Milking Machines And Mastitis Risk: A Storm In A Teatcup

Graeme Mein[1], Douglas Reinemann[2], Norman Schuring[3] and Ian Ohnstad[4]
[1] Sensortec (NZ), Werribee, Australia; [2] UW-Madison, WI, USA;
[3] Westfalia-Surge, Naperville, IL, USA; [4] ADAS, Taunton, UK

Paper presented at the 2004 meeting of the National Mastitis Council

According to English tradition, a "storm in a tea cup" means that a particular issue has been blown out of all proportion. This quaint English interpretation can be applied to the **teatcup and cluster** because milking machine effects on mastitis usually are smaller and less important than the effects of milking management, herd management and cow or teat characteristics. Nevertheless, the inside of a teatcup can be a wet and windy place with rapid pressure changes and occasional storm damage. Our main purpose is to explain the dynamics of teatcup/teat interactions and their likely contribution to new infection rates in commercial herds.

In 1987, five main milking-related mechanisms of mastitis infection were proposed by an International Dairy Federation (IDF) Group of Experts lead by Jerry O'Shea (IDF 215, 1987). These mechanisms, summarized in Table 1, provide a convenient starting point. Although most of the information remains relevant today, new information and some new perspectives on old information are available to either strengthen or modify the conclusions of the IDF Group.

Table 1. Machine-related mechanisms potentially affecting new infection (from IDF 215:1987)

Mode of Infection	**Main Milking Related Mechanisms**	**General Evidence for Importance**
1. Changing numbers of bacteria on the teat or teat orifice	Transfer of bacteria from: a) Environment to Teat b) Cow to cow c) Teat to teat (within cows) increasing skin and/or orifice lesions	- Teat disinfection reduces bacterial numbers on the teat skin and orifice and decreases new infection rate - Experimental bacterial challenges increase new infection rate
2. Changing the resistance of the teat canal to bacterial invasion	By affecting: a) Teat canal integrity b) Teat congestion and/or oedema	- New infection rates are increased by reaming keratin from the teat canal and by visible teat canal injuries - New infection rates are increased when pulsation is ineffective
3. Providing forces to overcome the resistance of the teat canal to bacterial invasion	By causing impacts of: a) Microscopic droplets \ Inertia b) Macroscopic droplets / Effects c) Slugs of milk By inducing penetration associated with: d) Low energy pressure events and/or flowrates e) High energy pressure fronts	- Endotoxin, *E. Coli*, and dyes have been jetted through the teat canal - Shields or valves reduced new infection rates - High velocity air/liquid flows toward the teat end, increased new infection rates * Little or no published evidence (d-e)
4. Dispersing bacteria within the udder	By dispersing pathogens from: a) Teat canal to the teat sinus b) Teat sinus to the gland sinus and/or ducts	- Few infections occur if bacteria placed within the teat sinus are carefully removed by stripping but bacteria placed within the gland sinus frequently cause new infections - In some experiments pre-squirting of teats after milking reduced new infection rates
5. Frequency and/or degree of udder evacuation	By changing: a) Susceptibility of gland to invading pathogens b) Concentrations of pathogens on teat end c) Duration of exposure to pathogens	- New infection rates are higher in dried off cows at the start of the dry period - Incomplete milking or omitted milking tend to increase new infections or clinicals

1. Changing Numbers of Bacteria on the Teat or Teat Orifice

A superb review on the significance of levels of exposure to pathogens by Dodd (1987) was re-published, as a tribute to Frank Dodd, in a recent IDF Newsletter (IDF 318:2003). Dodd listed the following examples to support the conclusion that frequency of new infections increases with increasing level of exposure to mastitis pathogens.

- Milking-time hygiene techniques lower bacterial exposure and also reduce the rate of new infection.
- Reducing levels of infection in herds results in lower subsequent rates of infection.
- Rates of new infection in completely uninfected quarters are lower than in the uninfected quarters of cows with one or more infected quarters.
- Infection rates are increased when teat lesions colonised by mastitis pathogens are common.
- Data from artificial challenge experiments indicate that new infection rates are much greater than those normally occurring in herds that are subjected to natural levels of exposure to pathogens.

Dr Dodd also pointed out that not all of these observations demonstrate a direct (causal) relationship between new infection and level of exposure but that, overall, they provide strong support for the likelihood of a causal relationship. According to Smith (1997), the same logic applies to environmental streptococci. Smith concluded that "the level of exposure is the major risk for environmental streptococcal mastitis in today's dairy herds and we need to continually learn ways to keep cows clean, dry, cool and comfortable."

While correlation does not necessarily imply causation, we agree that pathogen concentration in or near the environment of the teat orifice has a dominant influence on rate of new mastitis infection. Nevertheless, direct effects of milking machine function (or malfunctions) on increasing the degree of contamination of pathogens, at or near the teat orifice, are likely to be quite low relative to the influence of milking procedures and herd management. The most obvious sources of cross-contamination within the milking machine are the claw and teatcup liners. Cross-contamination does not necessarily lead to new infections, however. New infection rates have sometimes remained quite low in the presence of high bacterial challenge. The implication is that factors other than the simple transfer of bacteria from a given teat to the skin of another teat of the same cow, or to the external surfaces of another cow's teats, should be considered.

