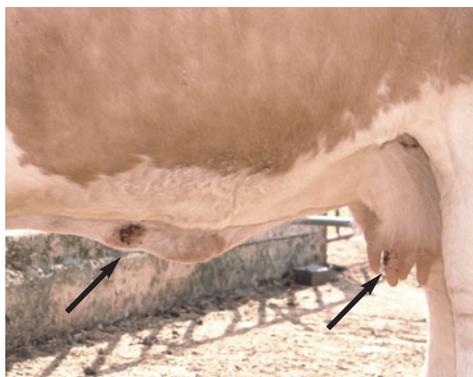


Plate 14.21. Summer sores: fly irritation and subsequent excess licking are the probable cause of these belly and teat sores.

Vaccines have been prepared by grinding up the wart to release the virus, inactivating it with formalin and then injecting the filtrate back into affected animals. A licence may be required to do this. Such vaccines seem to be of only limited value as there is often a poor response to treatment. Most animals eventually undergo self-cure and by the second or third lactation the warts have gone.

When present on the teats, these warts can cause considerable disturbance to milking:

- They may lead to poor liner attachment, air leakage and therefore teat-end impacts.

In some heifers the warts may be so extensive that the animal is impossible to milk.

- Warts may be painful, thereby inhibiting milk let-down, increasing residual milk and decreasing overall yields.
- Warts around the teat canal can predispose to mastitis.
- Skin damage from warts could predispose to secondary infections with *Staphylococcus aureus* and *Streptococcus dysgalactiae*.

The virus causing warts is thought to be transmitted by flies and certainly warts are seen more commonly in heifers reared near rivers and streams (an ideal habitat for flies). Fly control is therefore an important preventive measure. This is discussed on page 218.

However, flies are not the only vector for transmission, since warts may also be seen in housed heifers, especially when stocking densities are high. Similar warts may be seen on the genitalia of young bulls, especially if they are group-housed.

Black spot

This is the term given to a necrosis of the teat sphincter, often with a secondary bacterial infection with organisms such as *F. necrophorum*. A typical example is shown in Plate 14.24. Because of the extensive teat-end damage, the risk of mastitis is enormous. Affected cows are best not milked or just hand-stripped for 1 or 2 weeks and allowed to heal. The use of a chemical debriding ointment improves the rate of healing.

Black spot may initially be the result of machine damage to the teat end, followed by exposure of the teat to an adverse environment, e.g. dirty conditions. Low-emollient teat dips may exacerbate this, although there is some anecdotal evidence that hypochlorite dips are beneficial, in that they promote healing by removing dead tissue from the teat end. With infection already at the teat end, use of a cannula (Plate 14.26) carries a high degree of risk.



Plate 14.22. 'Fleshy' teat warts.



Plate 14.23. 'Feathery' teat warts.



Plate 14.24. Black spot, an infected erosion of the teat end.

Disorders Caused by Physical Trauma

Trauma to teats will be the result of either external crushing or damage caused by machine milking. Factors associated with external crushing are considered first.

Injuries from crushing of teats

Some herds seem to suffer almost epidemics of teat crushing and teat injury. Factors to consider as possible causes when a high incidence of physical injuries is encountered include:

- High stocking densities and inadequate loafing areas: cows are simply too tightly packed together.
- Slippery floors and passageways. Dramatic improvements are often seen following grooving of the concrete, to provide a surface with a better grip. (Heat detection may also improve.)
- Excessively narrow cubicle passages: cows either reverse clumsily into the cubicle opposite or may fall when pushing past one another.
- Very narrow cubicles: large cows may push their legs through into the adjacent cubicle and damage the teats of their neighbours.
- Poor cubicle comfort, for whatever reason, could lead to an increased number of cows lying outside on slippery surfaces and hence increase teat injuries. Cubicle design and dimensions are discussed in Chapter 8.
- Slippery cubicle beds, for example, rubber mats with inadequate bedding: if the bed surface is too smooth, a cow may fall and injure her teats while attempting to stand.
- Loose housing, particularly in long, narrow and poorly designed yards and where cows are heavily stocked; although cubicle systems can produce teat injuries, they are probably more common in poor loose yard systems.
- Rough handling, such as rushing the cows along passageways and around corners, excessive use of the backing gate, excess

use of dogs, etc., so that the cows are liable to fall.

- Continually changing groups: once cows have settled into a group of 50–100 animals, they are best left as such. Moving animals from one group to another leads to aggression and fighting and could produce teat injuries.
- Inadequate fly protection: cows grazing outside, irritated by flies, may chase around fields and through fences, injuring their teats. Dogs could conceivably produce a similar effect. However, most teat injuries occur in housed cattle.
- Increased lameness: cows feeling uncomfortable on their feet are stiff and cumbersome when rising and are likely to have an increased incidence of self-inflicted injuries.
- Poorly maintained buildings: jagged edges, especially on cubicle beds, could increase the incidence of teat damage.

Plate 14.25 is a typical example of a cow whose teat has been crushed, rendering her extremely difficult to milk. This photograph was taken immediately after milking and demonstrates that the affected quarter has not milked out properly. The preferred course of action by many herdsmen is to simply stop milking the teat until it has healed. Within a few days the pressure of milk within the udder declines and, by not applying the milking machine, healing



Plate 14.25. Crushed teat engorged with milk because the cow could not be milked.

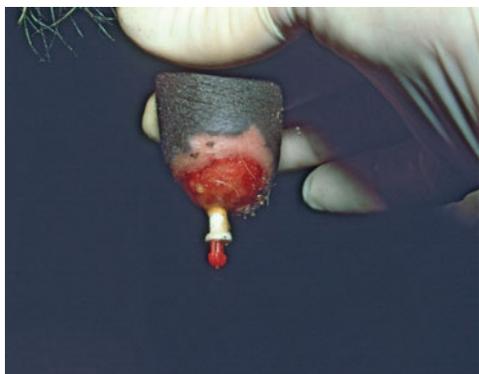


Plate 14.26. A teat cannula avoids the need to milk a cow following teat-end injury, but there is high risk of mastitis, especially when the cannula is removed.



Plate 14.28. Amputation of skin flap promotes healing.



Plate 14.27. Typical cut teat before removal of skin flap.

occurs much more rapidly. Admittedly there is a risk of mastitis, although this is no greater (and is probably less) than if a teat cannula (Plate 14.26) is inserted and left in position for 1 or 2 weeks. If the cow will permit it, the risk can be minimised by a few hand-strippings at each milking.

When milking is resumed, the quarter returns to production surprisingly quickly, even if it has not been milked for 3 or 4 weeks. However, the first few milkings should be discarded, as this milk will have a very high cell count.

Cut teats

Teats are subjected to a wide variety of cuts and lacerations, one of the most common being a horizontal cut on the lower third and towards the teat end, as seen in Plate 14.27. Although this cut has not penetrated through to the canal, the cow will be difficult to milk because the flap of skin will be pulled down each time the unit is pulled off. It is unlikely that the skin flap will be thick enough for successful suturing.

The most effective treatment is to remove the flap under local anaesthetic (as in Plate 14.28) and perhaps leave the teat to heal for a few days before starting to milk it again. Most of these cuts heal extremely well. During the early stages, the wound can be protected from dirt and flies with Micropore tape, a thin bandage that allows the wound to 'breathe', thereby promoting healing.

Cuts penetrating the canal

If the canal has been penetrated, then the teat needs to be sutured. This is best done in a crush, especially where the side of the crush can be removed. The hind leg of the cow should be lifted and fixed, as if for foot trimming, thus giving greater access to the

site and greater security for the operator. Local anaesthetic is infiltrated around the base of the teat as a ring block; the wound is cleaned and then sutured. Some suture the lining of the teat cistern, followed by the skin, but in many wounds a single layer is adequate, especially if the cistern lining can be pulled together using the external suture. Either stop milking the teat for 1 to 2 weeks or, if milking is continued, remove the machine very quickly.

Total amputation

It is surprising how many cows arrive for milking having totally amputated one of their teats at its base, as in Plate 14.29. Many of these cows continue to produce milk in all four quarters, the affected gland simply discharging onto the parlour floor when let-down occurs. Unfortunately the cow in Plate 14.29 developed a severe mastitis and had to be culled.

Teat Damage from Machine Milking

Teats are commonly damaged by the milking machine, and an assessment of teat condition at unit take-off is an extremely important part of assessing overall machine function. The fault is invariably related to



Plate 14.29. Teat accidentally amputated at its base – this is a surprisingly frequent occurrence.

machine milking, but the cause may be:

- in the design or function of the machine (e.g. poor pulsation or excess plant vacuum); or
- the way in which the machine is used by the milker. Examples include clusters reapplied to extract the final few millilitres of milk, or poor udder preparation leading to reduced and biphasic milk let-down and longer unit on times.

Any physical damage to the teat end reduces the effectiveness of the various defence mechanisms described in Chapter 3 and consequently increases the risk of mastitis. The extent of the increase in mastitis will clearly depend on a range of factors, such as the severity of the teat damage, the length of time that the damage has been present, and other factors influencing mastitis, such as the efficiency of postmilking teat disinfection and the cleanliness of the environment.

The following section describes some of the conditions seen, such as hyperkeratosis, oedema, wedging and haemorrhage, and the method of 'teat scoring' by which they are monitored. Teat scoring (see Plates 14.30–14.33) is an invaluable method of assessing the efficiency of machine milking.

Hyperkeratosis

Hyperkeratosis of the teat canal orifice is one of the most common teat lesions associated with machine milking. It is seen as a protrusion of dry, creamy brown or white tissue surrounding the teat sphincter. It has also been known as sphincter eversion, although this term is now rarely used. A typical example is seen in Plate 14.31, and a more severe case in Plate 14.33.

A degree of hyperkeratosis may be a normal feature of high-yielding cows, as it is seen particularly at, or soon after, peak lactation. If the herd scores are grouped into fresh calvers, high yielders and late lactation cows, then often fresh calvers have a low score, 'highs' a medium score and 'lows' the highest score, because they have had the longest exposure to adverse machine function. Even quite severely affected teats



Plate 14.30. Teat score zero – a perfect teat end.



Plate 14.32. Teat score 3 – note the protrusion of the teat canal and the keratin fronds.



Plate 14.31. Moderate hyperkeratosis and 'raised' teat orifice: teat score two.



Plate 14.33. Severe teat-end hyperkeratosis: teat score 4.

recover during the dry period, although damage may be cumulative, i.e. cows affected in one lactation are likely to get worse in the next lactation.

It is thought that hyperkeratosis lesions are most likely to occur at the end of milking when milk flow is minimal. Pointed teats are worse than flat ends (which may develop 'blisters'), and older cows are more affected than heifers because they have less elasticity in their teats.

Teat scoring

Attempts have been made to devise a teat scoring system based on the appearance of the teat orifice (Shearn and Hillerton, 1996). The examination is best done as soon as the milking units are removed. It is essential to use a head lamp to fully view teats, and surgical gloves should be worn. Ideally, the teats also need to be handled: first, to assess the degree of oedema (i.e. hardness) of the teats and, second, tipping the teat end into view allows the operator to fully visualize

changes to the teat end. In some herds, the cows resent teats being handled, and in these circumstances the operator may have to just examine the teats visually and then assess a score per cow – rather than a score per teat, as is described below.

Much of the scoring is based on the severity of hyperkeratosis of the teat canal. On a 0–4 basis, this is approximately as follows:

0. The perfect teat end. Although there may be a slightly thickened ring visible (the teat sphincter), there is no roughening (Plate 14.30).
1. The orifice appears slightly too ‘open’ and has lost its normal smooth, circular appearance. The canal ring has a slightly raised appearance, and there may be early keratin fronds.
2. Moderate hyperkeratosis: a few small, rough fronds of keratin are protruding 1–2 mm from the raised teat orifice (Plate 14.31).
3. Orifice very rough, with keratin protruding all the way round the teat sphincter (Plate 14.32).
4. Advanced protrusion of keratin, 2–4 mm long, and the sphincter has the appearance of having turned almost inside out (Plate 14.33).

