Milking machines & Mastitis

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Milking machines can influence mastitis, cell count and/or bactoscan. They are used more than other pieces of equipment on the dairy farm and yet their function, maintenance and effect on mastitis is poorly understood by the majority of farmers. As a result of this, they are often neglected despite the fact that they are responsible for generating the majority of dairy farmer’s income.

The milking machine can have an effect on the incidence of mastitis in six ways.

1. Fomite

Mastitis organisms may be physically transmitted from cow to cow or from one quarter to others in the same cow. Cow to cow spread can occur through contaminated milk remaining on the liner between milkings (Plate 1) and quarter to quarter spread may occur through backflow of milk up the liner.

*Staphylococcus aureus* infection can be spread to the next six to eight cows milked through a contaminated liner. This does not necessarily result in an intramammary infection, as these organisms may not invade the teat canal and should be killed through post milking teat disinfection. The risk of this occurring is increased if the liners are worn as bacteria adhere more easily to their roughened surface.

Liners are the only piece of the milking machine that comes into direct contact with the cow. They are made from complex rubber or silicone material and have a limited useful life. The majority of rubber liners are expected to last for 2,500 milkings or six months, whichever comes first. Dairy detergents, especially chlorine and iodine, will denature rubber after six months of washing. Rubber liners lose their elasticity after 2,500 milkings due to the physical action of opening and closing.

Silicone liners have a much longer life of 1,500 hours of use equivalent to about 10,000 milkings but are more expensive. Silicone is far more supple and gentle and softer than rubber. These liners are pre-collapsed when new and their life expectancy can be measured using special testing equipment.

2. Damage to the teat
The teat canal is the primary defense mechanism in preventing new infections. Any damage to the teat end will increase the risk of new infections. These may occur due to the impaired ability of the teat canal to prevent bacteria entering the udder.

Damage to the teat skin, especially cuts and chaps, provides an ideal environment for the growth of mastitis organisms such as *Staphylococcus aureus* and *Streptococcus dysgalactiae*. The efficacy of post milking teat disinfection may be reduced on damaged teats, as the solution is unable to penetrate damaged skin. For these reasons, quarters with teat lesions are often associated with an increased incidence of clinical and subclinical infection.

One of the commonest and most significant forms of damage is hyperkeratosis of the teat end. This can be characterised as a smooth or roughened ring of dry creamy white tissue surrounding the teat sphincter (Plate 2). Keratin acts as a type of blotting paper mopping up the bacteria that are trying to penetrate the teat canal. Any damage to the teat canal is likely to increase the new infection rate.

Hyperkeratosis can be caused by problems due to worn liners where the liners open slowly or not fully, poor pulsation, rough removal of the cluster while under vacuum, and overmilking cows in plants with poor pulsation possibly compounded by high vacuum levels.

### 3. Impact forces or RPGs (Reverse pressure gradients)

Impact forces result in milk particles being propelled up the short milk tube or claw piece against the teat end. This may occur when there is a pressure difference between the teat end and the cluster. Impact forces are most frequently caused by liner slip (Plate 3) where the liner slips down the teat allowing air to suck in between the side of the teat and the top of the liner. This results in a difference in vacuum levels between the teat-end and claw piece and need only occur for milliseconds to create impact forces.

**Table 1** shows that large irregular vacuum fluctuations result in a large increase in the new infection rate compared to a plant with good vacuum stability. Irregular vacuum fluctuations may be due to inadequate vacuum reserve, faulty equipment or a poorly designed plant.

With impact forces, milk particles may be driven up against the teat at speeds of up to 40 miles per hour. This force is such that penetration of the canal may occur. If these droplets are contaminated with bacteria, then infection may follow.
Some of the more common causes of liner slip are listed in Table 2. The majority of liner slips result in ‘squawking’ of air as it enters through the top of the liner. They tend to occur towards the end of milking and so pose two dangers. Firstly, there is a little resistance at the teat end because the canal is at its most open phase. Secondly, if bacteria penetrate, because there is very little milk left to flush through the teat canal, it is more likely that they will remain in the udder until the next milking. This will allow time for multiplication and possibly infection to develop.

Impact forces combined with poor premilking 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, 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.

4. Colonisation of the teat canal
During milkout, ‘shear’ forces of milk flow through the teat canal, strip off excess keratin and in so doing remove attached bacteria. Keratin acts as a type of blotting paper trapping bacteria that are trying to invade the udder.

If there is poor pulsation and milk flow rates are reduced, the shear forces may be less able to remove this excess keratin. This may lead to a build up in bacterial colonisation of the teat canal. These bacteria can then continue to grow up into the teat canal and result in a new infection. This is also why once or alternate day milking is likely to produce more mastitis than twice or three times daily.

5. Over and undermilking
Provided that no teat damage results, there is no data which shows that overmilking can increase mastitis if carried out by more than five minutes in a machine that is working satisfactorily. If the machine is faulty, then any undermilking can cause problems.

Undermilking may affect mastitis, in particular Strep agalactiae infections. If the udder is not milked out fully, then bacteria will remain and increase in numbers between milkings.

This should not be overlooked as it does occur from time to time. Milkers don’t pick this up as they wear rubber boots. The key changes will be behavioural. Cows may be reluctant to enter the parlour, there is an increased frequency of urination and dunging. Cows often kick units off. The net effect of this is total
disruption to the milking routine, poor milk let down, a lot of faeces in the parlour and increased mastitis, cell counts and bactos cans. If in doubt, an electrical engineer can check out the plant.

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Table 1. New infection rates for stable and irregular vacuum fluctuations

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Table 2. Some causes of liner slip
- Milking cows with wet teats
- Worn or perished liners
- Low vacuum levels
- Poor liner design
- Heavy cluster weight
- Cows with small or large teats
- High vacuum fluctuation during milking
- Machine stripping at the end of milking
- Poor unit alignment