2. Changing the Resistance of the Teat Canal to Bacterial Invasion

The conclusions of the IDF Group of Experts review of the effects of teat reaming to remove keratin, of management factors such as over-milking, and of machine effects such as vacuum level and pulsation failure are well known. Their conclusions (IDF 215:1987) support Dodd's view (Dodd, 1987) that "the main way that milking machines will influence the level of exposure is likely to be their direct effect on the health of the teat duct and the skin of the teat". Although most researchers and udder health specialists would agree with this view, it has been widely assumed to apply only or mainly to the contagious mastitis pathogens. This assumption appears to have been reinforced by the tendency to use only contagious pathogens in milking experiments involving high bacterial challenge until the early 1990s.

Jane Lacy-Hulbert was, perhaps, the first to demonstrate a clear link between a milking machine "fault" and higher new infection risk for *Str. uberis*, an organism previously regarded as an environmental pathogen. She reported a significantly higher rate of clinical infections (7 vs 0 for paired control cows) in identical twin cows milked without pulsation and subjected to a bacterial

challenge of 2.6 x 10^9 cfu/mL *Str uberis* (Lacy-Hulbert, 1998). Jane suggested that "reducing the keratin removal rate, such as by pulsationless milking, leads to a significant reduction in the keratin growth rate".
Woolford's thoughtful reviews of progress in understanding the effects of the milking machine on udder health included the following points on teat canal keratin (Woolford, 1995 & 1997).

- About 10-20% of the mature keratin cells lining the teat canal are lost during a single milking without pulsation whereas mechanical reaming of the teat canal removes up to 80% of the keratin.
- Both of these treatments increase the new mastitis infection rate, relative to milking with normal pulsation.
- Up to 40% of the keratin cells lining the teat canal are lost during a single milking with normal pulsation.
- These results suggest that regular removal of keratin during the milking process is desirable but that the degree of removal should be not too much or too little. Too much depletion of keratin (80%) will expose immature keratin cells that may be less effective at entrapping bacteria and sloughing off to remove the entrapped bacteria. Too little depletion (10-20%) may fail to remove the surface layers of keratin with any adherent bacteria and may slow the rate of turnover of keratin.
- Cyclic opening and closing of the liner promotes fracturing of the mature keratin layers within the teat canal, thereby increasing the effective shear forces acting on the keratin during milking. Periodic flushing of the canal during milking removes sufficient keratin (about 40%) to remove any bacteria trapped in, or adhering to, the surface folds of keratin.

Lacy-Hulbert's results prompted one of us (Mein) to review the original results of studies on pulsation failure conducted at the State Research Farm, Victoria, Australia (Mein et al. 1983). In those natural exposure experiments, the two-fold increase in new infections in quarters milked with shortened teatcup liners was due mainly to two major pathogens: *S. aureus* and *Str. uberis.* In the early 1980s, *S. aureus* was a common mastitis pathogen in the State Research Farm herd but *Str. uberis* was uncommon, especially during the middle part of lactation. With the benefit of hindsight, it is tempting to conclude that the increase in *Str. uberis* infections might have been due to teat-end changes resulting from the partial failure of pulsation applied in that experiment.

The effects of complete or partial failure of pulsation on teat-end condition, teat tissue responses, teat canal closure or teat skin condition, and on the risk of new mastitis infections, have been a recurring theme from the earliest days of milking machine studies. For example, Neave (1959) stated that "Much difficulty was experienced in designing the single-chamber teat-cup so as to prevent injury to the teats". The conclusion that milking with single-chambered teatcups was associated with a higher incidence of teat injuries, provided Mein & Schuring (2003) with their first major lesson from the scrapheaps of history during the last 100 years. This lesson was that:

> Successful milking is easier to achieve by means of a two-chambered teatcup that has a rubber liner and effective pulsation.

Effective pulsation is achieved when the combined actions of the pulsator and liner provide an adequate milk/rest time on the teat with optimum milk flow rates and minimal tissue changes evident at the end of milking. Pulsation is ineffective if teats are too long to allow complete collapse of the liner beneath the teat (Mein et al. 1983) or too short to be compressed by the collapsed liner (Hamann et al. 1994; Rasmussen et al. 1998). Examples of machine-induced

changes in teat condition are given in the Teat Club International materials (eg., Mein et al. 2001) and are available through NMC.

The results of both research herd studies and field observations confirm that, if pulsation is effective, new mastitis infection rates usually remain low despite the application of other practices or machine settings regarded as harmful or undesirable. For example, Mein et al. (1986) suggested that overmilking leads to more teat lesions and infections when, and perhaps only when, it occurs in conjunction with some form of pulsation failure. That suggestion was an over-simplification, however, as we shall see in the next section.

A storm in a teatcup: some risk factors for the "occasional storm damage"
To summarize Section 2, the risk of new infections by contagious as well as environmental pathogens such as *Str. uberis* is increased by machine-induced changes in teat-end condition. Such changes may include:

- increased congestion and oedema in the teat wall which results in slower closure of the teat canal and/or hypoxia in teat tissues (Hamann et al. 1994);
- slower rate of removal and regrowth of teat canal keratin (Woolford, 1997; Lacy-Hulbert, 1998);
- greater degree of openness of the teat canal orifice after milking (Mein et al, 2001),
- increased hyperkeratosis of the teat-end (Neijenhuis et al. 2001; Mein et al. 2003).