Every herd will have a proportion of cows with a degree of teat-end damage. Target herd values are a mean score of between 0.5 and 1.0 per teat. Herds with a mean score of 1.0 and above should be investigated. In the investigation, there may be value in subdividing the scores, for example, the mean score of front versus hind teats, or mean scores of high- versus low-yielding cows or of cows versus heifers.

As shown in Fig. 5.9 only higher scores are likely to lead to an increased incidence of subclinical mastitis in an individual cow. The cell count and clinical mastitis incidence of cows with high scores can be compared with those that have low scores to assess whether teat-end damage is indeed a cause of the clinical problems on a particular farm.

Other systems score teats 0–5, where

score 5 is an advanced stage of score 4. Most scoring systems involve handling individual teats, but where this is not practical, for example the cows resent being handled, then a mean score per cow from a solely visual examination can also be used, as already mentioned.

Teat club international scoring

In this system, teats are scored as:

- Normal. The teat end is smooth (score 0).
- Smooth ring. The teat sphincter is raised and visible (score 1 above).
- Rough rings (scores 2 and 3 above).
- Very rough rings (score 4 above).

Target values are no more than 20% rough and very rough rings and no more than 10% very rough rings. Values above this require investigation.

A parlour audit

In addition to scoring teat-end changes, the observer might also find it useful to record other factors that might have an influence on teat condition and overall mastitis incidence. Some examples follow (further details can be found in the Appendices):

- The number of biphasic let-downs, i.e. where there is initial milk flow, followed by a 30- to 90-second period of slow or zero flow and then a build-up to full flow. This is best recorded by the Lactocorder (on page 106), but simple visual observation of milk flow into the claw bowl is quite useful. More than 5% biphasic let-downs indicate a problem with udder preparation and milk let-down, and this can produce teat-end damage.
- The number of audible liner slips (on page 80). There should be no more than 5% of cows with liner slip, and any liner slip should be attended to by the milker as a matter of urgency.
- Teat skin condition, e.g. dry or cracked skin (Plate 14.34) will predispose to mastitis and lengthen unit on times.

- Teat oedema and wedging (see next section).
- Presence of haemorrhages (Plates 14.35 and 14.36).
- Faecal soiling of teats at unit removal. The authors classify a cow as 'dirty' if one or more teats have an area of soiling greater than the size of a fingernail. Ideally, no cows should be affected, and if more than 10% of cows score 'dirty' then this represents a problem.
- Is ACR (automatic cluster removal) function rough or gentle, i.e. does the cow kick or does she remain quiet at unit removal? If more than 20% of cows kick or defaecate, this indicates a problem.
- Is there milk present in the cluster bowl at unit removal, or is it empty? A totally empty claw at unit removal is a sign of overmilking, which in turn leads to teat-end damage.
- The number of cows where one or more teats are less than 50% covered with post-dip. This figure should be less than 5% (see pages 123–125).

Teat oedema and teat-end wedging

Oedema is one of the earliest changes likely to be detected with adverse machine milking. It is often more easily detected by palpation of the teats rather than visual examination. Teats should be soft and pliable at unit removal. In affected cows they are firm, almost hard to the touch, slightly discoloured, and may be painful. If significant oedema is found in more than 10% of cows, corrective action should be taken.

The small amount of swelling and oedema of the teat end seen immediately after unit removal (Plate 14.37) is an acceptable change, and is particularly common in heifers and freshly calved cows. The swelling and line of flattening of the teat will follow the plane of collapse of the liner, since liners always open and close in the same lateral plane. Hence, if the teat in Plate 14.37 were viewed from the side, it would in fact appear thinner, rather than fatter. In more advanced cases, compression of the teat may be so severe that a wedge forms

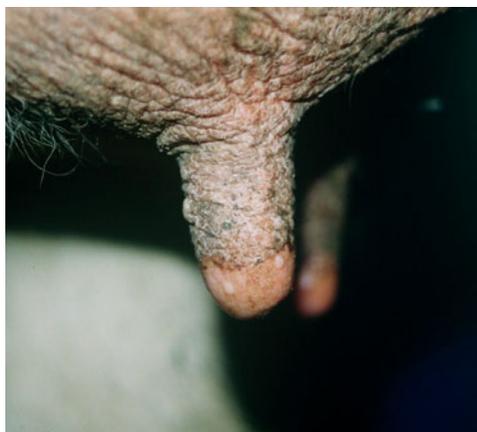


Plate 14.34. Dry and cracked teat skin predisposes to mastitis, especially contagious infections.



Plate 14.35. Mild teat-end haemorrhage.



Plate 14.36. Severe teat-end haemorrhage.



Plate 14.37. Teat-end oedema: note the swelling at the end of the teat, best seen immediately after removal of the milking machine.



Plate 14.38. A wedge across the teat end is caused by the liner, in this case triangular, closing.



Plate 14.39. Note the 'ringing' at the base of the teat following unit removal. This excess pressure from the liner mouthpiece reduces milk flow rates, prolongs milking times and predisposes to teat end-damage.

across the teat end, and in some cases this may crack and produce chaps. Triangular liners will produce a triangular compression of the teat end (Plate 14.38). Some triangular liners have an air vent inserted into the mouth piece of the liner (the air bleed in the claw is then closed off). Vented liners are claimed to provide better milk flow away from the teat and less teat end damage.

Teat ringing

In some cows, 'rings' will be visible at the base of the teat on unit removal, as shown in Plate 14.39.

If seen in a few freshly calved cows, it is of no major significance and is probably associated with a temporary periparturient oedema. However, if present in a greater number of cows, it indicates a problem with the liners or liner shell, or perhaps, plant vacuum. If the teat is constricted at the base, then this will compromise the rate of milk flow from the udder cistern into the teat cistern and, in so doing, it will decrease milk flow rates, increase unit on time and predispose to further teat-end damage.

Teat chaps

'Chaps' are cracks in teat skin. They occur particularly when cows are exposed to wet, cold and windy weather or to damp and dirty environmental conditions. Teat dipping in severe cold, for example, in sub-zero conditions, can produce chaps, especially if damp teats are exposed to a wind chill factor. The development of chaps may be aggravated by poor unit alignment, leading to twisting of the teats (and hence opening of skin cracks) when the cluster is removed. Postmilking disinfection with high-emollient dips, or even neat glycerine, promotes rapid healing. Not only are chaps painful, but they can also harbour mastitic bacteria, particularly *Staphylococcus aureus* and *Streptococcus dysgalactiae*.

Teat-end haemorrhage and pressure necrosis

Small haemorrhages, as seen in Plate 14.35, result from poor machine function and sub-optimal pulsation, leading to inadequate teat massage during milking. This is discussed in Chapter 5. Plate 14.36 shows a more advanced case; note the extensive haemorrhage around the teat end, the protrusion of the sphincter and the haemorrhage at the base of the teat, adjacent to the udder, caused by the liner crawling up the teat and pinching it closed. In advanced cases, there may be a slough of teat-end skin, such as in Plate 14.40.



Plate 14.40. Pressure necrosis of the teat end, resulting in a total slough of the superficial skin.

In liners with shields (see page 73) the effective length may be reduced so much that, in cows with long teats, the liner fails to fully compress the teat end during the rest phase of the pulsation cycle (see pages 68–70). This leads to poor blood circulation at the end of the teat and can sometimes be a cause of teat-end haemorrhage.

Machine Milking Factors Associated with Teat Damage

As mentioned earlier, teat damage will be the result of either the way in which the machine is set up, or the way in which the equipment is used by the milker. This is also discussed in Chapter 5.

Machine factors

A very wide range of potential factors are involved, and in any one farm, there may be more than one cause. As yields have increased, unit on times are longer, and this has increased the risk of teat damage. Some of the more important factors are as follows.

Overmilking

The greatest adverse effect of the milking machine occurs when vacuum is applied to a teat in the absence of milk flow. This is because there is then nothing to dissipate the vacuum, and teat-end vacuum rises to plant vacuum (teat-end vacuum would normally be 3–5 kPa lower than plant vacuum during milk flow). Overmilking can occur at unit attachment if udder stimulation was insufficient, or at the end of milking if the cluster is not removed before milk flow ceases. Whenever the unit is attached to the cow, there should always be milk present in the cluster bowl.

Excess plant vacuum

Excessively high or fluctuating vacuum can lead to teat damage. The correct vacuum level for the plant will depend on whether it is a high-line or low-line plant, and a number of other factors, such as cluster weight.

Poor pulsation

The most common pulsation defect leading to teat damage would be an inadequate massage phase ('d' phase), perhaps arising from an excessively wide pulsation ratio, e.g. 65:35 or less, or a 'd' phase less than 200 ms. An incomplete or slow opening of the liner (namely the 'a' phase of the pulsation cycle) might occur if milk flow starts while the liner is still partially closed, when milk is effectively being 'squeezed' out through the teat end. This may happen with old liners that open more slowly due to partial collapse of the rubber.

Cluster weights

There is some evidence that heavy cluster

weights lead to more teat end damage. It is certainly not advisable to put a brick or other weight on to the clawpiece. However, very lightweight clusters might increase the incidence of liner slip.

ACR settings

The setting of the ACR can certainly affect teat condition, and plants where cows kick and/or defaecate excessively at unit take-off probably need correction. The main settings influencing ACR function are: (i) the milk flow rate that triggers vacuum shut off; (ii) the delay interval between reaching this minimum flow rate and vacuum shut-off; and (iii) the delay between vacuum shut-off and the ACR cord pull.

The two most commonly incorrect settings are that the milk flow rate is too low, and the delay between vacuum shut-off and ACR pull is too short. A low flow rate trigger (e.g. less than 500 ml/min – see page 68) leads to excess unit on time and teat-end damage. If the ACR cord pulls before the vacuum in the claw has had time to vent (i.e. the delay is too short), then the cluster is pulled off with the teats under vacuum, a bit like pulling a cork from a

bottle. This can cause significant discomfort to the cows, leading to kicking, excess muck in the parlour and teat-end damage.

Milker factors

The major milker factors that might influence the degree of teat damage are briefly listed below, and are discussed more fully in Chapter 6.

1. Inadequate udder stimulation before applying the unit, such that there is a period of unit on time with no milk flow. This is often referred to as a biphasic let-down.
2. Aggressive handling of cows, thus inhibiting milk let-down. Examples include, the use of dogs or of electrified backing gates, and the milker chasing the cows into the parlour.
3. Poor teat skin condition, leading to slower milk flow rates and longer unit on times.
4. Over riding the ACR and/or reapplying units, for example, to get the final 250 ml of milk out of a cow or to milk nervous or stressed heifers.

15 Residue Avoidance in Milk

Reasons for Antibiotic Failures	240
'Off Label' Treatment	240
Steps to Avoid Residues	241
Cow identification	241
Identify all treated dairy cows	241
Record all treatments	242
All milk from treated cows must be discarded	242
Milk all treated cows last or separately	242
Withdrawal periods	243
Antibiotic screening tests	243
Use only licensed medicines	243
Store all drugs correctly	243
Medicine labelling	244
Purchased cows	244
Cows that calve early	244
Training	244
Recorder jars	244
Communication	244
Written treatment protocols	245
Antibiotic Screening Tests	245
Natural Inhibitors	246
Study Herd	246

Medicine residues in milk are a major food safety issue. They pose a potential human health hazard as some people are hypersensitive to antibiotics and they can cause antibiotic resistance. Also, they can interfere with product manufacturing processes by inhibiting yogurt and cheese starter cultures.

There has been a great drive to reduce the number of bulk tank failures, but the level of failures in the UK has been increasing slightly over the past years, despite the reduction in the number of dairy farmers.

