Proceedings of the 12th International Symposium on Lameness in Ruminants

9th - 13th Jan. 2002,
Orlando, FL, USA.

Invited State-of-the-Art Lectures : Infectious Claw Diseases

Scroll down to view documents

ETIOLOGY, TREATMENT AND PROSPECTS FOR VACCINATION AGAINST (PAPILLOMATOUS) DIGITAL DERMATITIS

S.L. Berry[1], DVM, MPVM; D.H. Read[2], BVSc, PhD; R.L. Walker[3], DVM, MPVM, PhD; D.W. Hird[4], DVM, PhD

[1] Department of Animal Science, University of California, Davis, CA 95616, USA
[2] California Animal Health & Food Safety Laboratory, San Bernardino, CA, USA
[3] California Animal Health & Food Safety Laboratory, Davis, CA, USA
[4] Department of Medicine & Epidemiology, School of Veterinary Medicine, Davis, CA, USA

Introduction

Based on reports about histopathologic and immunohistochemical evidence that papillomatous digital dermatitis (PDD) and digital dermatitis (DD) belong to the same disease complex[34], the abbreviation (P)DD will be used throughout the text to designate both terms. (Papillomatous) digital dermatitis was first described in Italy in 1974 by Cheli and Mortellaro[16] and reported in the USA in 1980.[37] (Papillomatous) digital dermatitis has been reported in many countries of the world and is associated most commonly with dairy cows in confinement operations. Dairy producers in the United States first observed (P)DD on their dairies in the early 1990's.[40,49] (Papillomatous) digital dermatitis is characterized histopathologically by a combination of ulcerative and proliferative changes consisting of ulceration of tips of dermal papillae, epidermal hyperplasia with parakeratosis and hyperkeratosis, colonization and invasion by profuse numbers of spirochetes, and inflammation.[35] Lesions are circumscribed, erosive to papillomatous, painful, and often surrounded by hyperkeratotic skin with hypertrophied hairs.[35] The most common location of lesions is on the rear, palmar surface of the foot near the skin-horn junction bordering the interdigital space. Economic losses result from premature culling, decreased milk production, decreased reproductive efficiency, and cost of treatment.[37] British researchers calculated the economic loss from a case of (P)DD to be approximately $128 per cow/lactation.[23]

Etiology

The precise etiology of (P)DD is unknown but is thought to be multifactorial, involving environmental, management, and microbial factors.[15,35,39-41,48,50,51] Two risk factors identified in epidemiological studies are wet conditions and purchasing replacements from off-premises.[39,49,50] Read and Walker[33] hypothesized that animals may be predisposed to (P)DD by prolonged exposure of digital skin to oxygen-depleted, wet, organic material containing the causative organism(s). In more recent experimental studies, it was found that prolonged moisture and reduced access to air were necessary for successful transmission.[31]{3318} Other management conditions that may be involved in (P)DD are those thought to contribute to poor digital skin health, e.g. rough flooring, poor drainage, accumulation of feces and urine on floors, dirty, wet, or uncomfortable bedding areas, and overcrowding. The mode of transmission between cows and between herds is unknown but clinically and subclinically affected cows and fomites might be sources of infection for naive herds.

[1] Contact at: UC; Department of Animal Science, 1 Shields Ave, Davis, CA 95616-8521, USA; Office: 530-752-1279; Fax: 530-752-0175; E-mail: SLBerry@UCDavis.edu

All microbes associated with (P)DD have been obligate anaerobic or microaerophilic organisms.[9,11,17-22,29,48] The organisms found most consistently are spirochetes from the genus *Treponema*.[11,17-22,36,48] These spirochetes comprise the bulk of the colonizing mat of microbes found on active lesions and are also found invading the epidermis and dermis.[19,36,48] Sequential study of experimental (P)DD in calves demonstrated that spirochetes were the first bacterial morphotype to invade and colonize the epidermis and dermis.[32] Proliferative mature (P)DD lesions have gross and histological appearance similar to viral papillomas; however, bovine papilloma viruses have not been found.[15,33,35,37]

In a California study, antibodies against two antigenically distinct spirochetes were increased on dairies with (P)DD compared to (P)DD-free dairies.[47] Cattle with (P)DD on a high prevalence dairy were much more likely to have antibodies against the spirochetes than were cattle without lesions on the same dairy.[47] Each of the two (P)DD spirochete groups produced specific antibodies and did not cross-react with each other or with other common spirochetes associated with diseases of cattle.[47] Higher prevalence of clinical disease in younger animals of an endemically affected herd suggests that immunity may develop in older cows or that younger cows are more susceptible.[33] Chronic or recurrent cases in otherwise healthy adult cattle have been reported suggesting that if immunity to (P)DD does develop, it may be incomplete or temporary. Up to 60% of successfully treated cows may develop recurrent lesions in 7 to 15 weeks.[2,33] Spontaneous regression of lesions and resolution of lameness may occur but has been rarely observed.