3. Providing Forces to Overcome the Resistance of the Teat Canal to Bacterial Invasion
The IDF Group of Experts (IDF 215:1987) provided a comprehensive review of the extensive scientific literature on irregular and cyclic vacuum fluctuations and liner slips, published during the previous 25 years. Our intention is to highlight the main results, to trace subsequent developments in liner design, and to offer some new interpretations.

Two Irish researchers were the first to show that inadequate vacuum pump capacity was associated with higher bulk milk cell counts (Nyhan & Cowhig, 1967). Subsequently, they produced the first research herd evidence to show that unstable vacuum was linked to an increase in new mastitis infection rate. Tragically, these Irish scientists died in an air crash en route to an international milking machine conference in 1968. After their deaths, the pioneering Irish work was continued at the National Institute for Research in Dairying (NIRD) in the UK and by a new Irish research team led, initially, by Jerry O'Shea and later by Eddie O'Callaghan.

A series of bacterial challenge experiments in the UK in the 1970s showed that 30-65% of udder quarters became infected when exposed to very high "cyclic" vacuum fluctuations, averaging over 40 kPa (12 inHg), in combination with experimentally-induced, large but relatively slow "irregular" fluctuations. In marked contrast, only 2-12% of quarters became infected when exposed to these astonishingly high cyclic fluctuations alone, or to large irregular fluctuations in combination with relatively small cyclic fluctuations, or to small cyclic plus small irregular fluctuations (Thiel et al. 1973; Cousins et al. 1973). "Impacts" were thought to be the main mechanism of infection. The term, "impacts", implied the rapid upward movement of small droplets or slugs of milk from the short milk tube towards the teat orifice. The new infection risk was higher if impacts occurred at or near the end of milking (Cousins et al. 1973).

One important result from this series of experiments in the UK is often overlooked in reviews of the effect of machine milking on mastitis: the first attempt to reproduce the results of Nyhan & Cowhig failed to show any treatment differences! New infection rates were NOT increased by high cyclic and/or large irregular vacuum fluctuations despite the immersion of cows' teats in a

high concentration of contagious pathogens before and after milking and the absence of post-milking teat disinfection (Thiel et al. 1973). The main "problem" was that the experimental clusters did not have a claw or a common milk hose or long milk tube. Instead, each of the four independent teatcups was connected directly to a separate recorder jar via its own milk tube. This result offers two important insights:

- Milking machine effects on mastitis are more likely to result from interactions between multiple factors rather than single-factor effects;
- Automatic milking systems may provide an inherently lower risk of mastitis because they incorporate independent teatcups, each with its own separate milk flow pathway from the individual udder quarters.

The new Irish research team also struggled, initially, to repeat the experimental results obtained by Nyhan and Cowhig. Their subsequent success resulted from a thoughtful observation by the herdsman who had worked with Nyhan and Cowhig. He commented that the original results had been obtained with a less stable type of teatcup liner than that used in the new Irish studies. The rest of this story is well known. O'Shea and O'Callaghan became famous for their research herd studies on effects of liner slips on new mastitis infection rate (O'Shea & O'Callaghan, 1978; O'Shea et al. 1987). Their conclusion was that the sudden inrush of air through one teatcup, which slips or falls during milking, drives droplets of milk through the claw and up into the adjacent teatcups. Cows may become infected if bacteria-laden milk droplets strike the ends of adjacent teats with sufficient force to carry pathogens into or through the teat canal, beyond the reach of a post-milking teat disinfectant.

This Irish "impact" mechanism is, essentially, the same mechanism as that proposed by the UK researchers but with one key difference. In the Irish studies, impacts were thought to result from "acute" irregular vacuum fluctuations with exceptionally fast rates of pressure change. Such acute fluctuations could be measured only in the adjacent teatcups within an individual cluster when one teatcup slipped. In the UK studies (Cousins et al. 1973), impacts were thought to result from high cyclic fluctuations acting together with comparatively slow changes in milking vacuum. The experimentally-induced "irregular" fluctuations in the UK studies took about 2 complete pulsation cycles to fall from 50 to 30 kPa (15 to 9 inHg) throughout the entire milking system. With the benefit of hindsight, it might be important to record that the extruded liner used in the UK studies was regarded as prone to slip frequently. Because liners tend to slip more frequently at lower milking vacuum, perhaps it is possible that liner slips contributed to the high new infection rates obtained in the UK treatment groups.

The effect of a 'high' versus a 'low' slip liner on new infection rate was assessed in the USA using a 160-cow research herd under conditions of natural exposure and post-milking teat disinfection (Baxter et al, 1992). Slips were recorded whenever a vacuum drop of 10 kPa or more occurred within a time of 0.25 sec or less. The 'high slip' liner averaged 7.6 major slips per cow-milking, compared with 3.1 for the 'low slip' liner. New infection rates were 0.49 per 100 cow-days for high slip compared with 0.27 for the low slip liner, which is about one new infection per 2500 liner slips for both the high and low slip liners. New infection rate was higher in cows that had one or more quarters already infected (1500-1850 slips per new infection) compared with the rate for previously uninfected cows (over 6000 slips per new infection).