The majority of failures are due to human error, with most farmers knowing why the failure has occurred. Part of this increase may be due to larger farms and fewer staff, and therefore a greater risk of human error. It may also be compounded by the increase in foreign milkers, where there may be language difficulties and a lack of adequate training.

In the event of an unexpected failure in which a tanker or silo is contaminated, the financial implications for dairy farmers are

very great. They may have to pay for the milk lost and any consequential costs for disposal. Some farmers may be complacent as some dairy companies have an insurance scheme that allows up to two failures each year without penalty, provided the farmer notifies the dairy company in advance. The costs of disposal of contaminated milk under the new EU Animal By-products Regulations will be significant. An unexpected residue failure presents a major problem to any dairy company.

Some dairy farmers consider that the only residues of significance are antibiotics. It must be remembered that other medicines, such as anthelmintics, hormones, steroids, etc., can also be responsible for causing residues in milk. Throughout the EU, random milk samples are collected from farms for extensive testing for these substances.

Reasons for Antibiotic Failures

The suggested reasons for bulk tank antibiotic test failures, taken from a UK survey by Booth in 1982, are shown in Table 15.1. The reasons suggested for test failures add up to more than 100%, as many farmers gave more than one possible reason. Poor or non-existent records of treatment for clinical mastitis and not withholding milk for the whole of the recommended period stand out as the major reasons for test failures. Cows calving early where the full withdrawal period has

Table 15.1. A UK survey of suggested reasons for bulk tank antibiotic test failures. (From Booth, 1982.)

Poor/no records	32%
Not withholding milk for full period	32%
Calving early/short dry period	15%
Accidental transfer of milk	14%
Prolonged excretion	12%
Contamination of recorder jars	9%
Withholding milk from treated quarter only	8%
Lack of advice on withholding period	6%
Mechanical failure	6%
Recently purchased cows	3%
Milking through jars	1%
Use of dry cow preparation during lactation	1%

not been observed, along with accidental transfer of milk from recorder jars, were other significant contributors to failures. At the time of this survey, herd sizes were significantly lower than they are at present and 'off label' medicine use was relatively unknown.

The Northwest Illinois Dairy Association carried out a similar survey in 2000 on the most common causes of bulk tank antibiotic residues, and the results are shown in Table 15.2. Farmers did not know why the failure occurred in 20% of cases. However, the use of 'off label' drug medicines accounted for 17% of failures. Poor cow identification, human error and milking a dry cow by mistake made up the majority of the rest of the failures.

Table 15.2. A US survey of suggested reasons for bulk tank antibiotic test failures. (From Northwest Illinois Dairy Association, 2000.)

Employee error	25%
Unknown/insufficient data	20%
'Off label' drug use	17%
Poor cow identification	16%
Milking a dry cow	13%
Poor communication	4%
Treated cows not separated	3%
Other	2%

Both of these surveys support the fact that the majority of failures are down to human error. But there can be other more unusual reasons for antibiotic failure, which include cows drinking from a medicated footbath or accidentally mixing medicated feed into the lactating cow ration. There are no known problems where residue failures occur if medicines have been used according to data sheet recommendations.

'Off Label' Treatment

The use of medicines 'off label' is increasing significantly. 'Off label' use is defined as any deviation from the manufacturers' data sheet recommendations. 'Off label' treatment includes:

- Increasing the dose rate (usually increased dose level, such as infusing two tubes of intramammary antibiotic at the same time).
- Changes to frequency of treatment, e.g. cows treated three times a day rather than twice (if that is the data sheet recommendation).
- Extending the duration of therapy.
- Unlicensed combination treatments (combination refers to the use of intramammary and injectable antibiotics).
- Changing treatments to another product before the milk withdrawal period of the initial treatment has lapsed.
- Using a product intended for a non-lactating animal in a lactating animal.

A typical example of ‘off label’ use would be an intramammary preparation combined with parenteral injections of penicillin as there is no licensed combination therapy with penicillin injections (see Plate 15.1). Another example would be treating a cow with antibiotics twice daily instead of the data sheet recommendation of daily treatment.

There are some combination mastitis treatments (intramammary and injectable antibiotics) that are licensed as such and have a milk withdrawal period for the combination. The number of licensed combination treatments for dairy animals is very small.



Plate 15.1. ‘Off label’ treatment includes use of unlicensed combination therapy.

In the EU, the minimum milk withdrawal period for any medicine used ‘off label’ is **not less** than 7 days. It may be longer depending on the products and how they are used. The onus is on the veterinary surgeon to caution the farmer to ensure the milk is safe from any treated cows prior to it entering the bulk supply. Ultimately, the final responsibility is with the farmer to ensure that all milk sold off farm is safe and free from residues. Bulk tank milk must pass whatever inhibitory tests are used by the milk purchaser.

Steps to Avoid Residues

The risk of residue violations will be negligible if the following steps are taken. A summary of these key points is shown in the ‘Best Practice Guides’ (Chapter 16).

Cow identification

There are still some farmers who fail to have any recognizable form of cow identification other than an official ear tag number. It is essential that all treated dairy cows are clearly identifiable so that the milker is able to identify individual cows from the milking pit. Herds with poor cow identification practices are more likely to have a higher risk of failures.

Identify all treated dairy cows

It is essential that all treated cows are identified as such by using leg or tail tape or spraying of the udder (see Plate 15.2), or, if possible, entering their details in the milking parlour’s computerized system. There are still some farmers who rely on their memories to identify all cows receiving treatment. Problems may occur if someone else comes in to carry out the milking, or if the farmer simply forgets which cow received what treatment and when.

It is best practice to mark cows for treatment before any medicine is administered to ensure that the farmer knows which cow is



Plate 15.2. All cows should be identified before any treatment is administered.

ANIMAL MEDICINE		USAGE RECORD			DAIRY HERD			
DATE	COW NUMBER	1/4	MEDICINE	SUPPLIER	BATCH NO	EXPIRY DATE	WITHDRAWAL PERIOD	APPROVED BY
14/07/15	285	REA	Bovicillin XTRM SC	vet	1301-15	03/15	72h	SA
17/07/15	389	REA	Bovicillin XTRM SC	vet	1301-15	03/15	72h	SA
27/06/15	417 462	REA	Bovicillin XTRM SC	vet	1301-15	03/15	72h	SA
28/08/15	227	REA	Bovicillin XTRM SC	vet	1301-15	03/15	72h	SA
28/08/15	425 459	REA	Oxytetracycline SC	vet	2001/15	08/15	72h	SA
31/07/15	415	REA	Bovicillin XTRM SC	vet	1301-15	03/15	72h	SA
02/08/15	405 413 453	REA	Bovicillin XTRM SC	vet	1301-15	03/15	72h	SA
04/08/15	356 424	REA	Bovicillin XTRM SC	vet	1301-15	03/15	72h	SA
04/08/15	465 460 461	REA	Enrofloxacin SC	vet	2001/15	04/15	72h	SA
05/08/15	421	REA	Bovicillin XTRM SC	vet	1301-15	03/15	72h	SA

Plate 15.3. Record all treatments in the medicine book.

being treated. Some farmers have treated a cow in the parlour but marked a different animal in error, which has resulted in a bulk tank failure.

Record all treatments

It is a legal obligation to record all treatments given to food-producing animals. These data must include the identity of the animals treated, date of treatment, name of product and amount dispensed, together with the batch number and the milk and meat withdrawal periods. This is not only important for all lactating treatments but also for dry cow therapy, where farmers can check records if cows calve early. In the event of an unexpected bulk tank failure, these records are essential as part of an investigation (Plate 15.3).

All milk from treated cows must be discarded

All milk from the treated cow must be discarded. Some farmers think it necessary to discard only milk from an individual quarter that is treated with an intramammary preparation, and the milk from the untreated quarters still enters the bulk supply. The udder is a very vascular organ, with 500 litres of blood flowing through the udder for every litre of milk produced. Antibiotics are picked up from the treated quarter and are



Plate 15.4. All milk from treated cows must be discarded, not just the milk from a treated quarter. Quarter milkers should not be used.

transferred to the other quarters via the bloodstream and then excreted from all four quarters (see Plate 15.4).

Milk all treated cows last or separately

It is advisable to milk any treated cows last to avoid any accidental transfer of milk to the bulk supply. This is easy in large herds where there can be a treatment group or holding facilities for small groups of animals

(see Plate 15.5). If this is not possible, it is advised that treated cows are milked through a dump bucket, which avoids accidental transfer of milk. Many new parlours are fitted with a dump line, which avoids accidents provided that milkers remember to identify treated cows and milk them into the dump line.

Some farmers still milk treated cows through a recorder jar in the parlour and then discard this milk. There are occasions where this milk may accidentally be transferred or, if the valves at the bottom of the jar leak, milk with residues can contaminate the bulk tank.

Withdrawal periods

It is important that farmers clearly understand and follow the withdrawal periods for all medicines. 'Off label' treatments must be clearly understood, the longer milk withdrawal periods observed and the milk tested before it is returned to the bulk tank.

Antibiotic screening tests

Screening tests are very useful in identifying whether the milk is safe to be returned to the bulk tank or not. The UK industry standard is



Plate 15.5. Ideally milk all treated cows last. Note that these cows are also colour marked to show they are under treatment.

the Delvo SP and many farmers have on-farm test kits. However, many tankers are initially screened with the BetaStar test, a rapid test taking about 10 minutes, before the tank is unloaded at the dairy. In other countries, a wide range of tests are used. In the US, specific tests are commonly used to detect traces of antibiotics used to treat the cow.

There is no need to screen milk before it goes back into the bulk tank where a medicine has been used following data sheet recommendations. Indeed, such screening is inadvisable as false positive or negative results may occur. If a product is used 'off label', milk must be screened 7 days after the end of treatment, and when it passes the test it can then be included in the bulk supply.

Use only licensed medicines

All medicines used in food-producing animals must be licensed with the regulatory authority of that country. These medicines will have a product licence printed clearly on the medicine. Foreign medicines of an identical brand name may have a different formulation and withdrawal period, which could result in a residue failure (see Plate 15.6).

Store all drugs correctly

Medicines must be stored in the correct manner according to the data sheet. Some



Plate 15.6. Use only licensed veterinary medicines.

medicines will have to be refrigerated; others may need to be stored away from sunlight, etc. It is a legal requirement in the UK that all medicines are kept under lock and key. Any products that have passed their expiry date must be discarded appropriately and not used. In the USA, there is a requirement to keep medicines for dairy cows and non-lactating animals separately to avoid accidental use.

Medicine labelling

Medicine labels must include the milk withdrawal period. The dispensing veterinarian, pharmacist or wholesaler must give advice on drug administration for all dispensed medicines. This will include the dose rate, frequency of treatment and route of administration (oral, intramuscular, intramammary, topical, intravenous, intrauterine or subcutaneous). Remember, if there is any deviation from any of this, then the medicine is being used 'off label' and the minimum milk withdrawal period must be applied.

Purchased cows

Farmers buy cows on trust and presume, unless told otherwise, that these animals are free from residues. It is possible that cows may have been treated prior to purchase or may have calved early and still have residues from dry cow therapy. It is advisable to screen all purchased cows before their milk enters the bulk tank (see Plate 15.7).

Cows that calve early

Check the dry-off date against the calving date. If the withdrawal period has expired, then this milk can be included in the bulk tank. Remember that it is a legal requirement not to sell milk from cows calved less than 96 hours in the EU, irrespective of any milk withdrawal period.

If the withdrawal period still has not expired, then this must be observed. If in



Plate 15.7. Be aware that purchased cows may have residues. Test before putting milk in the bulk tank.

doubt, test the milk. Dry cow therapy contains high levels of antibiotic that is contained in a slow-release base and the withdrawal period for some products can be as much as 54 days after drying off. Some of the cloxacillin dry cow preparations in the UK also have a requirement to withhold milk for up to 8½ days postcalving.