Treatment

Early anecdotal reports indicated that (P)DD lesions responded to treatment with topical antibiotics, thereby supporting the hypothesis of bacterial etiology. Clinical studies confirmed the response to treatment with topical antibiotics.[2-7,10,13-15,24-27,35,38,42,43,46] There have been fewer reports of non-antibiotic products being efficacious.[13,14,27] In the United States there are no antibiotics labeled for treatment of (P)DD, and therefore a veterinary prescription is legally required for antibiotic treatment of (P)DD.

The most commonly used antibiotic treatments are topical oxytetracycline, lincomycin, or lincomycin/spectinomycin used as an aqueous spray or applied with a bandage.[1,2,4,5,13-15,25,27,35,42-46] For topical spray treatments, oxytetracycline or lincomycin are mixed with deionized or distilled water in a 2-4 liter agricultural sprayer (25 g/l Terramycin-343, 8 g/l Lincomix) and applied directly to the heels of (P)DD affected cattle after first washing the manure off the feet. The recurrence rate is high enough that topical treatments need to be repeated every 45-60 days on affected cattle to control the disease. We have achieved similar clinical results with either daily spraying of the lesions (once per day for 10-14 days) or a single treatment with oxytetracycline or lincomycin under a bandage. Antibiotic milk residue violations due to topical application of antibiotics have not been reported.[8,12,15] Parenteral antibiotics (Ceftiofur or procaine penicillin G) have been reported to be efficacious by U.S. researchers[26,35,36] but were not efficacious in European studies.[10,15] Parenteral antibiotics have the disadvantage of requiring milk withholding whereas topical treatment does not.

Footbaths containing 5% formalin[10,15,28], lincomycin (1-4 g/l)[15], oxytetracycline (1-4 g/l)[15,25,28], copper sulfate (0.25-1 g/l)[25,28], or zinc sulfate (20%)[28] are reported to provide

control of the disease in infected herds. On large dairies, footbaths may be more effective at controlling (P)DD when the disease is at a low prevalence (<5-10%). When the disease has a high prevalence, individual topical treatment is probably more effective in reducing the prevalence while footbaths may be helpful to decrease the incidence. Footbath efficacy is reduced if organic detritus are allowed to accumulate in the treatment solution. If two footbaths are placed in tandem, with the first containing water or a mild detergent solution for cleaning the feet and the second containing the antibiotic or antiseptic solution the treatment solution should remain active longer. The footbath solution should be changed every 150 to 300 cow passages depending on the dairy. Footbaths should be a minimum of 8 feet long and 2 to 3 feet wide, with a depth of 5 to 6 inches. The baths should be kept out of the rain and should be located in an exit alley off of the milking parlor to prevent contamination of the teat ends before milking. Additional footbaths can be placed in other locations to treat bulls, dry cows, and heifers. Treatments should occur every 3 to 7 days.[25]

Improving cow comfort by providing clean stalls, corrals, and alleys, providing dry and comfortable bedding, reducing the stocking rate, and improving ventilation to allow drying of stalls and alleys may decrease the incidence and/or severity of clinical cases. Hoof trimming equipment, mobile tilt tables, and livestock trailers should be thoroughly cleaned and disinfected to prevent potential transmission of the agent(s) of (P)DD between dairies.

Prospects for Vaccination

Spirochetal vaccines have been developed for leptospirosis (*Leptospira* spp) in cattle and for Lyme disease (*Borrelia burgdorferi*) in humans. These vaccines are considered effective in preventing disease, which would lead us to believe that it would be possible to make an efficacious vaccine against the (P)DD-associated *Treponema* spp. Research by Walker et al found humoral antibodies against the spirochetes in cows affected with (P)DD and no antibodies in cows without (P)DD, which indicates that the organisms cause an immune response in infected cows.[47] Although the preponderance of scientific evidence indicates that several distinct *Treponema* spp are involved with (P)DD, it is not clear whether they are primary pathogens or secondary to some other agents.

Preliminary prophylactic vaccination studies were conducted using a bacterin containing a proprietary adjuvant and washed, killed suspensions of 4 anaerobic bacteria commonly isolated from active (P)DD lesions by Dr. Richard Walker at UC Davis.[30] The bacteria were: (P)DD-associated *Treponema spp* Groups 1 and 2, *Porphyromonas levii* and an unidentified straight Gram-negative filamentous bacillus. Eight calves (6 vaccinated and 2 unvaccinated controls) were used in 2 similar trials, which lasted 10 weeks. Calves were purchased from a calf raising facility with no history of (P)DD and all calves used for the vaccination studies were seronegative to (P)DD-associated *Treponema* spp. by ELISA. Calves were vaccinated at the beginning of the study and given a booster 2 weeks later. Serum samples were taken weekly for measurement of (P)DD-associated *Treponema* spp. antibody levels by ELISA.

