Milking Machines & Mastitis
– Sorting Fact from Fiction

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Introduction

At the end of lactation, dry cow therapy & appropriate culling are well accepted tools for controlling existing intramammary infections (identifiable as high SCC cows). Dry cow therapy also has the dual role of helping prevent new infections occurring through the dry period and decreasing clinical cases around calving time. By the time the cow leaves the colostrum mob however, milking management is the primary factor influencing new intramammary infections right through until the end of lactation. These new intramammary infections can then result in clinical mastitis and elevations in BSCC.

Milking Management encompasses several core areas which influence these new infections namely:

1. Shed design & equipment
    - Pipework, cluster, liners etc
2. Shed settings & machine function
    - Vacuum level & pulsation settings
3. Staff milking technique
    - Cup removal, teat spraying, cluster alignment etc
4. Response of the cow to the above influences
    - Milk out, teat condition etc

Deficits in milking management can be found in both old & new sheds although primary problems with pipework capacity are generally more of an issue in older sheds. The cost of rectifying these deficits vary from a major plant upgrade, through to changing liners or simply improving staff milking technique.

It is therefore important for herd owners to have a reasonable understanding of how milking machine function & management can influence mastitis to help recognise some of the more common problems & risk factors found in sheds e.g. teat end damage or poor teat spraying. This paper will describe how milking machines can spread mastitis and how to recognise some of the indicators of a machine related mastitis problem. Some examples will also be given of the more common problems encountered in respect to milking management.

The economics of mastitis

Simply reacting to a BSCC approaching 400,000 is not the basis for a proactive mastitis control program. By this point, due to the likely high infection rate within the herd, the damage has already been done on both a financial & animal health basis. The majority of milk companies in New Zealand offer no differential payout based on BSCC until a penalty threshold (400,000) is reached. Superficially, this system appears to provide little incentive for herd owners to strive for a lower BSCC. However, even without a differential payout it is important to understand that there are significant financial benefits in achieving a lower BSCC. This has been shown in overseas studies and also mirrored in a 2005 Dairy Insight study which assessed the financial impact of mastitis on the “average” New Zealand supplier.

The "average" supplier was defined as a 311 cow herd producing 320kgMS/cow with a BSCC of 212,000 in the 04/05 supply season. Please note that these financial calculations & costs relate to the 04/05 season (~$4.50/kg MS payout).
The study demonstrated that -

Each doubling of BSCC results in:

- 1.8% production loss for the season

The gains for the "average" supplier moving from a BSCC of 212,000 to the SAMM Plan recommendation of 150,000:

- Production gain of 2.8kg solids per cow or some $13 per cow
- Management gain (estimated) of some $7 per cow
- Total gain of $20 per cow or $6,221 for the average herd

The study also looked at the financial implication of clinical mastitis when averaged across the whole herd. Mastitis costs for the "average" supplier result in:

- Production loss of some $13 per cow
- Management loss of some $23.50 per cow
- Total cost of $36.50 per cow in the herd or $11,500 per average herd

Management losses refer to discarded milk, labour costs, antibiotic costs etc. Again these are historical costs based on 04/05 prices

Benchmarking for mastitis

Clinical Mastitis

Benchmarking for dairy herd health indices has historically been limited in New Zealand due to the lack of detailed health event recording across a sufficiently large number of dairy farms. Accurate recording requires a clear definition of the actual health event which in the case of clinical mastitis is when cows have visible inflammatory changes in the milk i.e. clots & discolouration possibly associated with heat and swelling of the associated quarter. In contrast sub clinical mastitis is associated with an elevated SCC but no visible changes in the milk. Some farmers choose to treat these subclinical cases through the season but it is important that they are not recorded as "clinical" cases. The majority of mastitis occurs in the Spring which is related to the increased susceptibility of the cow at calving time & the typically high environmental bacterial challenge. Suggested basic targets for clinical mastitis in Spring calving herds are -

- <5% of herd per month through calving then
- <1% of herd per month for the remainder of the season.

These figures refer to all cases of clinical mastitis including recurrent cases. Recording health event data does require a time commitment by herd managers but with improving technologies for collating & analysing data becoming available, hopefully this will minimise the time required and demonstrate the value of recording such information for the investigation of herd health problems. If more herds participate in health event recording then these targets can be fine tuned to a regional level e.g. spring conditions in Southland may be very different to those in North Canterbury.