Training

All individuals in the medicine chain must be trained to ensure that they play their part fully in minimizing error. This includes veterinary surgeons, agricultural merchants, herdsmen and farm managers. All need to be aware of their part in ensuring that milk produced is free from any residues.

Recorder jars

Ideally, treated cows should not be milked into recorder jars. However, if treated cows are milked through recorder jars, they must be rinsed out as antibiotics concentrate in the fat and traces could pose a risk of a bulk tank failure (Plate 15.8).

Communication

In herds where there is more than one milker, good communication is essential to



Plate 15.8. Milking cows into recorder jars is not recommended. If they are, ensure that the fat residues are rinsed before milking the next cow.

avoid treated cows being milked into bulk tank. A simple board system works well (Plate 15.9).

Written treatment protocols

Written treatment protocols should clearly explain treatments commonly administered by the farmer, along with any milk withdrawal periods. There should be a clear definition of 'off label' treatments. Written treatment protocols form best practice and will help to ensure that all cows are treated correctly and to avoid any risk of residues.

Antibiotic Screening Tests

Withdrawal periods are set by regulatory authorities and are based on the maximum residue limit (MRL). An MRL is the maximum concentration of residue following administration of a veterinary medicine that is legally permitted or acceptable in food under the laws of any regulatory authority such as the EU or, in the USA the FDA (Food and Drugs Administration). At the end of the withdrawal period milk will be below the MRL level and is safe for human consumption.

There are a variety of antibiotic screening tests that are available for use, including the Delvo SP, which is the test most frequently used by the EU dairy industry. The majority of screening tests used in the EU are designed for testing bulk milk rather than individual cows.

The Delvo SP tests to variable levels of antibiotic. These levels do not necessarily match the MRL. For example, the MRL for cloxacillin is 30 p.p.b. but the Delvo SP will detect levels as low as 15 p.p.b., half the MRL or legal limit. On the other hand, the MRL of oxytetracycline is 100 p.p.b. but the Delvo SP test detects levels at 400 p.p.b., four times the MRL.

This means that a farmer who uses a cloxacillin preparation and tests the milk with the Delvo SP test at the end of the milk



Plate 15.9. A board at the front of the parlour shows everyone which cows' milk needs to be kept out of the tank.

withdrawal period can have a positive result even though the correct withdrawal period has been observed. This is one of the limitations of individual cow screening tests.

There is no single test that detects all antibiotics at the MRL. This is the reason why it is not recommended to test cows that have been treated according to data sheet instructions, as many positive results may appear even though this milk is perfectly fit for consumption and gets diluted out in the bulk tank.

Some farmers dilute milk from individual cows to be tested one part in four with milk from the bulk tank. If the milk passes it is then returned to the bulk tank. This helps to overcome the oversensitivity of some of the tests.

There are also differences between screening tests. The BetaStar test is a rapid screening test that is commonly used to screen milk tankers before milk is offloaded into silos at the dairy. The BetaStar test can detect cloxacillin residues as low as 5 p.p.b. (the MRL is 30 p.p.b.) but does not detect tylosin, streptomycin or oxytetracycline. A comparison of the MRLs of the Delvo SP, BetaStar and Charm MRL tests is shown in Table 15.3.

There are other tests that can be used, and some test only for a specific antibiotic. However, it must be remembered that most dairy contracts require producers to ensure that all milk sold off farm passes any residue tests that the milk buyers choose to use and so this is the important criterion for farmers.

Natural Inhibitors

Natural inhibitors can cause problems that result in failure of a residue test in individual cows. For example, mastitic milk from individual untreated cases of coliform mastitis may fail the antibiotic residue test for up to 21 days after infection. This is due to high levels of the enzyme lysozyme, the protein lactoferrin, and complement. High temperatures kill off these natural inhibitors.

In a Japanese study, 24 milk samples failed the Delvo SP test after the milk withdrawal period had expired. These samples were then heated at 82°C for 5 minutes and the samples were retested. After this heat treatment, 21 of the 24 samples passed the Delvo SP test. Heating samples destroys natural inhibitors but has no effect on any antibiotic that may be present. Natural inhibitors are unlikely to be responsible for a bulk tank failing a residue test.

Study Herd

The owner of a herd of 150 dairy cows had been advised by his dairy company to test all cows after calving to ensure that they were free of residues before the milk entered the bulk tank. This was designed to improve food safety and reduce the risk of residues entering the bulk tank. This farm had never had any bulk tank failures in the past 10 years.

The farmer had been using a dry cow preparation containing cephalonium for several years and always observed the correct milk withdrawal period. The dairy company

Table 15.3. Comparison of MRL (maximum residue limit) with sensitivities of different antibiotic test kits.

Antibiotic	MRL	Delvo SP	BetaStar	Charm MRL
Penicillin	4	2	2–4	4
Cloxacillin	30	15	5–10	30
Ceftiofur	100	50	75–150	100
Tylosin	50	50	Not detected	Not detected
Streptomycin	200	300–500	Not detected	Not detected
Oxytetracycline	100	100	Not detected	Not detected
Cephalonium	20	5–10	7.5–15	3–5

tested individual cow samples in its own laboratory, using either the Delvo SP or the Charm MRL.

The farmer found that cows kept failing the Charm MRL test for up to 21 days after the manufacturer's withdrawal period. As a result he was discarding up to 1000 litres of milk per day, as instructed by his dairy company, which said that the milk from these cows was unfit for human consumption.

On one particular day, 21 milk samples from cows where the milk withdrawal period had expired were tested with both the Charm MRL and the Delvo SP test. Sixteen samples tested positive with the Charm MRL test but only three tested positive with the Delvo SP test. On the basis of these results, milk from the 18 cows that passed the Delvo SP test were included in the bulk tank at the next milking. The bulk tank passed all future residue tests.

The three samples that failed the Delvo SP test were sent off for antibiotic assay where individual antibiotics are measured. Two samples had no traces of cephalonium present, suggesting that this failure must have been due to natural inhibitors. The other sample had traces of cephalonium but these were well below the MRL. On further

questioning, the farmer realized that he had sampled this cow at 48 hours after calving and not at 96 hours, and so a trace of antibiotic would not have been surprising, as the milk withdrawal period had not yet expired.

The farmer had lost all faith in individual cow testing. He no longer tested individual cows after calving provided the withdrawal period had been observed, after which time all milk was put into the bulk tank. Any cows treated 'off label' had their milk diluted one part in five with milk from the bulk tank to overcome the sensitivity of these tests. If the milk passed, this was then included in the bulk tank.

What can we learn from this herd example?

1. There is a variation in sensitivity between different antibiotic residue test kits.
2. Some test kits are oversensitive to levels of antibiotics and detect levels below the MRL.
3. There is no benefit in testing cows after calving **provided** the correct milk withdrawal period has been observed.
4. Natural inhibitors can cause failures with residue test kits on individual cow samples.

16 Best Practice Guides

Top Tips to Reduce Cell Counts	248
Top Tips to Minimise Environmental Mastitis	249
Milking Routine Best Practice	249
Best Practice to Avoid Residues	249
Best Practice: How to Administer Dry Cow Antibiotic Therapy and an Internal Teat Seal	250
Best Practice for Circulation Cleaning	250
Sterile Milk Sample Collection for Bacteriology	251

Top Tips to Reduce Cell Counts

These are general recommendations for any dairy herd. Other control measures will apply depending on the herd management and mastitis problems present in the herd.

1. Carry out regular individual cow cell counts. Use the data to identify the persistently high cell count cows. Have these cows tested to identify the mastitis bacteria that are causing the high cell count on your farm.
2. Postdip every cow after every milking throughout the year. Postdipping kills bacteria that have been transferred onto the teats during milking. This happens all year round.
3. Use antibiotic dry cow therapy on all cows at the end of lactation.
4. Change the liners of the milking machine every 2500 milkings or every 6 months, whichever is the shorter period.
5. All milkers to wear clean gloves during

milking to reduce the risk of cross-contamination. Rinse the gloves regularly in disinfectant solution during milking. Do not use common udder cloths.

6. Detect clinical mastitis early and treat all clinical cases with antibiotics.
7. Have the milking machine serviced twice a year and follow the recommendations made in the test report.
8. Disinfect the milking cluster after milking any cow with clinical mastitis or high cell count to avoid the spread of infection.
9. Cull persistently high cell *Staphylococcus aureus* cows in lactation four and above. Other high cell count cows may need to be culled, so discuss this with your vet or adviser.
10. Ensure that you buy low cell count animals and not cows infected with *Staphylococcus aureus* or *Streptococcus agalactiae*. Check the herd and individual cell count history before purchasing animals.

Top Tips to Minimise Environmental Mastitis

These are general recommendations for any dairy herd. Other control measures will apply depending on the herd management and mastitis problems present in the herd.

1. Teats and udders should be clean. Keep cows on clean, dry beds. If udders and teats are dirty, the beds are not clean enough.
2. Predip cows to disinfect teats prior to milking. Teats must be dried. If the milk sock is dirty after milking, teat preparation must be reviewed.
3. Encourage cows to remain standing for 30 minutes after every milking so that the teat canal closes. The best way is to offer fresh feed. Lame and sick cows must be allowed to lie down.
4. Always use clean, dry bedding; this absorbs maximum moisture and does not get mouldy or damp. Scrape passageways twice daily.
5. Pay particular attention to the calving pens. These must be kept as clean as possible, as freshly calved cows are most prone to toxic mastitis.
6. Have a minimum of one cubicle per cow (ideally 5% more cubicles than cows). If on straw yards, allow at least 6.5 sq. m of lying space, bed up daily with clean, dry straw and clean out every 2 to 4 weeks.
7. Make sure you have stable vacuum levels throughout milking. You should not have liner slip and the regulator must always be letting in air.
8. Check teat-end conditions. If the teats are damaged, then there is an increased risk of mastitis as the teat end is the barrier that keeps infection out of the udder.
9. Use an internal teat seal with dry cow therapy, ensuring excellent hygiene prior to infusion. Pinch the teat halfway up and infuse the internal teat seal so that it remains at the bottom of the teat.
10. Monitor progress. Check on your mastitis incidence and timings of infection.

Use bacteriology tests to identify the cause of clinical mastitis on your farm.

Milking Routine Best Practice

Goal: milk clean, dry teats with minimal risk to udder health.

1. Wear gloves and keep them clean throughout milking.
2. Detect mastitis promptly and accurately by foremilking.
3. Prepare your cows in batches of five to eight.
4. Predip and strip the batch. Then go back and wipe and attach each cow. Units to be attached within 1 to 2 minutes of preparation.
5. Ensure the unit is aligned so it sits squarely on the udder.
6. Remove the unit to avoid overmilking. ACRs should be set to come off at a flow rate of 400–500 ml/min for twice-daily milking and 600–800 ml/min for 3 times a day milking.
7. After milking, thoroughly coat the entire surface of each teat with a postdip.
8. Mastitis management:
 - Milk mastitis cows last or through a separate cluster.
 - Disinfect the cluster after milking every mastitic cow.
9. Allow cow to exit from the parlour and encourage standing for 30 minutes by offering fresh feed during the housed period.
10. Remember that the milking parlour is a food factory and must be kept clean throughout milking.

Best Practice to Avoid Residues

1. Use only licensed veterinary medicines.
2. Store all drugs correctly. Ideally medicines for lactating and non-lactating animals are kept separately.
3. Ensure medicines are clearly labelled with the milk withdrawal period.
4. Ensure all treated cows are clearly identified.