Experimental challenge was conducted by the method shown by Read and Walker to be effective in transmitting the disease.[31]{3318} Calves were challenged at week 5 of the studies (2 weeks after the 2nd vaccination). The inocula were made from scrapings of active lesions taken from cows clinically affected by (P)DD. Trial 1 ended on post-inoculation day 32 and

trial 2 ended on post-inoculation day 35. Lesions were photographed and lesion areas measured in square millimeters by computerized morphometry. Values for the number of lesions and total lesion area for vaccinated calves were compared to that of the unvaccinated controls. Histopathology was performed on biopsies taken from all lesions (D.H. Read, personal communication).

Transmission was 100% in both trials, which signified a successful challenge. The results differed between trials 1 and 2. In trial 1, the lesions regressed and resolved in the vaccinated calves and lesions progressed in the control calf. In trial 2, there were no obvious differences in lesion development, progression, size and activity between control and vaccinated calves. When lesion areas in vaccinated and control calves in both trials were compared, a statistically significant difference occurred, which suggested a vaccine-induced protective effect. However, the difference was considered to be too small to be clinically important. Histologic results confirmed gross evaluations. The vaccine did not produce any detectable humoral antibody response to the (P)DD-associated *Treponema* spp. in either trial.

While the results from trial 1 are somewhat encouraging, the lack of humoral antibody response to the *Treponema* spp. in the vaccine and the lack of clinical response to vaccination in trial 2 indicate that this vaccine was not efficacious. We are not able to explain why the vaccine did not elicit an antibody response to the *Treponema* spp. Antibodies against other organisms were not tested for. It has been amply demonstrated that *Treponema spp* are invasive, primary and dominant in the (P)DD lesions. The pathogenicity of the other anaerobic organisms isolated by Dr. Walker and others has not been investigated.

The production and testing of a *Treponema spp* bacterin seems to be a good first step in developing an efficacious vaccine against (P)DD. However, further research is needed to clarify the etiology of the disease and the possible virulence factors of the *Treponema spp.*

References

1. Berry SL. Footwarts: What do we know about the epidemiology and treatment? *Proc 1999 Hoof Health Conference, Modesto, CA* 1999;41-44.

2. Berry SL, Graham TW, Mongini A, Arana M. The Efficacy of *Serpens* spp Bacterin Combined with Topical Administration of Lincomycin Hydrochloride for Treatment of Papillomatous Digital Dermatitis (Footwarts) in Cows on a Dairy in California. *The Bovine Practitioner* 1999; 33(1):6-11.

3. Berry SL, Maas J. Clinical Treatment of Papillomatous Digital Dermatitis (Footwarts) on Dairy Cattle. *Proc 1997 Hoof Health Conference, Batavia, NY* 1997;4-7.

4. Berry SL, Maas J, Reed BA, Schechter A. The Efficacy of 5 Topical Spray Treatments for Control of Papillomatous Digital Dermatitis in Dairy Herds. *Proc.Amer.Assoc.Bov.Pract.* 29, 188. 1996. San Diego, American Association of Bovine Practitioners. 9-12-1996. (Abstract)

5. Berry SL, Read DH, Walker RL. Topical Treatment with Oxytetracycline or Lincomycin HCl for Papillomatous Digital Dermatitis: Gross and Histological Evaluation. *Proc 10th Intl Symp on Lameness in Ruminants, Luzern, Switzerland* 1998;291-292.

6. Berry SL, Read DH, Walker RL. Recurrence of papillomatous digital dermatitis (footwarts) in dairy cows after treatment with lincomycin HCI or oxytetracycline HCI. *J Dairy Sci* 82[(Suppl 1)], 34. 1999. (Abstract)

7. Blowey RW. Interdigital Causes of Lameness. *Proc 8th Intl Symp Disorders Ruminant Digit, Banff, Canada* 1994;142-154.

8. Blowey RW. Studies on the Pathogenesis and Control of Digital Dermatitis. *Proc 8th Intl Symp Disorders Ruminant Digit, Banff, Canada* 1994;168-173.

9. Blowey RW, Carter SD, White AG, Barnes A. *Borrelia burgdorferi* Infections in UK Cattle: A Possible Association with Digital Dermatitis. *Vet Rec* 1994; 135:577-578.