A storm in a teatcup: some "wet and windy aspects"

The effect of liner slips on mastitis incidence has never been established in large-scale field trials. However, the economic importance of liner slips can be inferred from field studies with deflector shields or one-way valves fitted between the teat-end and claw. Field experiments using deflector shields in Britain and Australia indicated an overall reduction in new intramammary infections of

about 10% (from 20.6% to 18.4% of eligible quarters, Griffin et al, 1980). Similar results were obtained in field experiments with deflector shields in Norway (Binde et al, 1989) or with a valved claw in Britain (Griffin, Grindal and Bramley, 1988).

It is likely that these devices prevented all or most of the effects of liner slip, vigorous machine stripping and abrupt cluster removal on new infection rates. The implication is that these direct machine effects might cause 10% of the new mastitis infections on an 'average' farm. New infection rates may be above or below this estimate in an individual herd depending on factors such as the prevalence of sub-clinical infections and quality of milking management, and machine factors such as type of liner, bore of short milk tubes and claw volume. Slipping or falling early in milking often results from low vacuum level (especially in combination with excellent udder preparation), or with blocked air vents or restrictions within the milk hose or short milk tubes. Poor cluster alignment, poor liner condition or uneven distribution of the weight of a cluster, between the four quarters of an udder, are common causes of slipping and falling late in milking.

The common factor in published studies of machine-induced bacterial penetration of the teat canal has been an abrupt drop in vacuum within the teatcup or cluster. Thompson et al. (1978) calculated that a pressure difference of 25-40 kPa (7.5-12 inHg) between the claw and liner barrel was needed to generate air speeds within the short milk tube high enough to constitute a significant risk of infection by impacts. This critical pressure difference is markedly higher than the threshold of 10 kPa (3 inHg) set by Baxter et al. (1992) for recording liner slips.

Thiel et al. (1969) demonstrated that a stream of liquid containing endotoxin could penetrate the teat canal when directed towards the teat from a nozzle mounted in an experimental teatcup about 50 mm (2 in) below the teat orifice. Penetration of endotoxin into the teat sinus occurred at jet speeds of 6.2 or 9.8 m/s (20 or 32 ft/s) but not at 1.9 m/s (6.2 ft/s). [Note: complete penetration through the teat canal would be required to demonstrate a response when endotoxin is used to raise SCC in an individual quarter]. The lowest jet speed of 1.9 m/s was reported to be only just sufficient to keep the end of the teat wetted with endotoxin.

Such results beg the question: "What forces are available to generate air speeds of 2 m/s (6.5 ft/s) or more upwards through the short milk tube?" The rate of liner movement during a normal opening and closing cycle is surprisingly slow. Furthermore, most liners open 2-4 times more slowly than their rate of closure even when the lengths of the a- and c-phases are similar (Mein, 1992; Spencer, 2003). Spencer (2003) measured speeds of movement for 9 different liners. Liner opening speeds ranged from 33-75mm/s (mean: 53.6mm/s) while closing speeds were 112-235mm/s (mean: 166.7mm/s). Thus, the average speed of movement when the liner is opening is only about 0.05 m/s (less than 0.2 kilometre/hr or about 0.1 mile/hr). To put these numbers in another perspective, the typical rate of liner opening is about 40 times slower than a good walking speed for a relaxed adult and the rate of liner closing is 12 to 14 times slower!

Such slow rates of cyclic liner movement are unlikely to generate impact speeds high enough to induce bacterial penetration of the teat canal. Other conditions are required. Factors such as low pump capacity, poor vacuum regulation or limited capacity of milklines cannot (directly) generate transient pressure differences, within an individual cluster, that are capable of driving milk droplets at speeds greater than 2m/s towards the teat orifice. Such factors may have an indirect influence, of course, if they contribute to an increase in the frequency of liner slips.

The additional driving force required to impel milk droplets into a teat canal can be generated by sudden air leakage past one or more teats as a result of liner slips, vigorous machine stripping (only if slips occur) or abrupt cluster removal. Such events can produce very rapid, transient rates

of vacuum change within the liner (see Fig 2 in O'Shea & O'Callaghan, 1978, for example) and high transient air inflow speeds within the short milk tube (Woolford et al. 1980). By fitting a calibrated orifice, 7.5mm (0.3 in) in diameter, into the short milk tube, Woolford et al. (1980) measured average air inflow speeds of 6 - 8.3 m/s (20-27 ft/s) resulting from a simulated liner slip that was timed to occur just as the liner opened. In contrast, the average air inflow speed through the orifice was only 1.9 m/s in the absence of a simulated liner slip. Presumably, both the average speed and the estimated peak air inflow speeds would be reduced to roughly 50% of Woolford's values if the tube diameter was increased from 7.5mm (0.3 in) to 11mm (0.43 in).

