STUCK at home due to bad weather, I took a trip down memory lane when I came across a 2010 paper that aroused my interest because of its links with research work I did 40 years ago.

In the early 1960s, I worked at the Central Veterinary Laboratory (now VLA) in Weybridge and, like the authors of the paper, I was interested in the mechanisms and predisposing factors that caused intramammary infections (IMI) in cattle. My work culminated in a PhD in 1965.

Not all the work was published and, after getting my degree, I was offered another direction and I never again worked in mastitis. All these years later, and now happily retired, the paper made me realize how little has changed during 40 years.

In the intervening years, I did not even keep up with the literature in any diligent way, but I notice that, apart from three ‘standard’ references, the authors’ bibliography in general goes back to 1980. My bibliography obviously preceded theirs by many years, but I think the reader might be surprised by how much similarity existed between the research objectives in the two eras.

Murphy and Stuart (1954) had described a method for collecting milk from the teat cistern via teat wall puncture. Using this technique, I was able to confirm my findings to show that milk collected conventionally from the teat canal (with the usual aseptic precautions) could be infected, while milk taken directly from the teat sinus on the same occasion was less likely to be infected. This was a pure culture with no contamination in the conventional milk could be consistent with those interpreted to be due to IMI.

My early ideas led me to suspect that organisms might be transferred from the teat canal into the teat sinus by mechanical forces. It was my intention to investigate how IMI could become infected under normal management and milking conditions, and I wanted to avoid undue influence of the environment or the introduction of new organisms as markers. I had examined the bacteriology of naturally infected teat canals by swabs inserted 3mm into the canal and conventional hand-drawn milk and milk collected aseptically by syringing could persist for up to two days. The manual procedure may sound unduly invasive, but with acquired skill and experience — and the surprise cooperation of the cows — it caused no untoward effect.

The predominant organisms in the teat canals were Staphylococcus aureus, coagulase-negative Staphylococci and Micrococcus. All these organisms are commensal bacteria of the skin but could also establish IMI. It was the relationship of these microorganisms to the development of IMI that was of main interest. As all the bacteria were non-motile cocci, I labelled the mechanism by which they penetrated the teat canal was probably the same. The organisms that identified strain, allowing their epidemiology to be followed within the teat canals and the udders in the herd. By including all the Micrococcaceae that were present and samples, I had sampled the teat canals. Since the teat sinus by syringe. The basis of a comparison of weekly samples taken conventionally and those taken from the teat sinus by syringe.

In my conclusion was that the only definitive diagnosis of an IMI was by isolation of the organism from the teat sinus. Conventionally collected milk infected with recognized pathogens is also a very strong indicator when associated with an elevated somatic cell count (SCC). However, in the case of non-recognition of pathogens the elevation may be minimal, making diagnosis of an IMI equivocal. In investigating the passage of organisms through the teat canal the cause for the consequent effect, it is important to recognize the differing inflammatory response, because invasion through the teat canal is probably in an organocentric as well. I also showed that the acceptability of the gland to an invasion of a milk bacteria was dependent on the milk’s SCC, which was itself dependent on the presence of infections, many of which would never cause clinical mastitis. However, let us restrict our attention to the aetiology of the teat sinus, the prime site of invasion in the teat canal.

Mechanical stresses at the teat end of the teat cup liner

The resistance of the teat cistern to machine milking has been practiced. During the recent phase of the pulsation cycle the liner collars it to its maximum, approximately at its mid-length. The stresses on the teat depend not only on the shape of the teat end, but also on the diameter and the protrusion into the liner. Short teats will have relatively lower ‘crushing’ being in the space above mid-length, while for long teats, the teat end will experience much more pressure. However, with liner crowding towards the end of milk, all teats may be subject to the same stresses. Nevertheless, depending on shape and length, one can imagine the difficulty of possible distortions induced at the level of the teat canal.

The squamous epithelium lining the teat canal invades keratin, and because of the limited space in the canal, it can cause an erosion of the epithelium beyond the natural orifice, which I think may be the origins of the hyperkeratinized lesions observed. I can imagine the same phenomenon occurring at both ends of the canal and causing a keratin intrusion into the lumen of the teat canal. If keratin was carried an infection it could be a cause of IMI.

Also, if the keratin at the teat end was infected, I can imagine the action of the liner during milking gradually intruding keratin, that is, along the case of the non-recognition of pathogens the elevation may be minimal, making diagnosis of an IMI equivocal. In investigating the passage of organisms through the teat canal and the consequent effect, it is important to recognize the differing inflammatory response, because invasion through the teat canal is probably in an organocentric as well. I also showed that the acceptability of the gland to an invasion of a milk bacteria was dependent on the milk’s SCC, which was itself dependent on the presence of infections, many of which would never cause clinical mastitis. However, let us restrict our attention to the aetiology of the teat sinus, the prime site of invasion in the teat canal.

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The author’s PhD was on causes of intramammary infections.