10. Blowey RW, Sharp MW. Digital dermatitis in dairy cattle. *Vet Rec* 1988; 122:505-508.

11. Borgmann IE, Bailey J, Clark EG. Spirochete-Associated Bovine Digital Dermatitis. *Can Vet J* 1996; 37(1):35-37.

12. Britt JS, Carson MC, vonBredow JD, Condon RJ. Antibiotic residues in milk samples obtained from cows after treatment for papillomatous digital dermatitis. *J Amer Vet Med Assn* 1999; 215(6):833-836.

13. Britt JS, Gaska J, Garrett EF, Konkle D, Mealy M. Comparison of Topical Application of 3 Products for Treatment of Papillomatous Digital Dermatitis in Dairy Cattle. *J Amer Vet Med Assn* 1996; 209(6):1134-1136.

14. Britt JS, McClure J. Field Trials with Antibiotic and Non Antibiotic Treatments for Papillomatous Digital Dermatitis. *The Bovine Practitioner* 1998; 32(2):25-28.

15. Brizzi A. Bovine Digital Dermatitis. *The Bovine Practitioner* 1993; 27:33-37.

16. Cheli R, Mortellaro C. La Dermatite Digitale Del Bovino. *Proc VIII International Meeting on Diseases of Cattle* 1974;208-213.

17. Choi BK, Nattermann H, Grund S, Haider W, Gobel UB. Spirochetes from Digital Dermatitis Lesions in Cattle Are Closely Related to Treponemes Associated with Human Periodontitis. *Intl J Systematic Bacteriology* 1997; 47(1):175-181.

18. Collighan RJ, Woodward MJ. Spirochaetes and other bacterial species associated with bovine digital dermatitis. *FEMS Microbiol Lett* 1997; 156:37-41.

19. Demirkan I, Carter SD, Murray RD, Blowey RW, Woodward MJ. The Frequent Detection of a Treponeme in Bovine Digital Dermatitis by Immunocytochemistry and Polymerase-Chain-Reaction. *Vet Microbiol* 1998; 60(2-4):285-292.

20. Demirkan I, Walker RL, Murray RD, Blowey RW, Carter SD. Serological evidence of spirochaetal infections associated with digital dermatitis in dairy cattle. *Vet J* 1999; 157(1):69-77.

21. Doherty ML, Bassett HF, Markey B, Healy AM, Sammin D. Severe Foot Lameness in Cattle Associated with Invasive Spirochetes. *Irish Vet J* 1998; 51(4):195-198.

22. Döpfer D, Koopmans A, Meijer FA, Szakáll I, Schukken YH, Klee W et al. Histological and Bacteriological Evaluation of Digital Dermatitis in Cattle, with Special Reference to Spirochaetes and *Campylobacter faecalis. Vet Rec* 1997; 140(24):620-623.

23. Esslemont RJ, Peeler EJ. The scope for raising margins in dairy herds by improving fertility and health. *Brit Vet J* 1993; 149(6):537-547.

24. Graham PD. A survey of digital dermatitis treatment regimes used by veterinarians in England and Wales. *Proc 8th Intl Symp Disorders Ruminant Digit, Banff, Canada* 1994;205-206.

25. Guard C. Recognizing and Managing Infectious Causes of Lameness in Cattle. *Proc Amer Assoc Bov Pract* 1995; 27:80-82.

26. Guterbock W, Borelli C. Footwart Treatment Trial Report. *The Western Dairyman* 1995; 76(5):17.

27. Hernandez J, Shearer JK, Elliott JB. Comparison of topical application of oxytetracycline and four nonantibiotic solutions for treatment of papillomatous digital dermatitis in dairy cows. *J Amer Vet Med Assn* 1999; 214(5):688-690.

28. Mortellaro CM. Digital Dermatitis. *Proc 8th Intl Symp Disorders Ruminant Digit, Banff, Canada* 1994;137-141.

29. Ohya T, Yamaguchi H, Nii Y, Ito H. Isolation of *Campylobacter sputorum* from lesions of papillomatous digital dermatitis in dairy cattle. *Vet Rec* 1999; 145(11):316-318.

30. Oliver KH. The Efficacy of a Vaccine Developed for Papillomatous Digital Dermatitis (Footwarts) in Dairy Cattle. California Polytechnic University, Pomona, 1999.

31. Read D, Walker R. Experimental Transmission of Papillomatous Digital Dermatitis (Footwarts) in Cattle. *Vet Pathol* 33[5], 607. 1996. Ref Type: (Abstract)

32. Read DH. Pathogenesis of Experimental Papillomatous Digital Dermatitis (PDD) in Cattle: Bacterial Morphotypes Associated with Early Lesion Development. *Proc Seventy-Eighth Conf Res Workers Anim Dis, Chicago, IL* 78, No. 32. 1997. Ref Type: (Abstract)

33. Read DH, Walker RL. Papillomatous Digital Dermatitis of Dairy Cattle in California: Clinical Characteristics. *Proc 8th Intl Symp Disorders Ruminant Digit, Banff, Canada* 1994;159-163.