5. Record all treatments in the medicine book.
6. Treat cows only after carrying out step 4.
7. Have a separate treatment group where treated animals are milked last or, if this is not possible, milk separately into dump buckets.
8. Observe the correct withdrawal periods.
9. Discard **all** milk from treated cows where there is a withdrawal period, not just from a treated quarter.
10. Keep milking and dry cows separately if at all possible, or ensure that dry cows are clearly identified.
11. Use antibiotic screening tests to test any cows treated 'off label' or which calve early.
12. If in doubt, test milk from purchased cows to ensure they are clear of residues before putting the milk in the bulk tank.
13. Check treatment dates and withdrawal periods of cows that calve early and ensure that milk is discarded until the withdrawal period has expired.
14. Ensure that all milkers are trained in residue avoidance measures.
15. Have written treatment protocols and medicine withdrawal period data.
16. If in doubt, keep the milk out. Test to ensure the milk is safe.

Best Practice:

How to Administer Dry Cow Antibiotic Therapy and an Internal Teat Seal

No internal teat sealant contains antibiotics and so scrupulous hygiene during administration is essential.

Preparation is everything and a small amount of extra time will pay dividends. It is recommended that cows to be dried off are kept separately and brought into the parlour at the end of milking. This will allow plenty of time to administer treatments without the pressure of milking.

Best practice is as follows:

1. Identify the cows to be treated with a red spray or other marking method. This

is especially important if these treatments are carried out as and when these cows come into the parlour to be milked.

2. Strip out all quarters.
3. Wear clean gloves during the drying off procedure.
4. Teat-dip the furthest two teats.
5. Wipe off this solution with clean dry paper towel.
6. Scrub the teat end with cotton wool and surgical spirit until no more dirt appears on the cotton wool.
7. Infuse dry cow therapy and massage up the teat.
8. Infuse the internal teat sealant, pinching at the teat and udder junction to ensure it remains at the bottom of the teat. **Do not** massage up into the teat.
9. Repeat the above procedures for the two nearest teats.
10. Teat-dip all four teats.
11. Record the treatments in the medicine book.

Best Practice for Circulation Cleaning

Goal: clean parlour to maximize milk quality and extend parlour life. Remember:

- Too much dairy chemical is corrosive and expensive.
- Too little is ineffective and the plant will not be properly cleaned.
- Work out the wash volumes used and the correct concentration of dairy chemicals and write these clearly on the dairy wall or a noticeboard.

1. Wash the plant as soon as possible after milking so that milk does not solidify in the plant.
2. Clean off the clusters and jettors and attach. Remove the line into the bulk tank. Turn off the plate cooler water supply. Set up the plant for the cleaning cycle.
3. Remove the milk sock and replace with a clean sock.
4. Rinse warm water (body temperature) through the plant to waste. This

removes 95% of milk soil and keeps the plant warm.

5. If you are using a hot detergent solution, circulate the hot wash solution around the plant. Hot water should be entering the system at about 85°C. If using a cold solution, circulate it around the plant.
6. As the wash solution returns to the wash trough, add in the correct amount of dairy chemicals for the volume of water used.
7. Hot washes should circulate at between 60–70°C. All solutions should only circulate for between 5 and 8 minutes.
8. The parlour has now been cleaned. Discard the used wash solution.
9. Circulate cold water and hypochlorite to disinfect the plant.
10. Drain from the plant either after wash-up or before the next milking.
11. Hang the units to drain.

Sterile Milk Sample Collection for Bacteriology

It is essential that sterile milk samples are collected to identify the cause of clinical mastitis or high cell counts. If this procedure

is not followed, then the outcome will result in contaminated samples and will be of no benefit.

You must use sterile sample pots.

1. If the teat is dirty, wash and dry. If it is visibly clean, then dry-wipe with paper towel.
2. Discard three squirts of foremilk from each quarter to be sampled.
3. Coat the teat with a pre- or postdip, allow a contact time of 30 seconds and wipe dry with paper towel.
4. Put on a clean pair of gloves.
5. Scrub the end of the teats with cotton wool soaked in surgical spirit so that the end of the teat is spotless.
6. Take the top off the sample bottle, hold it at a 45° angle and squirt one stream of milk into the bottle, making sure that you do not touch the end of the teat.
7. Replace the top on the bottle.
8. Label with cow number, quarter/s, farm and date.
9. If there is any doubt about the sterility of the sample, repeat the entire procedure.
10. Freeze the sample or send directly to the lab keeping it cool if possible.

This page intentionally left blank

Appendix: Liner Life Charts

Table A.1. Liner life in days according to herd and parlour size and assuming a liner life of 2500 milkings: three times a day milking.

Herd size	Number of milking units											
	6	8	10	12	14	16	18	20	24	28	30	32
50	100	133	167	167								
60	83	111	139	167								
70	71	95	119	143	167							
80	62	83	104	125	146	167	182					
90	56	74	93	112	130	148	167	182				
100	50	67	84	100	117	133	150	167				
110	45	61	76	90	106	122	136	152				
120	42	56	70	84	98	112	125	139				
130	38	51	64	76	89	102	114	128				
140	36	48	60 ^a	72	84	96	108	120				
150	33	44	55	66	77	88	99	110	133			
160	31	42	53	62	74	84	93	105	125			
170	29	39	49	58	68	78	87	98	116	137		
180	28	37	46	56	65	74	84	93	112	130	139	
190	26	35	44	52	61	70	78	88	104	123	131	
200	25	33	41	50	58	66	75	83	100	116	125	
210	24	32	40	48	56	64	72	80	96	112	119	127
220				45	53	61	68	76	90	106	114	122
240				42	49	55	62	69	84	98	165	110
260					45	51	58	64	77	90	145	102
280					42	48	54	60	71	84	89	96
300					39	44	50	56	67	78	83	88
320					36	42	47	52	62	72	78	84
340					34	39	44	49	59	68	73	78
360					32	37	42	46	55	64	69	74
380					30	35	39	43	52	61	65	70
400					29	33	37	41	50	58	62	66
450					26	30	33	37	44	52	55	59
500					23	26	30	33	40	46	50	53

^aFor example, a herd of 140 cows milking twice daily with 10 milking units needs to have its liners changed every 60 days, or 2 months.

Table A.2. Liner life in days according to herd and parlour size and assuming a liner life of 2500 milkings: two times a day milking.

Herd size	Number of milking units											
	6	8	10	12	14	16	18	20	24	28	30	32
50	150	183	183	183								
60	125	167	183	183								
70	107	143	179	183								
80	94	125	156	183	183							
90	83	111	139	166	183	183						
100	75	100	125	150	175	183						
110	68	91	114	136	159	182	183					
120	82	83	104	164	145	166	183	183				
130	58	77	96	116	135	154	174	183				
140	54	71	89	108	124	142	162	178				
150	50	67	84	100	117	134	150	168	183			
160	47	62	78	94	109	124	141	155	183			
170	44	59	74	88	103	118	132	148	176	183		
180	42	56	70	84	98	112	126	140	168	183	183	
190	39	53	66	78	93	106	117	133	156	183	183	
200	38	50	63	76	88	100	114	125	152	175	183	
210		48	60	71	84	96	107	120	142	168	180	183
220				68	79	91	102	114	136	158	170	182
240				62	73	83	94	104	124	146	165	166
260					67	77	86	96	115	134	145	154
280					62	71	80	89	107	124	134	142
300					58	67	75	83	100	116	125	134
320					55	62	70	78	94	110	117	124
340					51	59	66	73	88	102	110	118
360					49	55	62	69	83	98	104	110
380					46	53	59	66	79	92	99	106
400					44	50	56	62	75	88	94	100
450					39	44	50	55	66	78	83	88
500					35	40	45	50	60	70	75	80

Appendix: Parlour Audit

References and Further Reading

References

- Berry, E. and Booth, J. (1999) Summer mastitis in England and Wales: 1992 to 1997. *Veterinary Record* 145, 469.
- Berry, E.A. and Hillerton, J.E. (2002) The effect of selective dry cow treatment on new intramammary infection. *Journal of Dairy Science* 85, 112–121.
- Blowey, R. and K. Collis (1992) Effect of premilking teat disinfection on mastitis incidence, total bacterial count, cell count and milk yield in three dairy herds. *Veterinary Record* 130, 175–178.
- Blowey, R. and Deyes, E. (2005) The effectiveness of drying off individual quarters as a treatment of mastitis. *Cattle Practice* 13, 99–102.
- Booth, J.M. (1982) Antibiotic residues in milk. *In Practice* 4, 100–109.
- Booth, J.M. (1993) Proceedings of the British Cattle Veterinary Association. In: *Cattle Practice* 1, 125–131.
- Bradley, A.J. and Green, M.J. (1998) A prospective investigation of intramammary infections due to *Enterobacteriaceae* during the dry period: a presentation of preliminary findings. *Cattle Practice* 6, 95–101.
- Bradley, A.J., Leach, K.A., Breen, J.E., Green, L.E. and Green, M.J. (2007) Survey of the incidence and aetiology of mastitis on dairy farms in England and Wales. *Veterinary Record* 160, 253–258.
- Bramley, A.J. (1981a) The role of hygiene in preventing intramammary infection. In: *Mastitis Control and Herd Management*, Technical Bulletin No. 4, National Institute for Research in Dairying, Reading, UK, pp. 53–66.
- Bramley, A.J. (1981b) Infection of the Udder with *Streptococcus uberis*, *E. coli* and minor pathogens. In: *Mastitis control and Herd Management*. Technical Bulletin No. 4, National Institute for Research in Dairying, Reading, UK, pp. 70–80.
- Bramley, A.J., Dodd, F.H. and Griffin, T.K. (1981) *Mastitis Control and Herd Management*. Technical Bulletin No. 4, National Institute for Research in Dairying, Reading, UK.
- Bramley, A.J. (1992) Mastitis. In: Andrews, A.H., Blowey, R.W., Boyd, H. and Eddy, R.G. (eds) *Bovine Medicine: Diseases and Husbandry of Cattle* Blackwell Scientific Publications, Oxford, pp. 289–300.
- Bramley, A.J., Godinho, K.S. and Grindal, R.J. (1981) Evidence of penetration of the bovine teat duct by *Escherichia coli* in the interval between milkings. *Journal of Dairy Research* 48, 379–386.
- Dingwell, R.T., Leslie, K.E., Schukken, Y.H., Sargeant, J.M., Timms, L.L., Duffield, T.F., Keefe, G.P., Kelton, D.F., Lissemore, K.D. and Conklin, J. (2004) Association of cow and quarter-level factors at drying-off with new intramammary infections during the dry period. *Preventive Veterinary Medicine* 63, 75–89.
- Egan, J. (1995) Evaluation of a homeopathic treatment for subclinical mastitis. MRCVS Thesis, The Veterinary Research Laboratory, Abbotstown, Dublin, Ireland.
- Galton, D.M., Pettersson, L.G., Merrill, W.G., Bandler, D.K. and Shuster, D.E. (1984) Effects of premilking udder preparation on bacterial population, sediment, and iodine residue in milk. *Journal of Dairy Science* 67, 2580–2589.
- Galton, D.M., Peterson, L.G. and Merrill, W.G. (1988) Evaluation of udder preparations on intramammary infections. *Journal of Dairy Science* 71, 1417–1421.