Bulk Somatic Cell Count

The current SAMM plan guidelines serve as a sensible target i.e.

- No BSCC grades
- BSCC <150,000 all season.

Additional to this is the positive correlation between BSCC & incidence of clinical mastitis i.e. implementing good strategies for controlling clinical mastitis will normally also lower

BSCC and visa versa. The New Zealand study mentioned above demonstrates that these targets are not simply a "number chasing exercise" and have a strong financial basis.

The relationship between milking management and mastitis

Suboptimal milking management can cause mastitis by any of four mechanisms-

1. Teat end damage.

The only route that mastitis causing bacteria can gain entry to the mammary gland is via the teat canal. It therefore follows that any disruption to the canal & surrounding tissues increases the risk of new intramammary infections.

Objective assessment of teat condition after cluster removal is therefore a useful tool in assessing whether teat health is being compromised or not. Most will be familiar with overt teat end damage often referred to as "Blackspot". However, many more subtle but still significant forms of teat end damage often go unnoticed. These various conditions are mentioned below.

Hyperkeratosis is a thickening of the skin around the teat canal in response to physical trauma. Virtually every herd shows some degree of hyperkeratosis but by using an objective scoring system we can judge when this is excessive and requires intervention. If moderate to severe hyperkeratosis is obvious in your herd then it is likely that this teat end damage will be contributing to new infection rates and further investigations are justified. The diagrams below show different stages of hyperkeratosis.

Diagram 1 Normal teat end with the typical smooth white ring seen around the teat end after cluster removal

Diagram 2 Mild hyperkeratosis

Diagram 3 Severe hyperkeratosis

More severe damage to the teat end results in cracking of the skin around the teat canal as shown in Picture 1. Less than 5% of the herd should have evidence of this teat end cracking. This lesion can then progress to the infected, ulcerative lesion commonly referred to as "Blackspot".

Picture 1 Cracked teat end

There are other teat characteristics we can assess at cup removal related to changes in blood flow to the teat. These include

- haemorrhages (pin point red spots) around the bottom of the teat
- swelling and reddening of the teat and
- distinct ringing at the base of the teat.

These three changes represent recent damage to the teat and are also associated with higher intramammary infection rates. If these changes are ignored then mores serious damage such as Blackspot can ensue. Broadly speaking, less than 10% of cows should show these teat changes at cup removal.

Teats from newly calved cows are far more susceptible to teat end damage as the skin is often oedematous due to impaired blood circulation to the udder and teat. This accounts for much of the seasonality in teat end damage we see in New Zealand. Many herds experience a cyclical problem of teat end damage in the spring which appears to "resolve" in mid-late lactation only to return the following spring. In these cases the actual initiating "problem" in the shed hasn't actually resolved, rather the teats have become "hardened" to the milking process. Close inspection of these teats later in the season will often highlight low level damage. Any sign of teat end damage in the Spring is therefore worth investigating.

In turn there are 4 possible causes of teat end damage

- Liner choice
- Pulsation failure
- Excessive vacuum
- Overmilking.

The term overmilking refers to leaving the cups on a cow more than 1-2mins after milk flow through the cluster has stopped. Teat ends are far more prone to damage during periods of low milk flow (<1kg/min) hence the relationship with overmilking. Inadequacies in machine function can cause milk flow to cease even when milk is still present in the udder.

Liners are a common area for confusion. A decision on liner choice should be made based on both the shell type and also the specific shed set up. For example a wide bore liner may perform well in a rotary shed with cup removers but could predispose to teat end damage in a HB shed running at a high vacuum. Liners, pulsation & vacuum level are all interrelated in relation to milk out and teat health so it is dangerous to be too prescriptive for these and should be assessed on a case by case basis.

Environmental conditions can compound teat end damage by affecting skin quality. The cold, muddy weather conditions often seen in the Spring will dry out teats predisposing to cracking around the base when teats stretch during milking. Additional teat spray emollient is often used to help counter this drying effect but poor teat coverage is often a factor as well. These environmental conditions may also compound a hyperkeratosis problem. However, where obvious teat end damage is present, the primary contributing factor is normally a fault in milking management.

