Antimicrobial prescribing guidelines for feedlot cattle

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Foreword – antimicrobial prescribing guidelines for feedlot cattle

Antimicrobial resistance (AMR) is one of the most significant emerging threats to human and animal health. Loss of the shared resource of effective antimicrobials jeopardises our ability to manage common infections, an ability that we take for granted in the modern era but one that has not been available at other times in history. Addressing AMR is a global endeavour that requires effective, coordinated action to minimise impacts on people, animals, and our shared environment.

AMR is to some extent a natural phenomenon. Each time antimicrobials are used the susceptible microorganisms are killed and the resistant microorganisms survive. Over time, continued exposure to antimicrobials selects for resistance in microbial populations. Certain human actions accelerate this process of increasing resistance, and the single most powerful contributor to this is inappropriate use of antimicrobials. This includes underuse, overuse, and misuse, and applies to the use of antimicrobials in human and animal health and in agriculture.

In human health, AMR infections necessitate additional investigations, more complex and expensive treatments and longer hospital stays and can result in greater mortality. In animals, AMR infections result in poor animal health, welfare, biosecurity, and production outcomes. AMR infections in animals can result in the transfer of resistant bacteria to people through direct contact or via the food chain.

The Australian Government has been working to protect humans and animals from the effects of AMR infections for more than twenty years. This work recognises the ongoing importance of antimicrobial stewardship by all prescribers, including veterinarians, and the need for guidance on best practice prescribing to support stewardship practices. These guidelines provide an important resource to inform prescribing decisions by those working with feedlot cattle and complement guidelines available for other livestock sectors.

I thank all those involved in developing these guidelines and ask veterinarians working with feedlot cattle to use these guidelines in decision making relating to the use of antimicrobials. Your efforts to ensure best-practice prescribing will help to secure the ongoing availability of effective antimicrobials for use in animal health, promote health and welfare in feedlot cattle, safeguard Australia's global reputation as a producer of high-quality food products and support the global response to AMR.

Dr Mark Schipp
Australian Chief Veterinary Officer

Dr Paul Cusack
BSc, BVSc(Hons), MVSt, FANZCVS, M.Agribus, PhD

Paul is a consultant cattle veterinarian, ruminant nutritionist, and beef producer with post-graduate training in the intensive and extensive ruminant industries.

Paul has held various positions within the livestock and veterinary industries. Paul has lectured veterinary and animal science students at the Universities of Queensland, and Sydney, and at Charles Sturt University, where he is an adjunct Professor. He also serves as Head Examiner in Beef Cattle Medicine and Production for the Australian College of Veterinary Scientists, and teaches post-graduate courses in ruminant nutrition and beef production medicine through the Centre for Veterinary Education.

Paul is the principal of Central Veterinary Services Cowra and director of Australian Livestock Production Services, which consults to intensive and extensive cattle and lamb enterprises throughout Australia and overseas, as well as being owner/manager of a cattle trading enterprise.

Dr Tony Batterham
BVSc, MClinEpid, GradDipVetClinStud, GAICD

Tony is a senior veterinary consultant to beef feedlots and feedlot business unit manager, employed by Apiam Animal Health. He is a veterinary graduate from Sydney University and also holds a Diploma of Veterinary Clinical Studies from Sydney University and Master of Clinical Epidemiology and Graduate Certificate of Data Analytics from University of Newcastle. Tony is also a Graduate of the Australian Institute of Company Directors and has served two terms as an ALFA councillor.

His special interest is in advanced diagnostics for feedlot conditions, objective measures of animal welfare and antimicrobial stewardship. Tony also donates his time as an external lecturer in feedlot medicine to University of Melbourne and is currently a PhD candidate with Sydney University. He has led projects for Meat and Livestock Australia investigating benefits of acclimation and comparing diagnostic modalities for BRD in beef feedlots, and is currently involved in several projects researching autogenous vaccines and wearable technology for remote diagnostics and well being assessment.

Professor Jacqueline Norris
BVSc, MVS, PhD, FASM, MASID, Grad Cert Higher Ed.

Jacqui is Professor of Veterinary Microbiology and Infectious Diseases and Head of School and Dean at the Sydney School of Veterinary Science, University of Sydney. She is a registered practicing veterinarian and is passionate about practical research projects and education programs for veterinary professionals, animal breeders and animal owners. Her main research areas include: 1) Development of diagnostics and treatments for companion animal viral diseases; 2) Q fever; 3) Multidrug resistant (MDR) *Staphylococcus* species; 4) Infection prevention and control in veterinary practices; 5) Chronic renal disease in domestic and wild felids and 6) Factors influencing antimicrobial prescribing behaviour of vets and health professionals.