34. Read DH, Walker RL. Comparison of papillomatous digital dermatitis and digital dermatitis of cattle by histopathology and immunohistochemistry. *Proc 10th Intl Symp on Lameness in Ruminants, Luzern, Switzerland* 1998;268-269.

35. Read DH, Walker RL. Papillomatous Digital Dermatitis (Footwarts) in California Dairy Cattle: Clinical and Gross Pathologic Findings. *J Vet Diagn Invest* 1998; 10:67-76.

36. Read DH, Walker RL, Castro AE, Sundberg JP, Thurmond MC. An Invasive Spirochaete Associated with Interdigital Papillomatosis of Dairy Cattle. *Vet Rec* 1992; 130:59-60.

37. Rebhun WC, Payne RM, King JM, Wolfe M, Begg SN. Interdigital Papillomatosis in Dairy Cattle. *J Amer Vet Med Assn* 1980; 137(5):437-440.

38. Reed B, Berry SL, Maas JP, Schechter A. Comparison of 5 Topical Spray Treatments for Control of Digital Dermatitis in Dairy Herds. *J Dairy Sci* 79[Suppl 1], 189. 1996. Ref Type: (Abstract)

39. Rodriguez-Lainz A, Hird DW, Carpenter TE, Read DH. Case-Control Study of Papillomatous Digital Dermatitis in Southern California Dairy Farms. *Prev Vet Med* 1996; 28(2):117-131.

40. Rodriguez-Lainz A, Hird DW, Walker RL, Read DH. Papillomatous Digital Dermatitis in 458 Dairies. *J Amer Vet Med Assn* 1996; 209(8):1464-1467.

41. Rodriguez-Lainz A, Melendez-Retamal P, Hird DW, Read DH, Walker RL. Farm- and host-level risk factors for papillomatous digital dermatitis in Chilean dairy cattle. *Prev Vet Med* 1999; 42(2):87-97.

42. Shearer JK, Elliott JB. Preliminary Results From a Spray Application of Oxytetracycline to Treat Control, and Prevent Digital Dermatitis in Dairy Herds. *Proc 8th Intl Symp Disorders Ruminant Digit, Banff, Canada* 1994;182.

43. Shearer JK, Elliott JB. Papillomatous Digital Dermatitis: Treatment and Control Strategies - Part I. *Compend Cont Educ Pract Vet* 1998; 20(8):S158-S173.

44. Shearer JK, Elliott JB, Giesy RG, Injoque RE. Effect of Oxytetracycline Topical Spray Treatment on Prevalence of Digital Dermatis. *J Dairy Sci* 1995; 78(Suppl. 1):257.

45. Shearer JK, Elliott JB, Injoque RE. Control of Digital Dermatitis in Dairy Herds Using a Topical Spray Application of Oxytetracycline. *J Dairy Sci* 1995; 78(Suppl. 1):170.

46. Shearer JK, Hernandez J, Elliot JB. Papillomatous Digital Dermatitis: Treatment and Control Strategies - Part II. *Compend Cont Educ Pract Vet* 1998; 20(9):S213-S223.

47. Walker RL, Read DH, Loretz KJ, Hird DW, Berry SL. Humoral Response of Dairy-Cattle to Spirochetes Isolated from Papillomatous Digital Dermatitis Lesions. *Amer J Vet Res* 1997; 58(7):744-748.

48. Walker RL, Read DH, Loretz KJ, Nordhausen RW. Spirochetes Isolated from Dairy Cattle with Papillomatous Digital Dermatitis and Interdigital Dermatitis. *Vet Microbiology* 1995; 47:343-355.

49. Wells SJ, Garber LP, Wagner B, Hill GW. Papillomatous Digital Dermatitis on U.S. Dairy Operations (Footwarts). *National Animal Health Monitoring System (NAHMS)* 1997;1-28.

50. Wells SJ, Garber LP, Wagner BA. Papillomatous digital dermatitis and associated risk factors in US dairy herds. *Prev Vet Med* 1999; 38(1):11-24.

51. Zemljic B. Current investigations into the cause of dermatitis digitalis in cattle. *Proc 8th Intl Symp Disorders Ruminant Digit, Banff, Canada* 1994;164-167.

FMD IN THE UK – A PRACTISING VETERINARIAN'S PERSONAL EXPERIENCE

Roger W Blowey, BSc, BVSc, FRCVS

This paper is not intended to be a comprehensive scientific review of the UK 2001 FMD outbreak, but rather a personal perspective from a practising veterinarian who was deeply involved at one stage of the catastrophe. The presentation will be liberally illustrated with pictures taken during the incident.