Returning to the metaphor of a storm in a teatcup, the physical basis for the droplet impact theory seems clearer now. We can conclude that:

- A high gust of air blowing from the short milk tube up towards the teat end is not sufficient, by itself, to cause an infection.
- Some additional energy, (eg., the momentum of slugs or milk droplets picked up in the claw and lower end of the short milk tube), is needed to penetrate the defences of the teat canal.
- The speed of air flowing up the short milk tube must, inevitably, fall quickly as the air enters the dead-end space beneath the teat-end.
- Table 1 from the IDF Group correctly indicates that the 'impact' mechanism is an inertial effect for droplets of macroscopic size. However, this is unlikely to be true for microscopic droplets. Small droplets are effectively embedded in the air stream and, although they may reach the same high speed as the air moving up the short milk tube, they must decelerate and stop when they enter the dead space beneath the teat-end (Woolford et al. 1980).
- Larger droplets may be picked up in the claw bowl by sudden air admission from one short milk tube and impelled into adjacent claw inlets, then accelerated up the lower part of the short milk tube. If a milk droplet has reached a sufficient velocity, its inertia could carry it towards the teat end after the air inflow has decelerated and stopped. In support of this concept, it is worth noting that the frequency and intensity of teat-end impacts, recorded on an experimental test rig in the UK, were greater when a claw type with poor draining characteristics was used (unpublished data, personal comm. from D.Akam to G. Mein, 1980).

Reverse Pressure Gradients (RPGs)

Woolford et al. (1978) observed what they regarded as reverse flow at or near the end of milking in the course of their experiments with a Swinging Vacuum Single-Chambered (SVSC) milking system. Importantly, their observations of reverse flow occurred in the absence of a liner to close the teat canal.

Transient RPG's have been measured at the time of attachment of a liner to an empty teat, at the instant of teatcup removal or, occasionally, at times when air is admitted suddenly into a teatcup via the short milk tube (Galton et al, 1988; Rasmussen et al, 1994). High risk factors for RPG (Rasmussen et al, 1994; Rasmussen, 1995) include:

- teats with little or no milk to fill the teat sinus or to maintain a clear, continuous milk pathway between the udder cistern and teat sinus;
- use of liners having a small mouthpiece lip relative to the teat diameter;
- manipulation of empty teats before milking (and, presumably, during re-attachment of a slipping teatcup or a fallen cluster near the end of milking);
- detachment with liners stopped in their closed position (although detachment with liners pulsating normally or stopped in their open position is not risk-free).

To date, however, the RPG hypothesis remains unproven as a significant contributor to new mastitis infections. Based on physical principles, it seems unlikely that small transient RPGs can produce enough energy to penetrate the teat canal under normal milking conditions. In all situations where a two-chambered teatcup is used with effective pulsation, bacterial penetration of the teat canal has been demonstrated only in association with extreme pressure events.

4. Dispersing Bacteria within the Udder

Cinefilm and cine-radiographic studies as well as ultra-sonic techniques have shown that about one third of the milk volume present in a teat sinus just before the liner starts to close, is "pumped" back up into the udder cistern by the closing liner.

In 1967, Nyhan showed that only 2 new infections (out of 100 experimental quarters) occurred when bacteria were placed in the teat sinus but, before machine milking, the quarters were hand-stripped carefully to remove the foremilk from the teat sinus. In marked contrast, 33 of 172 quarters became infected when bacteria were placed within the teat sinus and the teats were manipulated to "milk" the sinus contents up into the udder cisterns before machine milking.

Despite these old research results, the practical importance of dispersing bacteria within the udder has not been established. Perhaps the only new information that can be added to this conclusion, reached by the IDF Group in 1987, are the results of a radiographic study by Williams (Mein et al. NMC, 2003). Williams' radiographs showed that the teat canal is first closed, under the influence of the closing liner, somewhere in the region between its mid-point and about one-third up the canal from the external orifice. This elegantly simple study provides a further demonstration of the value of an effective liner and pulsation in minimising the risk of moving any pathogens, which may have contaminated the teat-end, into or through the teat canal under normal milking conditions.

5. Frequency and/or Degree of Udder Evacuation

Compared with new infection rates in the early dry period or when milkings are omitted, the new infection rate is relatively low in cows that are milked regularly two or more times per day. Thus, machine milking has a positive effect in reducing the risk of new mastitis infection. In general, the clinical symptoms of mastitis are decreased as milking frequency is increased provided that teat-end condition is not compromised by milking too many times per day.

Discussion

Influence on liner development

The scientific revelations from the Irish and UK experiments in the late 1960s and early 1970s provided the springboard for three main development pathways of liner design.

> *1: Large-bore tapered moulded liners*. O'Shea and O'Callaghan's conclusion that the high transient pressure difference induced by liner slips or falls was the major mechanism of new mastitis infection, led to the evolution of a family of liners based on a key design goal to improve teatcup stability on the teat. This large-bore, tapered family of liners is characterised by an unusually large upper bore, a larger than average mid-bore and a tapered barrel with a relatively small diameter mouthpiece lip and small-bore short milk tube (further details in Mein et al. IDF, 2003). Cyclic vacuum fluctuations tend to be much higher in clusters fitted with such liners, especially when they are used with small volume claws and operated with simultaneous pulsation.