Professor Glenn Browning
BVSc (Hons I), DipVetClinStud, PhD, FASM

Glenn Browning is Distinguished Professor in Veterinary Microbiology, Director of the Asia-Pacific Centre for Animal Health and Director of Research at the Melbourne Veterinary School at the University of Melbourne. He completed a Bachelor of Veterinary Science with First Class Honours at the University of Sydney in 1983, a postgraduate Diploma of Veterinary Clinical Studies in Large Animal Medicine and Surgery at the University of Sydney in 1984 and a PhD in Veterinary Virology at the University of Melbourne in 1988.

He was a Veterinary Research Officer at the Moredun Research Institute in Edinburgh from 1988 to 1991, investigating viral enteritis in horses, then joined the staff of the Faculty of Veterinary Science at the University of Melbourne, and has been a member of teaching and research staff there since 1991. He teaches in veterinary and agricultural microbiology.

He is a Life Fellow of the Australian Veterinary Association, a Fellow of the Australian Society for Microbiology and past Chair of the International Organisation for Mycoplasmology.

He is a co-author of 320 peer reviewed research papers and book chapters, has edited two books on recent progress in understanding the mycoplasmas, and has co-supervised 66 research higher degree students. His research interests include the molecular pathogenesis and epidemiology of bacterial and viral pathogens of animals, the development of novel vaccines and diagnostic assays to assist in control of infectious diseases, and antimicrobial stewardship in veterinary medicine.

Dr Stephen Page
BSc(Vet)(Hons), BVSc(Hons), DipVetClinStud, MVetClinStud, MAppSci(EnvTox), MANZCVS(Pharmacology)

Stephen is a consultant veterinary clinical pharmacologist and toxicologist and founder and sole director of Advanced Veterinary Therapeutics– a consulting company that provides advice on appropriate use of veterinary medicines to veterinarians, veterinary organisations (Australian Veterinary Association, World Veterinary Association, World Organisation for Animal Health), state and national government departments and statutory bodies (APVMA, Department of Agriculture, Department of Health, US Environmental Protection Agency), and global organisations (OIE, FAO, Chatham House).

He is a member of the AVA Antimicrobial of Resistance Advisory Group (ARAG), a member of the ASTAG committee on antimicrobial prioritisation; in 2017 he became President of the ANZCVS Chapter of Pharmacology, and is a Stephen is a consultant veterinary clinical pharmacologist and toxicologist and founder and sole director of Advanced Veterinary Therapeutics– a consulting company that provides advice on appropriate use of veterinary medicines to veterinarians, veterinary organisations (Australian Veterinary Association, World Veterinary Association, World Organisation for Animal Health), state and national government departments and statutory bodies (APVMA, Department of Agriculture, Department of Health, US Environmental Protection Agency), and global organisations (OIE, FAO, Chatham House).

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He has more than 100 publications on which he is author or editor, including chapters on antimicrobial stewardship, clinical pharmacology, adverse drug reactions, use of antimicrobial agents in livestock, and antimicrobial drug discovery and models of infection.

He has been a teacher and facilitator of courses at the University of Sydney on food safety, public health and antimicrobial resistance since 2003.

He is regularly invited to speak nationally and internationally at a broad range of conferences and symposia, especially on the subjects of antimicrobial use, antimicrobial stewardship and risk assessment. He gave his first presentation on veterinary antimicrobial resistance and stewardship at the AVA Conference in Perth in 2000 and remains passionate about improving the use and effective life span of antimicrobial agents.

The 5R Framework for Good Antimicrobial Stewardship

Derived from: Page S, Prescott J and Weese S. *Veterinary Record* 2014;175:207-208.
Image courtesy of Trent Hewson, TKOAH.

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Core principles of appropriate use of antimicrobial agents

While the published literature is replete with discussions of misuse and overuse of antimicrobial agents in medical and veterinary situations there has been no generally accepted guidance on what constitutes appropriate use in the context of feedlot cattle. To address this omission, the following principles of appropriate use have been identified and categorised after an analysis of current national and international guidelines for antimicrobial use published in the veterinary and medical literature. Independent corroboration of the validity of these principles has recently been provided by the publication of a proposed global definition of responsible antibiotic use, derived from a systematic literature review and input from a multidisciplinary international stakeholder consensus meeting.[1] The 22 elements of responsible use are outlined below.