When the first case of Foot and Mouth was announced on 19 February 2001, because it was in Essex, the other side of the country to our Gloucestershire practice, it seemed as if it would not have a major impact. The second case occurred in Northumberland, even further away, but then cases were reported in neighbouring counties and the pressure began to mount. Should we continue to offer a veterinary service? If we did, then there was clearly a risk that we might spread infection from farm to farm. Could we stand the risk of this happening? We were still receiving calls for emergency cases, so some farm visits were going to be essential. A meeting was held within the practice and we discussed what biosecurity measures we would impose upon ourselves. A letter was then sent to all of our clients informing them of these measures, partly to allow them the option of deciding for themselves whether they wished to continue using veterinary services, and partly to ensure that we were thoroughly committed to these actions ourselves! In 1968 the outbreak lasted for 7 months, so a total cessation of veterinary services was clearly impractical.

Some of the measures adopted were as follows

- Clean outer clothing at each farm, either waterproofs thoroughly soaked in disinfectant (and hopefully reasonably dry!) after the previous farm, or freshly laundered garments. (the latter was the common option during the summer)
- Minimise the amount of equipment taken onto each farm, and carry it in a clean polythene bag that could be discarded after each visit.
- Either park the car at the end of the farm drive, or on a very clean area of concrete within the farm area.
- On leaving the farm, wash arms, head and face in disinfectant, and disinfect the outside of the car (and often the floor mat) using a knapsack sprayer carried specifically for the purpose.
- At the end of each day, wash the car, including the floor mats. Remove all clothing from the car, including waterproofs and car seat cover, and put through a hot detergent wash. Soak boots overnight in disinfectant

If we visited a farm that subsequently went down with FMD then all other farms visited over the next seven days were placed under 'Form D' movement restrictions. This initially caused some consternation both to ourselves and to the clients placed under restriction, but it soon became part of our working life, and most people adapted to it well. Two of my colleagues joined the Ministry to work in the Foot and Mouth effort, leaving two of us in the Practice, with some assistance from one other colleague, to look after the remaining clinical work. This meant that we were working or on call on alternate nights for almost eleven weeks continuous.

As the first cases of FMD entered our own Practice, we feared for the worst. This was particularly the case as one of the outbreaks was in the centre of the Practice. Fortunately the owner insisted on immediate destruction of the affected animals and these were destroyed within a few hours of the diagnosis being made. However, it did not prevent the slaughter of all the stock on two of his neighbours' premises. In one case this was particularly unfortunate in that the 'contiguous cull' was not carried out until two weeks after infection had been confirmed at the adjacent premises. In retrospect the logical approach would have been to continue with intense clinical monitoring.

Why Cull?

If virus spread is to be controlled, it is vital that affected animals are slaughtered almost immediately and at an absolute maximum of 24 hours of disease being diagnosed. Virus excretion from animals in the early stages of infection is massive, especially from pigs, which excrete up to 400million infective particles per day. Cattle are particularly susceptible to infection from inhalation, with disease being recorded after inhalation of only 10 units, so a single pig excretes sufficient virus in one day to infect 40 million cattle! At the end of the cull, carcases are sprayed with citric acid, and although it would be preferable to destroy the carcases immediately, it is the culling of the animals, which has the most dramatic effects on reducing virus excretion rates. It was clearly not known where the next case would strike, and the logistics of sourcing sufficient railway sleepers, straw, wooden pallets and coal for a fire and then getting it all onto a suitable site on the affected farm (where access was often poor) was enormous. During the 1968 outbreak 'environment' was less of an issue, and many carcases were buried. In 2001, the Environment Agency was often unable to decide with sufficient speed whether or not the site was suitable for burial.

We also learnt that disease was most likely to spread to contiguous, i.e. adjoining, farms. To prevent the spread of infection, it was therefore decided that farms adjoining the infected premises would be eliminated as soon as possible, and preferably within 48 hours of the initial case being diagnosed. This is because the highest rate of virus excretion occurs in the 4-6 days *before* clinical signs are seen. Wait for clinical signs and you will get a further wave of virus spread. The "48 hour" contiguous cull policy was not implemented until late March, i.e. approximately four weeks after disease began. Initially it seemed an absolutely draconian measure. One very small flock of infected sheep could lead to the destruction of three or four dairy herds, pig units and other flocks of sheep, simply on the contiguous premises concept. The rate of slaughter was unimaginable and the problems of destruction of the subsequent carcases became enormous. It was during this period that several farms within our own practice were affected, and for a week or two it seemed that the disease was unstoppable. The fear within the Practice increased and for me personally reached a peak when disease was confirmed on my next door neighbour's premises. It is extremely easy to agree with a policy of contiguous culling until you are faced with the horror of having to agree to cull your own herd. Admittedly mine was an extremely small beef suckler herd and it is not my main livelihood, but I was still attached to those cows. I had fed and attended them for the past 14 years and they were due to calve within a week. It would have been absurd for 'the local veterinarian' to ask for an exception to the contiguous cull policy and I found myself having to agree to their immediate slaughter. Fortunately the anticipation was worse than the realisation and within a few hours I found myself dosing them with Rompun (zylazine), for

