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PHYSICAL EFFECTS WITHIN THE TEATCUP RELATED TO INTRA-MAMMARY INFECTION

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SUMMARY

Bacterial challenge studies, using identical-twin cows, are described. One member of each twin set was milked with a machine having periodic air inlets to the cluster, synchronous with the liner opening, thus generating high velocity airflows towards the teat. The other twin member was milked without air inlets. Substantially higher infection rates were associated with the air inlet treatment when a standard bacterial challenge was applied.

INTRODUCTION

Numerous experimental studies of widely varying designs have variously implicated milking machine vacuum fluctuations as either predisposing to or as a casual mechanism in intramammary infection (Nyhan and Cowhig, 1969; Thiel *et al.*, 1973). However, specific physical effects at the teat end whereby vacuum fluctuations promote the penetration of organisms into the teat canal remain undefined, other than the proposal that the teat orifice is subject to impacts of high velocity milk droplets (Thiel *et al.*, 1969; O'Shea *et al.*, 1975; Thompson *et al.*, 1977; Thompson and Hayden, 1977; Callaghan and O'Shea, 1979) when air inlets to the system occur. The fitting of small deflector shields in the base of the teatcup liner has been effective in preventing such impacts (Thiel, 1974) and field trials (Griffin *et al.*, 1980) have shown variable reductions in new infection rates. Other studies (O'Callaghan and O'Shea, 1979) have failed to find shields fully effective in preventing transducer recorded impacts.

In all these studies the predisposing agent has been generally accepted as being liquid impacts on the teat orifice, the principal evidence for which rests with high speed differential pressure events recorded within the teatcup liner (O'Callaghan and O'Shea, 1979) and droplet impact sensing (Thompson and Hayden, 1977) in the short milk tube (SMT).

SMT airflows, as introduced by liner slip during milking, have recently been shown (Woolford *et al.*, 1980) to range up to 10 m/s and to depend on the magnitude and timescale of vacuum fluctuations within the clawpiece. This study also demonstrated an increase

in new infection rate under bacterial challenge conditions, resulting from defined SMT airflows. In conclusion it was hypothesised that a fast rate of change of pressure beneath the teat, as a consequence of high speed SMT airflows, may be the primary causal factor rather than droplet impacts. An infection mechanism induced by rapid pressure changes beneath the teat may also have been responsible for substantially higher infection rates reported for single chambered teatcups with an alternating vacuum level (Woolford *et al.*, 1978) wherein the physical design made droplet impacts unlikely.

This paper illustrates the magnitude of the air inlet-infection effect within conventional two-chambered teatcups.

METHODS

Fifteen sets of uninfected identical twins were split between two groups and milked with separate but identical milking systems, with the exception that the treatment group received air inlets of 0.5 second duration to one SMT of the cluster assembly every fourth pulsation throughout milking, with the intention of simulating the situation wherein one teatcup of a cluster slips during milking.

It was estimated from bench measurements (Woolford *et al.*, 1980) that the air inlets induced transient SMT airflows towards the teats in the other three teatcups of up to 10.4 m/s, depending on the magnitude of the vacuum depression in the claw (this varied with milkflow). The airflow duration was typically 75 milliseconds. Fig. 1 shows typical slow speed vacuum recordings at the claw-piece outlet for one member of a twin set receiving air inlets, the other not.

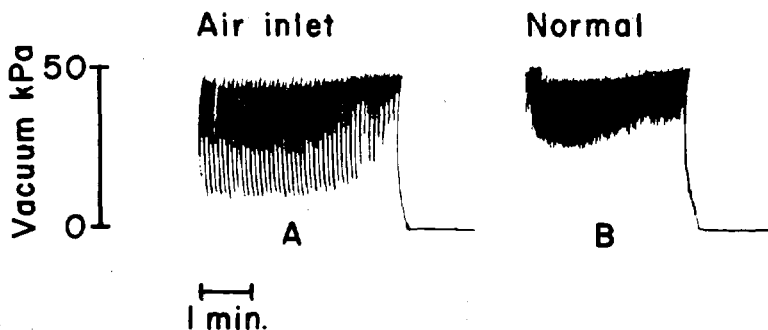


FIG 1: Vacuum recordings taken at the claw with (A) and without (B) air inlets to one SMT of the cluster.

The velocity of the SMT airflow generated by the air inlet was maximised by electronically timing the air inlet to coincide with the opening phase of the teatcup liners. Typical recordings of SMT airflow velocities (Woolford *et al.*, 1980) are shown in Fig. 2 both with and without air inlets. This velocity recording was obtained by recording the differential pressure across a calibrated orifice inserted into the SMT.