Pre-treatment principles

1. Disease prevention

Apply appropriate biosecurity, husbandry, hygiene, health monitoring, vaccination, nutrition, housing, and environmental controls. Use Codes of Practice, Quality Assurance Programmes, Herd Health Surveillance Programmes and Education Programmes that promote responsible and appropriate use of antimicrobial agents.

2. Professional intervention

Ensure uses (labelled and off-label) of antimicrobials meet all the requirements of a bona fide veterinarian-client-patient relationship.

3. Alternatives to antimicrobial agents

Efficacious, scientific, evidence-based alternatives to antimicrobial agents can be an important adjunct to good husbandry practices.

Diagnosis

4. Accurate diagnosis

Make a clinical diagnosis of bacterial infection with appropriate point-of-care and laboratory tests, and epidemiological information.

Therapeutic objective and plan

5. Therapeutic objective and plan

Develop outcome objectives (for example clinical or microbiological cure) and an implementation plan (including consideration of therapeutic choices, supportive therapy, host, environment, infectious agent and other factors).

Drug selection

6. Justification of antimicrobial use

Consider other options first; antimicrobials should not be used to compensate for or mask poor farm or veterinary practices. Use informed professional judgment, balancing the risks (especially the risk of selection for antimicrobial resistance (AMR) and dissemination) and benefits to humans, animals and the environment.

7. Guidelines for antimicrobial use

Consult disease- and cattle feedlot guidelines to inform antimicrobial selection and use.

8. Critically important antimicrobial agents

All antimicrobial agents should be carefully considered before use. However, there is a rating system for the importance of antimicrobial agents. In Australia, the Australian Strategic and Technical Advisory Group (ASTAG) on Antimicrobial Resistance has created a system that ranks the importance of each antibacterial for human and animal health as low, medium or high.[2] Use all antimicrobial agents, and especially those considered of high importance in treating refractory infections in human or veterinary medicine, only after careful review and reasonable justification.

9. Culture and susceptibility testing

Use culture and susceptibility testing when clinically relevant to aid selection of antimicrobials, especially if initial treatment has failed.

10. Spectrum of activity

Use narrow-spectrum antimicrobials in preference to those with a broad-spectrum of activity whenever appropriate.

11. Off-label (extra-label) antimicrobial therapy

Antimicrobials must be prescribed only in accordance with prevailing laws and regulations. Confine off-label use to situations where medications used according to label instructions have been ineffective or are unavailable and where there is scientific evidence, including residue data if appropriate, supporting the off-label use pattern and the veterinarian's recommendation for a suitable withholding period and, if necessary, export slaughter interval (ESI).

Drug use

12. Dosage regimens

Where possible, optimise dosage regimens for therapeutic antimicrobial use following current pharmacokinetic and pharmacodynamic (PK/ PD) guidance.

13. Duration of treatment

Minimise therapeutic exposure to antimicrobials by treating only for as long as needed to meet the therapeutic objective.

14. Labelling and instructions

Ensure that written instructions on drug use are given to the end user by the veterinarian, with clear details of the method of administration, dose rate, frequency and duration of treatment, precautions and withholding period.

15. Target animals

Wherever possible limit therapeutic antimicrobial treatment to ill or at-risk animals, treating the fewest animals possible.

16. Record keeping

Keep accurate records of diagnosis (indication), treatment and outcome to allow therapeutic regimens to be evaluated by the prescriber and permit benchmarking as a guide to continuous improvement.

17. Compliance

Encourage and ensure that instructions for drug use are implemented appropriately.

18. Monitor response to treatment

Report to appropriate authorities any reasonable suspicion of an adverse reaction to the antimicrobial medicine in either treated animals or farm staff in contact with the medicine, including any unexpected failure to respond to the medication. Thoroughly investigate every treated case that fails to respond as expected.

Post-treatment activities

19.Environmental contamination

Minimise environmental contamination with antimicrobials whenever possible.

20. Surveillance of antimicrobial resistance

Undertake susceptibility surveillance periodically and provide the results to all members of the production chain who handle or have responsibility for the administration of antimicrobials.

21. Continuous evaluation

Evaluate the prescribing practices of veterinarians continually, based on such information as the main indications and types of antimicrobials used in feedlot cattle and their concordance with available data on antimicrobial resistance and current use guidelines.

22. Continuous improvement

Perform an objective and evidence guided assessment of current practices and implement changes when appropriate to refine and improve infection control and disease management.