sedation, and standing beside them while they were shot. The carcases were removed within 24 hours, but as they bloated, one of my cows began to give birth. By that stage both calf and dam were already dead and all I had to look forward to was their stacking onto the massive funeral pyre which was being built at the neighbouring farm. On this farm they somehow had to build fires big enough to burn some 3000 sheep, 700 pigs, 400 dairy cows and approximately 300 beef animals. Fires were burning for 2 – 3 weeks.

Towards the end of April the number of cases began to subside, and it was realised many of the cases that had been diagnosed as positive on clinical grounds came back negative on test, so the rules were changed again. In most instances only *sheep* contiguous with an infected premises were culled. Unless they were very close to infected stock, contiguous cattle were monitored for clinical signs. The fate of pig units varied with the proximity of the unit.

Livestock Movement Ban

As the movement of animals represents the greatest risk for the spread of FMD, a total movement ban of all livestock was imposed on Friday 23rd February 2001. At the time this seemed a draconian measure, and in itself produced enormous problems. Nothing could be sold, not even for killing, and this remained in force for up to 6 weeks, when a relaxation of the rules allowed limited movements to slaughter. Pig units were grossly overcrowded, leading to vices, damage to buildings and, of course, severe cash flow problems. Cows and heifers calving on away ground could not be brought back for milking, and worst of all, many flocks of sheep were on pastures totally unsuitable for lambing. Lambs born into overcrowded and muddy fields got stuck in the mud and soon died. The problem was compounded by the fact that it was one of the wettest winters on record.

To alleviate some of the problems after a few weeks a 'Livestock Welfare Disposal Scheme – LWDS – was introduced. Government would purchase the animals at pre – FMD prices and either transport them direct to abattoirs specially designated for LWDS culls, or slaughter on farm and dispose of the carcases. The meat could not be used for human consumption because at that stage under EU Law meat sourced from FMD infected areas could not be sold on the open market. The rush of farmers trying to get stock onto the scheme was akin to a gold rush! Unfortunately the prices set for LWDS animals was above the market price, and this exacerbated the rush into the scheme. It was the responsibility of the practising veterinarian to confirm that sales into LWDS were on welfare and not economic grounds, which aslo produced difficulties. As all animals on the farm had to undergo individual veterinary inspection and certified free from FMD prior to transport or on-farm slaughter, this put even greater stress on our now understaffed practice.

By mid April, two months into the outbreak, limited animal movements were permitted, but only within the certified 'Infected Area', and then mainly either to slaughter or from one premises to another under the same ownership. Licences had to be issued for each movement, and for most movements, including to slaughter, every animal on the farm had to be individually inspected and certified free from FMD within 24 hours of the movement. The issuing of licences became the responsibility of the veterinary practice, which produced further changes in the way we worked. The licenses were later issued by the veterinary practices themselves and were categorised into

- Occupational. A short movement of less than 0.5km
- Local. Movement of not more than 5km between premises in the same sole occupancy in cleaned and disinfected vehicles
- Long Distance. Movement between premises of differing ownership, allowed for welfare reasons only, in a certified and sealed cleaned lorry and escorted by a 'supervising officer' from the Government

Decontamination of Premises

After the carcase disposal, the lengthy process of premises decontamination starts. All muck, and general organic debris is removed, stacked sprayed with citric acid and left to stand for 6 months to decontaminate. All hay and straw is burnt, and many of the wooden structures that cannot be adequately disinfected are also burnt. Earth floors are dug out to a depth of approximately 70 – 100mm, covered with lime and then with a layer of compacted stone. All surfaces are thoroughly washed: walls, floors, and roofs roof timbers, guttering, equipment and anything potentially in contact with FMD virus. The washing is extremely thorough. No trace of organic material is visible, and if anything is seen at the inspection, then the whole premises have to be washed for a second time. The cleaning is paid for by government, and the farmer has the option of doing it himself (and thereby generating some income after his stock have been culled) – or employing contractors. Often the cleaning process took months, and the cost of cleaning could be greater than the cull itself.

Restocking could start under licence after the decontamination was completed and when all sheep flocks in the area had been blood tested negative for FMD, or after 12months if the premises had not been cleaned.

What Went Wrong?