2: Medium-bore moulded liners with large bore short milk tubes. The cradle of evolution for this branch of liners may have been Scandinavia. Short milk tube diameters were increased to 12-14 mm to improve milk flow and drainage from the liner barrels during milking, especially when the liner was closing. Such free drainage reduced cyclic vacuum fluctuations at the teat-end and, presumably, reduced mastitis risk by breaking the "harmful" combination of high cyclic plus high irregular vacuum fluctuations. This design change seemed doubly attractive in that cyclic fluctuations could be reduced by a simple modification controlled by manufacturers of liners and claws. By contrast, manufacturers have no control over cluster handling techniques used by milking staff. It is common knowledge that poor cluster handling techniques can have an over-riding influence on the frequency and amplitude of irregular vacuum fluctuations.

Because very large-bore short milk tubes tend to kink more easily and may impair the ease of cluster handling and weight balance characteristics, slightly smaller bore sizes of 10-11mm have become more common.

3: Small-bore moulded liners with large-bore short milk tubes. Liners with mid-barrel bores as small as 18mm (0.7 in) were developed in the USA in the belief that minimising machine-induced congestion and œdema in teat tissues was important for cow comfort and udder health. Liners with small barrels and large-bore (10-11mm, or 0.4-0.43 in) short milk tubes helped keep the amplitude of cyclic vacuum fluctuations below 10 kPa (3 inHg) in a highline milking system or below 7 kPa (2 inHg) in a lowline system.

This initial introduction of narrow-bore moulded liners in the USA in about 1960 was reported to cause an increase in the incidence of teatcup slips and falls. However, California dairymen persisted in using such liners because they were associated with less mastitis (John Dahl, personal communication to Mein, 2003).

Teatcup and teat interactions and their likely contribution to overall infection rates

At the 1987 International Mastitis Symposium in Montreal, Canada, Dr. Eberhard asked the question "What percentage of all infections are due to milking machine factors?" The answers he was given were "we don't really know"; "probably quite low"; "anywhere between 0% and 100%". Can we provide any more definitive answers today?

Informed estimates of direct and indirect milking machine effects range from about 6% to 20% of the overall new mastitis infection rate. The direct effects (including bacterial transport, cross-contamination and impacts) might account for about 10% of new infections on most farms. Indirect effects (including effects on the health of the teat canal, teat tissues and skin) might account for another 10% in an average herd. However, it is difficult to go beyond these broad-brush estimates. When reading published articles or discussing field experience, remember that correlation does not imply causation. The world is full of testimonials and chance correlations. Attempts to correlate single-factor effects with poor teat condition or udder health often provide false hopes in reaching the ultimate goals of herd health and milk quality. Thus, we agree with the conclusion of Woolford (1995) that "further quantification of the overall contribution of the machine contribution is difficult and elusive because of the multi-factorial nature of the disease".

Despite these elusive difficulties, it is clear that the majority of new infections are caused by factors other than the milking machine. A significant reduction in machine-related infections might account for a change in the overall NIR of about 10%. Therefore, for a herd with a NIR of 10% per 100 cows per month, the number of new infections would be reduced by one per month.

The most likely improvement would be a reduction in the NIR in uninfected quarters of cows with one or more quarters already infected.

The value of a stable liner has been demonstrated in many research herd experiments and field observations. Machine-induced infections are more likely to result from acute transient vacuum fluctuations occurring in the liner during low milk flow periods. Liner slips, vigorous machine stripping and rough or abrupt cluster removal are potential causes of acute vacuum fluctuations. Other factors such as poor cluster alignment or improperly positioned long milk tubes also contribute to sudden air admission and, therefore, to fast rates of pressure change in the short milk tube and liners. High airflow rates are unlikely, by themselves, to cause infections. Additional energy in the form of fast-moving slugs or droplets of milk is needed to penetrate the defences of a healthy teat canal.

The value of effective pulsation has been demonstrated in both research herd and field experiments as well as in field observations. It is clear that:

- new infection rates are higher if a liner is absent or if pulsation is deliberately omitted
- new infection rates are higher if pulsation fails or if pulsator settings are outside acceptable limits
- pulsation fails (either partially or completely) if teats are too long or too short to allow the liner to collapse and compress the end of the teat.

Healthy teat ends are critical to the maintenance of low numbers of infected quarters. The condition of the teat should be analyzed immediately upon removal of the cluster at the end of milking. The Teat Club International has presented examples of the effects of poor liner action, ineffective pulsation and inadequate milking management on teat condition at the end of milking. Proper maintenance and operation of any milking system is a key aspect of successful milking.

Summary and Conclusions

- Most new infections are caused by factors other than the milking machine.
- Direct and indirect milking machine effects might account for 6-20% of NIs in an "average herd".