- Green, M.J., Green, L.E. and Cripps, P.J. (1996) Low bulk milk somatic cell counts and endotoxin-associated (toxic) mastitis. *Veterinary Records* 138, 305–306.
- Green, M.J., Huxley, J. and Bradley, A. (2002) A rational approach to dry cow therapy 1. Udder health priorities during the dry period. *In Practice* 24, 582–587.
- Grindal, R.J., Walton, A.W. and Hillerton, J.E. (1991) Influence of milk flow rate and streak canal length on new intramammary infection in dairy cows. *Journal of Dairy Research* 58, 383–388.
- Harrison, R.D., Reynolds, I.P. and Little, W. (1983) A quantitative analysis of mammary glands of dairy heifers reared at different rates of live weight gain. *Journal of Dairy Research* 50, 405–412.
- Hill, A.W. (1981) Factors influencing the outcome of *Escherichia coli* mastitis in the dairy cow. *Research in Veterinary Science*, 31, 107–112.
- Hill, A.W. (1990) *Proceedings of the British Mastitis Conference*, p. 49.
- Hill, A.W. (1992a) *Proceedings of the British Cattle Veterinary Association 1991–1992*, p. 279.
- Hill, A.W. (1992b) *Proceedings of the Mastitis Conference*, Frampton, UK, p. 281.
- Hillerton, J.E. (1988) Summer mastitis – the current position. *In Practice* 10, 131–137.
- Lee, C.S., Wooding, F.B.P. and Kemp, P. (1980) Identification, properties, and differential counts of cell populations using electron microscopy of dry cows secretions, colostrum and milk from normal cows. *Journal of Dairy Research* 47, 39–50.
- Leigh, J.A. (2000) *Streptococcus uberis*: current and future prospects for a vaccine. *Cattle Practice* 8, pp. 265–268.
- Lewis, S., Cockraft, P.D., Bramley, R.A. and Jackson, P.G. (2000) The likelihood of clinical mastitis in quarters with different types of teat lesions in the dairy cow. *Cattle Practice* 8, 293–299.
- Logan, E.F., Meneely, D.J. and Mackie, D.P. (1982) Enzyme-linked immunosorbent assay for *Streptococcus agalactiae* antibodies in bovine milk. *Veterinary Record* 110, 247–249.
- Mackellar, Q.A. (1991) Intramammary treatment of mastitis in cows. *In Practice* 13, 244–249.
- Mackie, D.P., Pollock, D.A., Meneely, D.J. and Logan, E.F. (1983) Clinical features of consecutive intramammary infections with *Streptococcus agalactiae* in vaccinated and non-vaccinated heifers. *Veterinary Record* 112, 472–476.
- Meany, W.J. (1992) In: *Mastitis and Milk Quality*. Handbook for Veterinary Practitioners, Irish Vet Association and Irish Veterinary Union, Teagasc Research Centre, Moorepark, Ireland.
- Menzies, F.D., Gordon, A.W. and McBride, S.H. (2003) An epidemiological study of bovine toxic mastitis. *Proceedings of the British Mastitis Conference*, Garstang, pp. 1–13.
- Milner, P., Page, K.L. and Hillerton, J.E. (1997) The effects of early antibiotic treatment following diagnosis of mastitis detected by a change in the electric conductivity of milk. *Journal of Dairy Science* 80, 859–863.
- Neave, F.K., Dodd, F.H., Kingwill, R.G. and Westgarth, D.R. (1969) Control of mastitis in the dairy herd by hygiene and management. *Journal of Dairy Science* 52, 696–707.
- Northwest Illinois Dairy Association (2000). [Details unknown.]
- Owens, W.E., Watts, J.L., Boddie, R.L. and Nickerson, S.C. (1988) Antibiotic treatment of mastitis: comparison of intramammary and intramammary plus intramuscular therapies. *Journal of Dairy Science* 71, 3143–3147.
- Pankey, J.W., Wildman, E.E., Drechsler, and Hogan, J.S. (1987) Field trial evaluation of pre-milking test disinfection. *Journal of Dairy Science* 70, 867–872.
- Peeler, E.J., Green, M.J., Fitzpatrick, J.L. and Green, L.E. (2002) Study of clinical mastitis in British dairy herds with bulk milk somatic cell counts less than 150,000 cells/ml. *Veterinary Record* 151, 170–176.
- Philpot, W.N. (1984) Economics of mastitis control. *Veterinary Clinics of North America: Large Animal Practice* 6, 233–245.
- Philpot, W.N. and Nickerson, S.C. (1991) *Mastitis: Counter Attack*. Babson Bros. (Westfalia-Surge), Naperville, Illinois.
- Rendos, J.J., Eberhart, R.J. and Kesler, E.M. (1975) Microbial populations of teat ends of dairy cows, and bedding materials. *Journal of Dairy Science* 58, 1492–1500.
- Schukken, Y.H., Vanvliet, J., Vandegheer, D. and Grommers, F.J. (1993) A randomized blind trial on dry cow antibiotic infusion in a low somatic cell count herd. *Journal of Dairy Science* 76, 2925–2930.
- Shearn, M.F.H. and Hillerton, J.E. (1996) Hyperkeratosis of the teat duct orifice in the dairy cow. *Journal of Dairy Research* 63, 525–532.
- Sol, J., Sampimon, O.C. and Snoep, J. (1995) Proceedings of the 3rd International Mastitis Seminar, Israel, pp. 5–68.
- Spencer, S.B. (1990) The basics of vacuum in milking systems and milking management. *Proceedings of the National Milking Center Design Conference*, Harrisburg, Pennsylvania.

- Tyler, J.W. and Baggot, J.D. (1992) Antimicrobial therapy of mastitis. In: Andrews, A.H., Blowey, R.W., Boyd, H. and Eddy, R.G. (eds) *Bovine Medicine: Diseases and Husbandry*. Blackwell Scientific Publications, Oxford, pp. 836–842.
- Williamson, J.H., Woolford, M.W. and Day, A.M. (1995) The prophylactic effect of a dry-cow antibiotic against *Streptococcus uberis*. *New Zealand Veterinary Journal* 43, 228–234.
- Woolford, M.W., Williamson, J.H., Day, A.M. and Copeman, P.J.A. (1998) The prophylactic effect of a teat sealer on bovine mastitis during the dry period and the following lactation. *New Zealand Veterinary Journal* 46, 12–19.

Further Reading

- Andrews, A.H., Blowey, R.W., Boyd, H. and Eddy, R.G. (2004) *Bovine Medicine: Diseases and Husbandry of Cattle*, 2nd edn. Wiley-Blackwell, Chichester, UK.
- Biggs, A. (2009) *Mastitis in Cattle*. The Crowood Press, Marlborough, UK.
- Blowey, R.W. (1999) *A Veterinary Handbook for Dairy Farmers*, 3rd edn. Old Pond Publishing, Ipswich, UK.
- Bramley, A.J., Dodd, F.H. and Griffin, T.K. (1981) *Mastitis Control and Herd Management*. Technical Bulletin No. 4, National Institute for Research in Dairying, Reading, UK.
- Bramley, A.J., Bramley, J.A., Dodd, F.H. and Mein, G.A. (1992) *Machine Milking and Lactation*. Insight Books, Newbury, Berkshire, UK.
- Countdown Downunder see <http://www.countdown.org.au> (accessed December 2009).
- International Dairy Federation. <http://www.fil-idf.org>

Index

- Acid boiling wash (ABW) 90
Acidified sodium chlorite 121
ACRs *see* Automatic cluster removers
Actinomyces pyogenes *see* Arcanobacter
 pyogenes
Acute mastitis 34
ADF (automated dipping and flushing) 125
Aesculin 47
Aggressive therapy 203–204
Air bleed hole 66, 83
Air injectors 84–85, 88
Alveoli (in mammary gland) 5, 50
Aminoglycosides 200–201
Amputation (of teats) 232
Antibiotics
 acidity and liquid solubility 202
 aggressive therapy 203–204
 bactericidal and bacteriostatic 201–202
 coliforms responses 201
 intracellular effects 202
 residues 3, 111, 245
 sensitivity 57–58
 therapy
 benefits 199
 combination 203–204
 Streptococci and *Staphylococcus*
 aureus cure rates 198
 versus self-cure rates 198–199
 udder penetration 199–201
 withdrawal period 202
Antibiotic screening tests, residue avoidance
 BetaStar and Charm MRL 246
 Delvo SP 245–246
 MRL level 245
Arcanobacterium pyogenes 55
Ash (as cubicle bedding) 133–134
Aspergillus mastitis 55
Automatic cluster removers (ACRs) 63, 68, 111,
 238
Automatic milking system (AMS) *see* Robotic
 milking
Automatic teat disinfection 124–125
Back-flushing units 109–110
Bacillus 36, 41, 44, 54–55, 57, 58, 148, 173, 177
Bacillus species, mastitis
 B. cereus 54
 B. licheniformis 54–55
Bacterial eczema 226–227
Bacterial sources, milk
 dirty milking equipment 174
 environmental contamination
 coliform count 173
 TBC versus coliform count 173
 winter bedding 174
 mastitis organisms
 Streptococcus agalactiae and *Streptococcus*
 uberis 172–173
 Streptococcus agalactiae infection 173
Bacteriology of milk 42, 167, 182, 251
Bacteriostatic 201–202
Bacteriocidal 201–202
Bacteroides melaninogenicus 215, 216
Bactoscan and TBC
 BTA *see* Bulk tank analysis
 comparison 172
 refrigeration failure
 milk quality 174–175
 pasteurized milk, shelf life 176
 plate and tube coolers 175
Balance tank 63–64
Barrier dips 122–123
Bedding *see* Cubicles
BetaStar antibiotic test 243, 246
Beta-lactamase producing bacteria 200–201,
 204, 205
Biphasic let down 81, 84, 234, 238
Black spot 25, 121, 212, 227, 229
Blitz therapy 206
Blood in milk 221
BMSCC *see* Bulk milk; Somatic cell count
Bovine herpes mammillitis 227
Bovine somatotrophin (BST) 17
Brisket boards *see* Cubicles
Bulk tank analysis (BTA)
 applications 178–179
 methodology 59
 sample interpretation 179

- Bulk tank analysis (BTA) (*continued*)
 study herds 180–183
 tests
 bacteria individual significance 176–177
 Bactoscan problems 177
 coliform count 176
 Pseudomonas count 176
 psychotrophics bacteria 177
 sample collection 177–178
 target levels 177
 thermoduric count (TD) 176
- Bulk milk 160–161, 176
 Bulk tank, cleaning 86
 Butterfat 85
- Cai-Pan peppermint oil 209
 Calcium 3, 15, 17, 85, 154, 208
 California mastitis test (CMT)
 benefits 155
 cow cell count 163–164
 disadvantages 155
 method 155–156
- Candida* 55
 Casein 2, 3, 15, 16, 17, 32, 47, 48, 154
 see also Milk protein
- Cell counts
 action for high cell count cows
 early dry cow therapy 163–164
 lactation, treatment 164–165
 milking order 165
 milk withholding 165
 quarter drying off 164
 treatment efficacy 166
 effect on milk
 consumption 154
 production 153–154
 factors affecting 157–159
 financial penalties 153
 individual cow cell count 166–170
 legal compliance 153
 low cell counts 153
 measurement 154–155
 milk yield reduction 153–154
- Cefquinome 200
 Cephalosporins 201, 211
 Chaps (on teats) 236
 Charm test 246–247
 Chemical teat damage 226
 Chemotaxins 27, 29
 Chlorhexidine 120
 Chlorine (in plant washing) 86–87
 Chronic mastitis 34
 Circulation cleaning 86–89
Citrobacter 36, 46
 Claw piece 66, 68, 79–81
 Cloxacillin 199, 200, 211, 246
 Clusters *see* Milking machine
 Clusters – flushing between cows 44, 109, 110
 Cluster removal *see* ACR
 CMT *see* California mastitis test
 Coagulase-negative staphylococci (CNS) 42
 Coagulase test 42–44
- Coliform counts (CC) 58, 59, 104, 127, 173–174, 182
 Coliforms including *E. coli* 44–46
 Colony forming units, CFU 171, 172
 Colostrum 15
 Complement 26
 Contagious organisms, mastitis
 CNS 42
 Mycoplasma species 44
 overall incidence, clinical 36–37
 Staphylococcus aureus 38–41, 59
 Streptococcus agalactiae 42–43
 Streptococcus dysgalactiae 43–44
- Contaminated samples 251
Corynebacterium bovis 55, 125, 177, 180, 181
Corynebacterium pyogenes see *Arcanobacter pyogenes*
Corynebacterium ulcerans 56
- Costs of mastitis 191
 Cow mattresses 134
 Crushed teats 230
 Cubicle/free-stall systems
 bases
 concrete pyramid 142–143
 floor slope 143–144
 bedding types 145–146
 brisket boards 141–142
 discomfort 139
 division height 140
 features 138
 length 140
 management
 cleaning and renewing 144, 145
 semi-automated system 144
 neck rails 141
 rejection 141–142
 single and double row facing 139
 size 138–139
- Cut teats 231
- DCC (DeLaval cell counter) 155
 Defences, teat
 canal
 flow rate and mastitis incidence relationship 24
 Furstenberg rosette 22
 milk lakes 22
 short teat versus open teat 23
 closure
 importance, sphincter closure 22–23
 required pressure, force fluid back up 23
 keratin
 flush 22
 plug 22
 mastitis susceptibility and milking frequency 24–25
 skin
 average teat condition and average milkout 21
 intact surface 21
 teat-end damage
 black spot, milking machine damage and excessive dilatation 25