The infrastructure that we had in place to control Foot and Mouth was not prepared for the scale of the epidemic. We thought that the imposition of a total livestock movement ban throughout the UK on Friday 24 February was a draconian measure, but how wrong we were. If only the Government had not dithered for those few days, then we would not have seen the extensive outbreak that resulted. It was undoubtedly the first major mistake. At that time Government was unaware of the extent of both the degree of movement and the level of infection in sheep. There was a huge demand for lamb on mainland Europe and large numbers were being exported, keeping the UK price very buoyant. However, the announcement of Foot and Mouth disease, confirmed on Tuesday 20 February, meant that all exports were stopped. The lorries already loaded with sheep and on their way to mainland Europe, then had to be turned back and the sheep were then taken to various markets throughout the UK, particularly in Devon, Wales, Herefordshire and Northampton. The sheep were sold on Wednesday 21 and Thursday 22 February and because trade was good, they were purchased by a large number of farmers and spread around the UK. This was disastrous. Instead of having one single focus of infection, we now had large numbers of outbreaks throughout the country.

The second major problem is that disease in sheep can be quite difficult to diagnose. With the current Pan Asian FMD strain, lesions can be quite mild and many sheep will recover. There

was one horror story told by a veterinarian who examined 580 sheep individually, catching each one, sitting it up, examining all four feet and also the mouth. Lesions of disease were only found in three sheep, but disease was confirmed on subsequent laboratory examination. This sent shock waves throughout the veterinary profession. When examining sheep for Foot and Mouth, did we really have to go through each animal in flocks of one, two, three or four thousand, checking each individually before we could be sure they were free? As a consequence it is likely that some of the early cases were either totally missed or left too long and virus was allowed to disseminate.

Because speed of cull is all-important to prevent the spread of disease, it was not possible to take samples to confirm the diagnosis. If disease was suspected, culling had to start. The veterinary officer visiting the farm was instructed to phone a central office in London. The case would be discussed, and a decision on whether or not to cull was made. This procedure was bound to produce some errors, but it was preferable to cull occasional suspect herds that were negative than miss a herd and allow infection to spread. Preliminary results for our own county of Gloucestershire were a bit surprising however. Of the 78 cases 'confirmed' on clinical grounds, samples were only taken from 50 farms, and of these *only 13 tested positive,* an apparent error rate of over 70%. Possible reasons for this apparent high error rate include:

- inappropriate samples taken
- insufficient numbers of animals sampled (this was certainly the case with some sheep flocks)
- laboratory testing was carried out incorrectly
- clinical diagnosis was incorrect

We were told by the Pirbright Laboratory that FMD animals were initially virus positive, then antibodies formed after 3 – 5 days, so infected animals would always be positive for virus or antibody or both. If the lesions seen were not FMD, then what was the cause? An erosive condition on the anterior gum surface of sheep was later the subject of considerable discussion in the veterinary press. In retrospect it is a great shame that more samples were not taken. Even if there was not sufficient testing capacity at the time, samples could have been stored and tested at a later date. We cannot afford to make the same mistakes next time!

Should Vaccination Be Introduced?

This was a major topic of discussion at the height of the outbreak, and again later when the outbreak recurred in Northumberland. Many of the arguments against vaccination were political and economic. What would happen to vaccinated animals? Would they be culled soon after vaccination or would they be allowed to live out their natural life span? What type of movement restrictions would be imposed on vaccinated herds? Could meat and milk from vaccinated stock be sold on the open market? Because only cattle were to be vaccinated, and the main source of disease was in sheep, was there any point? As vaccinated cattle can remain as carriers for up to 2 years would vaccination prolong the outbreak? For some reason the answers to these questions were not immediately available, and unless the farming community could be persuaded that vaccination was a good thing, then it was pointless trying to go ahead. Initially some of the milk purchasers also stated their reluctance to purchase milk from vaccinated herds because they felt this might jeopardise any product export potential.

However, as all footpaths were closed, and we were lead to believe that the costs to our tourism industry were even greater than the costs to agriculture, was vaccination a cheaper option?

The Future

At the time of writing the end of the outbreak remains unknown. Although coming under control, sporadic cases continue to occur, and the disease is by no means eradicated. By June 2001, around 4.2 million animals had been slaughtered, compared with only 434,000 in the whole of the 1967 outbreak. Of these, 1.04 million were slaughtered under the Livestock Welfare Disposal Scheme, and 3.2 million were associated with the 1675 confirmed cases, 34% being from infected premises, 39% dangerous contacts, and 24% contiguous culls. The total loss represented 12% of the national sheep flock, 6% of our pigs, 5% of cattle and 4% of all dairy herds. It is highly probable that swill feeding to pigs will be banned and there will be tighter controls on the movement of sheep. One thing is certain. We must do our utmost to prevent a similar outbreak from occurring in the future.