2. Undermilking

This refers to an excessive amount of residual milk left in the quarter after cluster removal. This is defined as more than ~80-100ml milk per quarter (4-6 good squirts). Well milked out quarters will have a wrinkled appearance down towards the teat base. Undermilking is one of the more common problems we see and is commonly associated with outbreaks of clinical mastitis & high BSCC in early lactation. The mechanism is thought to relate to inadequate "flushing" of the udder in terms of removing low levels of bacterial contamination within the

teat cistern. Failure to do so results in bacterial proliferation between milking time and an established infection.

Premature removal of cups can be the cause of under milking e.g. when ACR take off is triggered at too high a milk flow or staff remove cups too early. This can occasionally be a factor but more commonly it is the efficiency of milk out which is at fault. An example of this is when a cow stops milking but milk is still present in the quarters. Simply leaving the cups on these undermilked cows achieves little as there is minimal further milk flow and this simply serves to slow down milking time and predispose to teat end damage as previously mentioned. Alternatively, re-cupping or pressing down on the cluster ("machine stripping") usually results in further milk flow. This machine stripping is not only time consuming but can also result in more mastitis via teat end impacts.

Our goal is therefore to achieve rapid, complete milk out without compromising on teat health. Achieving good milk out is affected by numerous factors. Some of the more common are-

- Vacuum level. An excessively low or high vacuum can lead to undermilking
- Liners. Liner models vary in their milk out characteristics. Old, stretched liners although not obviously perished, will also slow milk out due to altered pulsation response. These should be replaced at least every 2500 milkings
- Cluster positioning in relation to the udder is a common problem. The cluster should be roughly in line with the backbone of the cow. Twisting of the cluster often results in uneven milk out. Excessive backwards pull can also result in both liner slippage and uneven milk out.
- Where air leaks are identified at machine testing ensure these are rectified. If these become excessive and then impact on vacuum reserve, the resulting unstable vacuum can result in poor milk out as well as liner slippage.
- Pulsation settings. Normally pulsation ratio is set at 60:40 but the exact setting is dependent on the influence of vacuum & liner type.

There will be the occasional cow with poor udder conformation which is virtually impossible to milk out fully without intervention. In a herringbone shed you then have to weigh up the pros and cons of slowing row time and overmilking other cows for the sake of this one animal. Unfortunately culling is often the only long term solution.

3. Impacts on the teat ends during milking.

A sudden drop in vacuum within the cluster during milking time can result in the propulsion of bacterial laden droplets back up the short milk tube hitting the teat end. In some instances these droplets can enter the teat canal, allowing bacterial entry into the gland resulting in a new infection.

Liner slippage is one example of how these impacts can occur. The characteristic noise is made by air being sucked between the liner mouthpiece and teat wall into the liner. It is most common in front quarters probably due to teat position relative to the cluster. Other risk factors include poor udder conformation, liner design, twisting or pulling on the cluster, machine stripping and low cluster vacuum level. Studies of cluster vacuum during milking have shown that liner slippage can occur without any associated noise.

Another less commonly recognised cause of impacts is poor cup removal technique. Releasing the cluster vacuum by flicking the claw button or pulling on a shell results in the same violent drop in cluster vacuum as occurs with liner slippage and can therefore spread mastitis via teat end impacts. The optimum technique is to clamp the long milk tube close to the cluster using the air admission hole to release the vacuum. The cups should then come away from the teats with minimal force.

4. Direct transfer of bacteria

This refers to the mechanism of physically transporting bacteria from one teat to another. The transfer of bacteria between cows' teats occurs via contaminated liners. Animals which have clinical mastitis produce a very high bacterial load per volume of milk and it has been shown that these bacteria can be transferred to the teats of the following five cows during milking. In practice, this means that any cow diagnosed with mastitis at cups on should be drafted out for milking after the main mob and clinical cases should be milked last as a separate herd. Flushing test bucket clusters during milking time is unreliable due to air pocketing in the liners and inadequate contact time if disinfectants are used.

Effective post milking teat spraying helps minimise the survival of any bacteria which do contaminate the teat during milking and prevents viable bacteria gaining entry to the open teat canal in the immediate post milking period. New Zealand trial work has shown effective teat spraying to decrease new infection rates by ~50%. Teat spraying should therefore be done all season, after every milking. Complete teat coverage is important and not just the teat end.

Conclusions

Regular monitoring for teat condition, degree of milk out, liner slippage & staff milking technique will help detect milking management problems. Some of these such as cup alignment & teat spray technique can be self regulated but where more complex problems such as poor milk out or teat end damage exist, further advice should be sought.