1. Contamination
   - Mastitis risk is a numbers game.
   - NIR is reduced by keeping bacterial numbers low on or near the cows' teat-ends.
   - Herd management and milking management practices probably have over-riding effects compared with the potential contribution from milking machines.
2. Teat health and teat canal integrity
   - NIR is reduced by pulsation characteristics that provide effective teat massage.
   - 'Effective pulsation' involves much more than the present industry pre-occupation with recording and analyzing pulsator rate and ratio plus the a, b, c & d phases of pulsation.
   - The key factor is the cyclic over-pressure applied by the closed liner to the teat tissues to overcome the dilating and congesting effects of the milking vacuum.
   - In addition to pulsator rate, ratio, the b and d phases, the main variables affecting the cyclic over-pressure applied to a teat are:
     - pressure conditions inside the liner barrel
     - geometry and mounting tension of the liner
     - physical properties of the liner material
     - teat size and shape

- As a tentative guide, the cyclic over-pressure should be about 8-12 kPa (2.5-3.5 inHg) above atmospheric pressure and this pressure should be applied for about 15% or 150 ms within each pulsator cycle.

3. Penetration of the teat duct
   - Air speeds > 2 m/s (6.5 ft/sec) up the short milk tube may assist bacterial penetration into or through the teat canal
   - Normal liner movement is much too slow to generate air speeds > 2 m/s.
   - The real action takes place within an individual cluster due to a sudden, transient air inrush through a teatcup when:
     - a liner slips or squawks loudly
     - a cluster is kicked off or detached abruptly
     - a cow is machine-stripped vigorously enough to break the seal between a teat and the liner mouthpiece
   - Such events can produce "acute irregular vacuum fluctuations". These are:
     - large (15-30 kPa; 4.5-9 inHg) transient drops in claw vacuum (often lasting less than 1-2 secs) with very fast rates of change (150-300 kPa per sec; 45-90 inHg/s)
   - The resulting high transient pressure gradients between the claw and adjacent liners can increase the NIR by accelerating milk droplets to speeds > 2 m/s towards the teat-ends in adjacent teatcups within the same cluster.
   - High cyclic fluctuations in cluster vacuum (up to 20 kPa; 6 inHg) are unlikely to generate air speeds > 2 m/s in the absence of sudden, unplanned air admission through one of the teatcups.
   - If, however, sudden air admission occurs within a cluster that induces such high fluctuations, the NIR is likely to be higher than 'normal'
   - Vacuum fluctuations in the milkline or receiver are too slow to increase the NIR unless they increase the frequency of slipping liners or cluster falls.
   - Correlations linking unstable milkline or receiver vacuum with increased mastitis are likely to be associative rather than cause-effect relationships. That is:
     - NIR is increased by sudden air admission through one or more teatcups
     - Air admission through teatcups is the primary cause of transient vacuum fluctuations in milklines.
   - The 'RPG' hypothesis remains unproven. It is unlikely that small transient RPGs can produce enough energy to penetrate the teat canal.
   - Eliminating the common claw (as in AM Systems) should reduce the NIR. Don't expect miracles with automatic milking, however, because:
     - A 10% reduction in NIR would mean, for example, 9 instead of 10 NIs per month per 100 cows. Other factors may mask this modest potential improvement.
   - Probably, the widespread use of more stable clusters, larger-bore SMTs and larger, free-draining claw bowls has already reduced the potential gain from eliminating the claw in many milking systems.

References

Baxter, J.D., G.W. Rogers, S.B. Spencer, and R.J. Eberhart. 1992. The effect of milking machine liner slip on new intramammary infections. J. Dairy Sci. 75:1015-1018.

Binde, M., H.P. Melby, A. Ask, A. Lang, and S.A. Vangdal. 1989. Effect of a shielded liner on new mastitis infection. J. Dairy Res. 56:55-59.

Cousins, C.L., C.C. Thiel, D.R. Westgarth, and T.M. Higgs. 1973. Further short-term studies of the influence of the milking machine on the rate of new mastitis infections. J. Dairy Res. 40:289.

Dodd, F.H. 1987. Bovine mastitis - the significance of levels of exposure to pathogens. Re-published in International Dairy Federation Newsletter 25, IDF Bulletin 318:2003, pp 3-6.

Galton, D.M., D.J. Aneshansley, L.G. Petersson, C.S. Czarniecki, and N.R. Scott. 1988. Pressure gradients across the teat canal during machine milking. Proc. Milking System Management, Harrisburg, PA. Northeast Reg. Agric. Eng. Service, Ithaca, NY, p 114.

Griffin, T.K., G.A. Mein, D.R. Westgarth, F.K. Neave, W.H. Thompson, and P.D. Maguire. 1980. Effect of deflector shields to reduce intramammary infection by preventing impacts on the teat ends of dairy cows during machine milking. J. Dairy Res. 47:1-9.

Griffin, T.K., R.J. Grindal, and A.J. Bramley. 1988. J. Dairy Research 55:155.

Hamann, J., O. Osteras, M. Mayntz, and W. Woyke. 1994. Functional parameters of milking units with regard to teat tissue treatment. International Dairy Federation Bulletin No. 297.

IDF 215: 1987. Machine milking factors affecting mastitis - a literature review. In International Dairy Federation Bulletin 215, Machine Milking and Mastitis, pp 2-32.

Lacy-Hulbert, J. 1998. Physical characteristics of the teat canal and the relationship with infection. Proc. 37th Annual Meeting of the National Mastitis Council, pp 54-61.