- hyperkeratosis and physical trauma 25
- DelvoSP test 245–247
- Diapedesis 29
- Dipping *see* Teat disinfection
- Disinfection, teat
- assessment 118–119
 - dipping
 - anti-spill teat dip cups 117
 - chemicals 120–123
 - dips versus spraying 117, 119
 - pots 118
 - preparation and storage 119
 - iodine residues 128
 - postmilking
 - automatic system 124–125
 - limitations 125
 - mastitis bacteria removal 123–124
 - postdip products 127
 - skin quality, dip additives 124
 - sore, bacteria removal 124
 - premilking (predipping)
 - advantage 127
 - dry wiping 126
 - ineffectiveness, predip 128
- Dodecyl benzene sulfonic acid (DDBSA) 121
- Dry cow therapy
- blanket versus selective 211
 - failure 211
 - infusion technique 213–214
 - intramammary pathogen types 210
 - long-acting antibiotics
 - benefits 210
 - Staphylococcus aureus* 210–211
 - quarter treat
 - drying off 211
 - Staphylococcus aureus* infection response 211
 - teat sealant
 - tube administration 212–213
- Drying off quarters 164, 204
- Dry matter intake (DMI)
- metabolic disorder 54
 - rumination rate 53
- Dry period infections
- description 50
 - host immune response 53
 - phases
 - IMIs incidence 50–51
 - keratin plug formation 52
 - versus lactation infections 50, 51
 - short dry periods 54
- Dry wipe 101–102, 128
- Drying off quarters 164, 168, 204
- Dump line 195, 196
- Dump bucket 91, 110, 111, 195
- Dynamic testing (of milking plant) 76–78
- Eczema 224–226, 228
- Edwards media 42, 43, 47, 57
- Effective reserve 82
- Electric current *see* Stray voltage
- Emollients 124
- Endotoxins 30, 45
- Enterobacter* 36
- Environmental organisms, mastitis
- E. coli* (*Escherichia coli*)
 - chronic recurrent coliforms 46
 - culture in milk 22–23
 - dry period infections 45
 - strain variation 45–46
 - total bacterial count 58–59
 - toxic effects 45
 - Klebsiella pneumoniae* 46–47
 - NLFs (non-lactose fermenters) 47
 - Pseudomonas aeruginosa* 47
 - Streptococcus uberis* 47–50, 59
- Environment
- bacterial contamination factors 131
 - bedding types
 - ash 133–134
 - bacteria growth 131–132
 - coliform level 132
 - cubicle sanitizer 135
 - mats and mattresses 134–135
 - sand 133
 - sawdust and shaving 133
 - shredded paper 134
 - space allowances 135–136
 - straw 132–133
 - cow handling 149
 - cubicle/free-stall systems
 - draughts 149–150
 - dry cow hygiene 151
 - heat stress 150
 - open sand yard 131
 - postcalving group 150–151
 - rubber parlour floor surface 149
 - sand yards 147–148
 - stocking densities 148
 - straw yard
 - bedding 145–146
 - design 146–147
 - stocking density 145
 - ventilation
 - conventional roofing cowls 137
 - heat and humidity 136–137
 - multiple-span buildings 138
 - roofing sheets 137
 - straw yards 137
 - wooden cubicle house 138
 - waste food 148
- Erythromycin 16, 200, 201, 202
- Fast milkers 22, 24, 52
- Fat in milk *see* Butterfat
- Fatty acids 17
- Five-point plan (NIRD) 1
- Flaming udders 98, 99
- Flooding (of claw piece) 66
- Fluid therapy
 - intravenous administration 207
 - oral administration 207–208
- Fly control 218
- Foot-and-mouth disease 34
- Foremilking
 - advantages and disadvantages 97
 - internal teat sealants 97

- Foremilking (*continued*)
 risk, infection transfer 97–98
- Forestripping *see* Foremilking
- Fossomatic counter 154–155
- Framycetin 53, 200, 210
- Free fatty acids 17
- Free stalls *see* Cubicles
- Frequency of milking
 effect on mastitis 114
 effect on yield 18, 114
- Fresh calved cow – immune response 30
- Frozen samples 177, 178
- Fungi* 36, 55
- Fusobacter necrophorum* 25, 215, 226
- Furstenburg's rosette *see* Rosette of Furstenburg
- Galactose 16
- Gangrenous mastitis 40, 41
- Gland cistern 9, 13, 14, 18, 106, 212
- Gloves 40, 50, 56, 85, 96, 97, 103, 123, 196, 213, 233, 248, 249, 250, 251
- Glucose 15–16, 208
- Glycerine (added to teat dips) 124
- Gram negative 26, 44, 57, 199–201
- Gram positive 57
- Gram stain 57
- Hard milkers 24
- Heat stress 137, 147, 150
- Heifers – poor milk let down 14–15
- Hock sores (from cubicles) 134
- Humectants 124
- Hyperkeratosis
 causes 78
 high-yielding cows 232–233
 occurrence 233
 teat scoring 234
 immunoglobulins 26
- Immunoglobulins (antibodies) 26
- Immunosuppression (in fresh calved cow) 150
- Impact forces 73, 76, 79–81, 84, 102, 107, 111, 113
- Impetigo (staphylococcal) 228
- Incidence of mastitis 1–4, 36
- Individual cow somatic cell counts (ICSCCs) 162
- Inducible mechanisms, udder defences
 chemotaxin alarm
 interleukin 8 and TNF 27
 phagocytosis process 27
 inflammatory response
 alarm signals 28
 diapedesis and epithelial cells damage 29
 endothelial cell junction 27, 29
 increased blood flow and margination 27
 PMN response, mid-lactation 29
 serum ooze and phagocytosis 29
- Indurated quarter 53
- Infection – reservoirs of
 host response to 35–36
 penetration of teat canal 34–35
- Inhibitory protein 18
- Interceptor vessel 63
- Internal teat seal 97, 204, 212, 249, 250
- Intertrigo *see* Ulcerative mammary disease (UMD)
- Intramammary infections (IMIs) 50–51
- Intrinsic mechanisms, udder defence
 cellular response
 cell types, milk and colostrum 26
 PMNs 27
 SCC 26
 complement 26
 immunoglobulins 26
 lactoferrin
 bacteriostatic effects 26
E. coli infection, lactating and dry cows 25
 lactoperoxidase 26
- Involution 138–140
- Iodophor dips 127
- Iodine
 in milk 128
 in teat disinfectants 118
- Ischaemic teat necrosis 224–225
- Keratin 12
- Keratin flush 22
- Klebsiella pneumoniae* 46–47
- Laboratory pasteurized count (LPC) 59, 174
- Lactation, control of 167–170
- Lactoferrin 25–26
- Lactose 15–16
- Lactoperoxidase 26
- Lanolin 124
- Lateral suspensory ligaments 7–9
- Leptospira hardjo* 55
- Licking eczema 228
- Ligaments (of udder)
 udder suspension 9
 rupture of 8–9
- Lime (as cubicle bedding) 135
- Liner life charts
 three times a day milking 253
 two times a day milking 254
- Liners 71–73
- Liner slip 76, 79–81, 108, 127, 223–224
- Lipase (in mastitic milk) 2, 17
- Lipopolysaccharide 30, 45
- Listeria monocytogenes* 56
- LPC *see* Laboratory pasteurized count
- Lysozymes 246
- Machine stripping 112, 113
- Macrophages 27, 31, 208
- 'Magic water' 102
- Macrophages, function 27
- Margination 27
- Mastitis
 categories 34
 clinical incidence, UK 1, 3, 37
 detection 98–100
 detectors 98–100
 economics 191

- effects, milk components 2, 3
- epidemiology 37–38
- financial penalties 3
- infection, development of 34–36
- lactation stage 192
- and milking frequency rate 24–25
- overall incidence, clinical 36–37
- Maximum residue limit (MRL) 245, 246
- Milk
 - blood in 7
 - components effect of mastitis 2–3
 - composition 15
 - culturing
 - antibiotic sensitivity testing 57–58
 - bulk tank analysis 58–59
 - laboratory plating and incubation 56–57
 - methodology 59
 - mastitic milk appearance 1, 98–99
 - pH 16
- Milk let-down
 - in heifers 14–15
 - phases
 - erectile tissue engorgement 14
 - myoepithelial contraction 13
 - teat canal relaxation 14
 - reflexes and speed of milking 105–107
- Milking routine
 - best practice 249
 - clusters disinfection 111–112
 - drying teats 102–104
 - effect on mastitis 95
 - frequency 114
 - hygiene regimes 95–96
 - infection transfer, sources
 - gloves 97
 - hands 96–97
 - liners 96
 - mastitic infected cows 109–111
 - mastitis detection 98–100
 - order 113–114
 - teat preparation 101–103
 - assessment 104–105
 - unit attachment 108
 - unit removal 111
- Milk synthesis
 - BST 17
 - causes, poor quality 17
 - colostrum 15
 - dry period length 18–19
 - environmental temperature 18
 - fat 17
 - lactose 15–16
 - milking frequency 18
 - minerals 17
 - protein
 - casein 16
 - plasmin 16
- Milk yield reduction and cell count 153–154
- Milker's nodules 228
- Milking cow tube usage 188
- Milking frequency
 - effect on cell count 159
 - effect on mastitis 24
- Milking machines
 - ACRs (automatic cluster removers) 68
 - air admission during milking 63
 - air bleed hole 83
 - balance tank 63–64
 - cluster 66
 - common faults 91–94
 - direct to line 68, 84
 - dynamic test 76–78
 - effects on mastitis 78–81
 - function 61–62
 - high line/low line 67–68
 - interceptor vessel 63
 - maintenance 74–76
 - pipelines 65–66
 - pulsation 68–71
 - pulsation rate and ratio 70
 - pulsation ~ single and dual 70
 - receiver vessel 67–68
 - recorder jar 68
 - regulator 64–65
 - sanitary trap 65
 - simple checks without testing equipment 82–83
 - static test 75–76
 - test report 76, 78
 - vacuum gauge 65
 - vacuum levels and reserve, plant 75
 - vacuum pump 62–63
 - vacuum reserve 82
 - wash up routines 84–86
- Milestone removal 85
- Moulds causing mastitis *see* Yeasts
- Mycoplasma* 44, 78, 109–110
- Necrotic dermatitis/interigo* 221, 223–225, 227
- Neutrophil *see* Polymorphonuclear leucocytes
- Nitric acid 90
- Nocardia asteroides* 55
- Non-lactose-fermenting (NLF) coliforms 47
- Oedema, teat-end 235–236
- Oedema, udder 223–224
- Off label treatment 240–241
- Open sore suckler 226
- Overmilking 81
- Oxytocin
 - continual stripping 208–209
 - level 105–106
- Parlour audit report 255
- Parravaccinia, *see* Pseudocowpox
- Partial insertion technique 197
- Pasteurella/Mannheimia* 55, 57
- Pasteurised milk, effect of temperature on shelf life 176
- Pea in teat 222
- Penicillins, sensitivity and udder penetration 199–200
- Peppermint oil as topical treatment 209
- Peptococcus indolicus* in summer mastitis 215
- Phagocytosis 27, 29
- Photosensitization 222
- Phosphoric acid 90