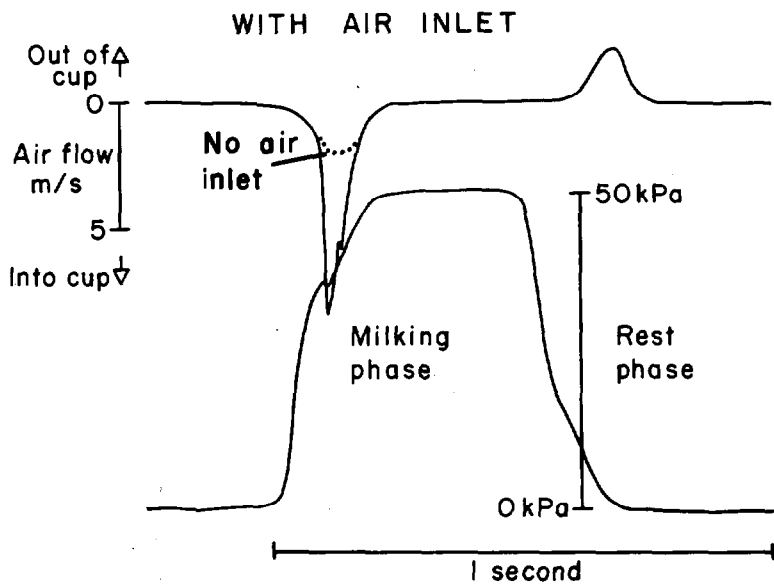


FIG 2: Recording of pulsation chamber vacuum (lower trace) and SMT air velocity (upper trace)

Two separate experimental periods used average SMT airflow velocities (due to liner opening + air inlet flows) of 6.0 m/s and 8.3 m/s in the treatment clusters, the SMT air velocity in the control clusters due to liner opening alone, being 1.9 m/s.

To standardise the infection challenge, all teats were dipped in a fresh 24 h mixed culture of *Staph. aureus* (10^8 /ml) and *Strep. agalactiae* (10^7 /ml) immediately prior to cup attachment. Teats were disinfected following cup removal. Aseptic samples of foremilk were drawn three times weekly for bacteriology and cell counting, a subclinical infection being defined as the isolation of a challenge organism at two consecutive samplings.

RESULTS

Table 1 gives the infection numbers and cell count levels for both experimental periods and clearly demonstrates an increased new infection rate associated with air inlet treatment, regardless of how infection is defined (Woolford *et al.*, 1980).

TABLE 1: INFECTION LEVELS AND TREATMENT DETAILS FOR THE TWO EXPERIMENTAL PERIODS

Experimental period No. of cow-milkings	I 1320		II 600	
	Treated	Control	Treated	Control
SMT peak air velocity (m/s)	6.0	1.9a	8.3	1.9a
Clinical infections	2	0	3	0
Subclinical infections	4	1	7	0
Cell counts (10^6 cells/ml)	15	4	23	0
Mean cell count (10^3 /ml)	234	124	270	142
Cow-milkings/infection	220	1320	60	600

a Due to liner movement

Over the total experimental period 16 clinical and subclinical infections by challenge organisms occurred among the treatment group (distributed between 10 cows) while only one subclinical infection was observed in the absence of air inlets. The somewhat higher infection rate among treatment cows in trial two may be attributable to the higher SMT airflow treatment. However, it may also have resulted from a higher level of pathogenicity in the bacterial challenge although the infection levels among the control group suggest this is unlikely.

INFECTION MECHANISMS

Interpretation of these results in terms of current concepts of the infection process would implicate the impact on the orifice of infected milk propelled towards the teat end by high velocity SMT air flows.

However high rates of pressure rise beneath the teat are a consequence of rapid SMT airflows but have not received consideration as the primary physical effect inducing teat canal penetration. A recent half-udder challenge experiment (O'Callaghan, 1980) has

shown a higher infection rate when using fast, as opposed to choked, air inlets. Localised small amplitude air displacements in the vicinity of the teat orifice associated with a rapid pressure rise may assist in introducing pathogen-carrying milk into the outer teat canal. Further, a small lag time in teat retraction while responding to a very fast local pressure increase is likely to result in a small inward displacement of the contents of a dilated teat canal. Such a displacement would require the upper teat sinus to be occluded, a small effective internal volume change and the liner to be close to the fully open position. Pathogens may thus be progressively transported deeper into the teat canal by such events and hence raise the probability of infection.

Direct droplet impacts on the external teat orifice appear to be the currently accepted mechanism of teat canal penetration. Evidence for such an effect is largely indirect or circumstantial, although there is little question that liquid milk does arrive at the teat surface as a consequence of air flows. The important question is at what velocity and whether such impact does any more than act as a vector in transporting organisms to the teat surface for some other, possibly multi-step, penetration mechanism.

CONCLUSION

A relationship between new infection rate and air inlets to the cluster has been well established by the results presented here and those of other workers.

Impacts of small milk droplets on the teat orifice may not be the primary infection mechanism. Physical events within the teatcup and at the teat orifice induced by rapid SMT air flows need more detailed scrutiny on a microscopic scale.

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