Mein, G.A. 1992. In "Machine Milking & Lactation" (ed. Bramley, A.J., Dodd, F.H., Mein, G.A. & Bramley, J.A). Insight Books, Huntington, Vermont, USA. pp 101-129.

Mein, G.A., M.R. Brown, and D.M. Williams. 1983. Pulsation failure as a consequence of milking with short teatcup liners. J. Dairy Res. 50:249-258.

Mein, G.A., M.R. Brown, and D.M. Williams 1986. Effects on mastitis of overmilking in conjunction with pulsation failure. J. Dairy Res. 53:17-22.

Mein, G.A., E. O'Callaghan, D.J. Reinemann, and I. Ohnstad. 2003. Pulsators and liners: where the rubber meets the teat. Proc. IDF Centenary Seminar, Belgium, "100 years with liners and pulsators in machine milking".

Mein, G.A., and N. Schuring. 2003. Lessons from scrapbooks and scrapheaps of history. Proc. IDF Centenary Seminar, Belgium, "100 years with liners and pulsators in machine milking".

Mein, G.A., D.M. Williams, and D.J. Reinemann. 2003. Mechanical forces applied by the teatcup liner and responses of the teat. Proc. 42nd Annual Meeting of National Mastitis Council.

Neave, F.K. Milking Machines and Mastitis. 1959. Chapt 4 in "Machine Milking", Bull 177, Ministry of Agriculture, Fisheries & Food, Her Majesty's Stationery Office, London.

Neijenhuis, F., G.A.Mein, J.S.Britt, D.J.Reinemann, J.E.Hillerton, R.Farnsworth, J.R.Baines, T.Hemling, I.Ohnstad, N.Cook, W.F.Morgan, and L.Timms. 2001. Evaluation of bovine teat condition in commercial dairy herds: 4. Relationship between teat-end callosity or hyperkeratosis and mastitis. Proc. AABP-NMC International Symposium on Mastitis and Milk Quality, Vancouver, BC, Canada, pp 362-366.

Nyhan, J.F. and M.J. Cowhig. 1967. Inadequate milking machine reserve and mastitis. Veterinary Record 81:122-124.

O'Shea, J. and E. O'Callaghan. 1978. Milking machine effects on new infection rate. Proc. International Symposium on Machine Milking, 17th Annual Meeting of National Mastitis Council, KY, USA, pp 262-268.

O'Shea, J., E. O'Callaghan, and B. Meaney. 1987. Liner slips and impacts. Proc. International Mastitis Symposium, Montreal, Canada. pp 44-65.

Rasmussen, M.D. 1995. The movement of bacteria by reverse pressure gradients across the teat canal. Proc. 3rd IDF Intnl. Mastitis Seminar, Israel. Eds. A. Saran and S. Sobach, Kimron Veterinary Inste. Israel, Book II, Section 7, p13.

Rasmussen, M.D., E.S. Frimer, and E.L. Decker. 1994. Reverse pressure gradients across the teat canal related to machine milking. J. Dairy Sci. 77:984.

Rasmussen, M.D., E.S. Frimer, L. Kaartinen, and N.E. Jensen. 1998. Milking performance and udder health of cows milked with two different liners. J. Dairy Res. 65:353-363.
Smith, K.L. 1997. Risk factors for environmental streptococcal intramammary infections. In Proceedings of Symposium held at Ontario Veterinary College, Canada, June 1997, on "Udder Health Management for Environmental Streptococci", pp 42-50.
Spencer, S.B. 2003. Defining the wave form of liner wall movement. Proceedings of IDF Centenary Seminar, Brugge, Belgium, "100 years with liners and pulsators in machine milking".
Thiel, C.C., C.L. Cousins, D.R. Westgarth, and F.K. Neave. 1973. Influence of some physical characteristics of the milking machine on rate of new mastitis infections. J. Dairy Res. 40:117.
Thiel, C.C., C.L. Thomas, D.R. Westgarth, and B.R. Reiter. 1969. Impact force as a possible cause of mechanical transfer of bacteria to the interior of the cow's teat. J. Dairy Res. 36:279.
Thompson, P.D., W.D. Schultze, J.N. Sauls, and S.C. Arapis. 1978. Mastitis infection from abrupt loss of milking vacuum. J. Dairy Sci. 61:344.
Woolford, M.W, D.S.M. Phillips, and A. Twomey. 1978. A comparison of mastitis infection rates using a conventional intermittent milk flow and a continuous milk flow under conditions of an elevated standard bacterial challenge. Proc. International Symposium on Machine Milking, 17th Annual Meeting of National Mastitis Council, KY, USA, pp 275-290.
Woolford, M.W., J.H. Williamson, and D.S.M. Phillips. 1980. Aspects of milking machine design related to intramammary infection. Proc. International Workshop on Machine Milking and Mastitis, Moorepark, Ireland, pp 45-59.
Woolford, M.W, 1995. Milking machine effects on mastitis progress 1985-1995. Proc. 3rd IDF International Mastitis Seminar, Israel, Book II, Section 7, pp 3-12.
Woolford, M.W, 1997. Perspectives on Mastitis from "Downunder". Proc. 36th Annual Meeting of National Mastitis Council, pp 56-64.