- Plasmin content of mastitic milk 2–3, 16, 17
 Plate cooler 175
 Polymorphonuclear leucocytes (PMNs)(neutrophils) in milk 26
 Post calving group, establishment of 150–151
 Post milking teat disinfection
 application 116–117
 automatic sprayers 117–119
 chemicals
 barrier dips 122–123
 dodecyl benzene sulfonic acid (DDBSA) 121
 foam dips 121
 hypochlorite and acidified sodium chlorite 121
 iodophors and chlorhexidine 120
 quaternary ammonium compounds (QACs) 120
 viscosity and surfactant 122
 dipping 117
 dipping and spraying equipment 117–119
 effect on teat skin condition 127
 iodine residues 128–129
 limitations 125
 post and pre dipping comparison 127, 128
 rate of chemical use 125
 removal of bacteria from teat sores 124
 removal of mastitis bacteria 123–124
 seasonal use of post dips 126
 versus predipping 128
 Predipping
 application 117
 chemicals used
 foam dips 121
 hypochlorite and acidified sodium chlorite 121
 iodine products 120
 free iodine 120
 reasons for 126, 127
 speed of kill 101, 117
 versus postdipping 128
 Prolactin in control of milk synthesis 17
 Protein
 inhibitor, influence on yield 18
 removal during machine washing 85
 synthesis in milk 16
Proteus, culturing in milk 58
Prototheca mastitis 55
 Pseudocowpox 227–228
Pseudomonas aeruginosa 47
 cause of environmental infection 47
 causing mastitis 177
 dry cow therapy 47, 102
 Pseudomonad count 59
 Psychotrophs 177
 Pulsation
 chamber 68–70
 checks during milking 69
 cycle 70
 massage phase 68, 69
 milkout phase 68, 70
 pulsator device 70–71
 rate and ratio 70
 single and dual 70
 Quaternary ammonium compounds (QACs) 120
 Receiver vessel 67–68
 Record keeping for mastitis 185
 Recorder jar 68
 Recurrence rate 187, 190–192
 Refrigeration failure, milk 174–176
 Regulator of the milking machine
 clean air system 64
 effect of dirt 64
 function 75–76
 maintenance 64
 multiple weight controlled 93, 94
 servo 64
 spring 64
 testing 82–83
 Residual milk 112–113
 Residue avoidance, milk
 antibiotic screening tests
 BetaStar and Charm MRL 246
 Delvo SP 245–246
 MRL level 245
 bulk tank antibiotic test failures reasons 239, 240
 natural inhibitors 246
 ‘off label’ treatment
 data sheet recommendations 240–241
 unlicensed combination therapy 241
 written treatment protocols 245
 Rinse cycle in circulation cleaning 86
 Robotic milking (VMS) 74, 114–115
 Rosette of Furstenburg 22
 Rubber mats for bedding 132
 Rupture of the suspensory apparatus 8–9

Salmonella 56
 Sample collection
 bulk tank 155
 high cell count cows 162
 sterile 251
 Sand yards 147–148
 Sanitary trap of the milking machine 65
 Sawdust bedding 133
 Self cure 198–199
 Selenium and vitamin E effects 31–32
Serratia 55
 Shavings, bedding 133
 Shredded paper bedding 134
 Single quarter agalactiae 226
 Size of cubicles 138–140
 Slow milkers, speeding up 108, 109
 Somatic cell counts, *see* Cell count
 Sphincter eversion of teat-end 25
 Staphylococcal impetigo 228
Staphylococcus aureus 38–42
 acute gangrenous mastitis 40–42
 adhesive properties 35
 cause of contagious infection 37
 cell count variation 39
 culturing for in milk 56
 dry cow therapy 163
 mechanism of attack 35
 resistance to treatment 205
 response to antibiotics 198

- spontaneous cure 198–199
- subclinical, treatment during lactation 164–165
- teat contamination 40
- transfer from cow to cow 37
- treatment 38–39
- Staphylococcus epidermis* 42
- Staphylococcus hyicus* 42
- Staphylococcus intermedius* 42
- Staphylococcus xylosus* 42
- Staphylococci, coagulase negative 42
- Static test of the milking machine 75–76
- Stewart-Schwann cocci in summer mastitis 215–216
- Stocking density in housing systems 145
- Straw yards
 - advantages/disadvantages 144–145
 - bedding
 - anaerobic fermentation 145–146
 - design 146–147
 - hard-core base 146
- Streptococcus uberis* infections 47–49
- Stray voltage 81–82
- Streak canal 57
- Streptococcus agalactiae* 42–43
 - adhesive properties 35
 - blitz therapy 43
 - cause of contagious infection 37
 - cell count 43
 - culture 43
 - mechanism of attack 35
 - response to treatment 206
 - spread of infection 97
 - total bacteria count 176
 - undermilking 43, 81
- Streptococcus bovis* 47
- Streptococcus dysgalactiae* 43–44
 - cause of contagious infection 43
 - in summer mastitis 215
 - response to treatment 198
- Streptococcus faecalis* 55, 58, 180, 181
- Streptococcus uberis* 47, 49, 50, 58, 59, 102, 173, 176, 200, 203, 210, 224
 - association with straw bedding 176
 - cell count 97
 - clinical mastitis 36–37
 - culture 58
 - dry period infections 35, 49
 - effect of lactoperoxidase 26
 - outbreaks at pasture 49–50
 - prevalence 50
 - response to treatment 47
 - strain variation 45–46
 - total bacterial count 176
 - vaccination 50
 - variation in strains 45–46
- Streptococcus zooepidemicus* 55
- Stress
 - effect on cell count 158
 - effect on cows 149
- Subclinical infection
 - definition 43
 - herd cell counts 167
 - total bacterial count 59
- Summer mastitis
 - bacteria 215–216
 - control 218–219
 - Hydrotoea/sheep head fly 216
 - treatment 217–218
- Summer sores *see* Licking eczema
- Sunburn 223
- Supernumerary teats 9–11
- Supportive therapy 206–207
- Suspensory ligaments 8–9
- Targets for mastitis
 - Bactoscan 186
 - cell count 188
 - mastitis rate 185
 - milking cow tubes per cow per year 188
 - percent of herd affected 185–187
 - recurrence rate 187
 - seasonal variations 188
 - stage of lactation 188
- TBC *see* Total bacterial count
- Teat canal 14, 22–24, 34–35, 78–79
- Teat club international scoring 234
- Teat congestion and oedema 92–94, 235–236
- Teat defences against mastitis *see* Defences, teat
- Teat diseases 226–229
- Teats, supernumerary/accessory 11
- Teat length and shape during milking 11
- Teat wall and structure 13–14
- Teat damage 232–237
- Teat disinfection *see* Predipping; Post milking teat disinfection
- Teat preparation
 - automatic washing in hot climates 103
 - dry wiping 101–102
 - drying teats before milking 103–104
 - ‘magic water’ 102
 - pre-dipping 101–103
 - robotic milking 114
- Tetracyclines 201
- Total bacterial count (TBC) 3, 58–59
 - Bulk tank analysis 178
 - dirty milking equipment 174
 - effect of foremilk 97
 - environmental contamination 173–174
 - failure of refrigeration 174–176
 - methodology 59
- Toxins 27, 29, 207 *see also* Endotoxins
- Treatment, mastitis
 - antibiotic
 - acidity and lipid solubility 202
 - bactericidal and bacteriostatic 201–202
 - coliform response 201
 - intracellular effects 202
 - sensitivity and udder penetration 199–201
 - tube withdrawal period 202
 - antibiotic therapy
 - combination 203–204
 - cure rates 198
 - self-cure rates versus antibiotics 198–199
 - Staphylococcus aureus* 197–198

- Treatment, mastitis (*continued*)
- blitz therapy and *Streptococcus agalactiae* 206
 - intramammary antibiotics 196–197
 - mastitic cow separation 195–196
 - Staphylococcus aureus* resistance 205
 - supportive therapy
 - anti-inflammatory drugs 208
 - calcium and glucose 208
 - continual stripping and oxytocin 208–209
 - fluids 207–208
 - homoeopathy 209
 - non-antibiotic intramammary infusions 209
 - topical preparations 209
- Treatment, poor response reasons 39
- Tube usage 188
- Tylosin 200, 202, 246
- Udder
- blood supply 7
 - bruising 42
 - developmental phases 6–7
 - rupture 8–10
 - structure 5, 6
 - suspension 7–8
- Udder and teats disorders
- anterior udder sore/intertrigo/UMD *see* Ulcerative mammary disease (UMD)
 - chemical damage 226
 - machine milking 232
 - oedema 223–224
 - wet eczema/necrotic dermatitis/udder skin slough 224
- Udder defences *see* Inducible mechanisms;
- Intrinsic mechanisms
 - individual cow variation 31
 - inducible mechanisms
 - chemotaxin alarm 27
 - inflammatory response 27–29
 - intrinsic mechanisms 26–27
 - PMN activity reduction 32
 - poor response 30–31
- Ulcerative mammary disease (UMD) 221–222
- Undermilking 81
- Uneven quarters 9
- Vaccine 46, 228
- Vacuum fluctuations 76, 77
- Vacuum gauge 65
- Vacuum level 75, 82
- Vacuum pump 62–63
- Vacuum recovery time 82
- Vacuum reserve 62, 75, 82
- Ventilation
 - conventional roofing cowls 137
 - heat and humidity 136–137
 - multiple-span buildings 138
 - roofing sheets 137
 - straw yards 137
 - wooden cubicle house 138
- Vitamin E 31–32
- VMS (voluntary milking systems) *see* Robotic milking
- Wet eczema 224
- Warts 228–229
- Wash-up routine
- ABW 90
 - air injectors 84–85
 - air lines 86
 - best practice 250–251
 - bulk tanks 86
 - circulation cleaning 86–88
 - efficiency evaluation 91
- Wedging 235–236
- Yeasts 55, 177
 - cause of environmental mastitis 55
 - culture 55
 - treatment 55
- Yersinia pseudotuberculosis* 56

Mastitis Control in Dairy Herds 2nd Edition

Roger Blowey and Peter Edmondson

Mastitis in dairy herds is a worldwide problem, with significant implications for milk yields and quality, economic losses, and animal health and welfare. Effective control makes a considerable difference to the farmer and the animal and this new edition includes updated information throughout, as well as new chapters covering organic dairy herds, dry period infections, robotic milking, residue avoidance and best practice procedures. The authors, both fellows of the Royal College of Veterinary Surgeons and recognised experts in the field, provide basic principles relevant to farmers, vets and veterinary students in a clear and practical way, covering anatomy, epidemiology, milking machines, disinfection, somatic cell counts, and diseases of the udders and teats, in order to provide a thorough understanding of the causes of mastitis and the measures of control and prevention. It is an indispensable resource for large animal vets, dairy industry personnel, farm managers, dairy herdsmen, and researchers and students in animal sciences and related disciplines.

Related titles

Breeding for Disease Resistance in Farm Animals, 3rd Edition

Edited by S.C. Bishop, R.F.E. Axford, F.W. Nicholas and J.B. Owen
2010 c. 512 pages ISBN 978 1 84593 555 9

Principles of Cattle Production, 2nd Edition

Modular Texts Series
C.J.C. Phillips
2009 336 pages ISBN 978 1 84593 397 5

Farm Business Management: the Human Factor

N.L. Nuthall
2009 240 pages ISBN 978 1 84593 598 6

The Economics of Animal Health and Production

J. Rushton
2008 400 pages ISBN 978 1 84593 194 0

For further information on these titles and other publications, see our website at www.cabi.org

CABI Head Office

Nosworthy Way, Wallingford, Oxfordshire, OX10 8DE, UK

CABI North American Office

875 Massachusetts Avenue, 7th Floor, Cambridge, MA 02139, USA

Space for bar code with
ISBN included