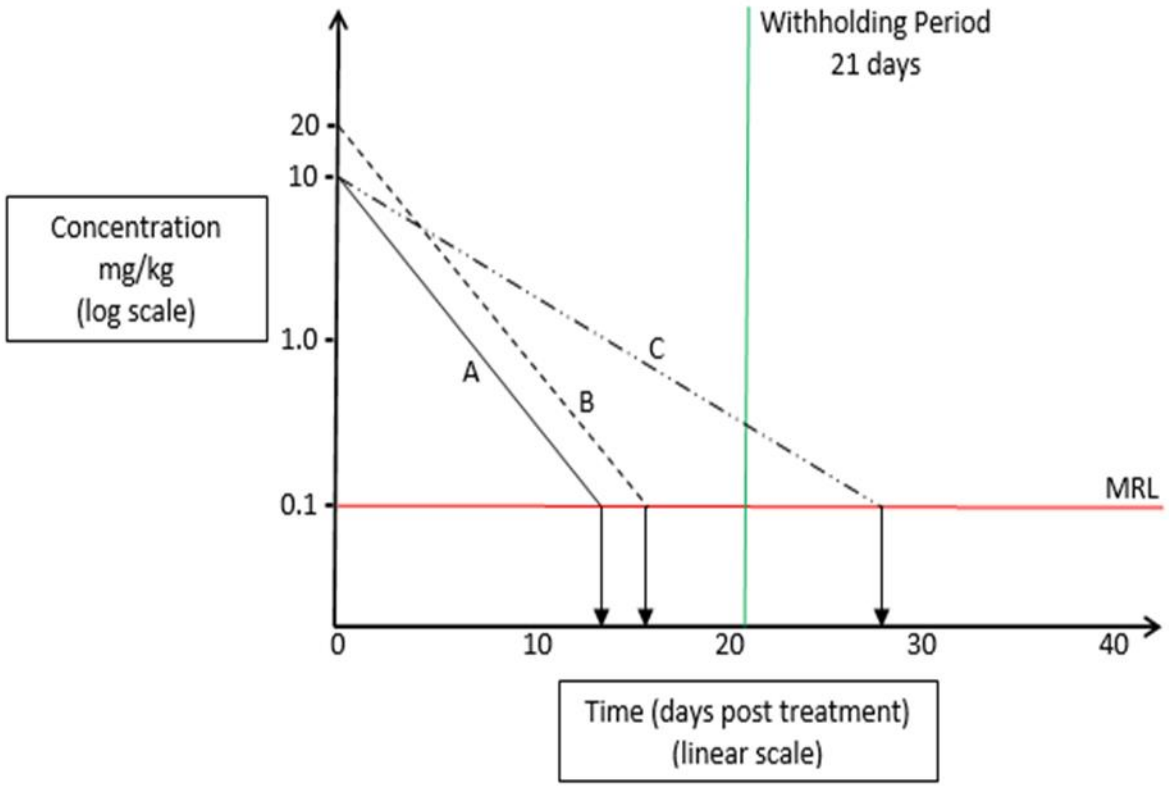
Core principles of appropriate use of antimicrobial agents.

Each of the core principles is important but **CORE PRINCIPLE 11 Off-label (extra-label) Antimicrobial Therapy** warrants additional attention as veterinarians treating production animal species, with professional responsibility for prescribing and playing a key role in residue minimisation, must consider the tissue residue and withholding period (WHP) and, if necessary, export slaughter interval (ESI) implications of off-label use before selecting this approach to treatment of animals under their care.[3, 4]

The subject of tissue residue kinetics and calculation of WHPs is very complex, requiring a detailed understanding of both pharmacokinetics (PK) and statistics, as both these fields underpin the recommendation of label WHPs. Some key points to consider when estimating an off-label use WHP include the following:

1. The new estimate of the WHP will be influenced by (i) the off-label dose regimen (route, rate, frequency and duration); (ii) the elimination rate of residues from edible tissues; and (iii) the maximum residue limit (MRL).
2. Approved MRLs are published in the MRL Standard, which is linked to the following APVMA website page: https://apvma.gov.au/node/10806.
3. If there is an MRL for cattle tissues, then the WHP recommended following the proposed off-label use must ensure that residues have depleted below the MRL for each tissue at the time of slaughter.
4. If there is no MRL for cattle tissues, then the WHP recommendation must ensure that no detectable residues are present in any edible tissues at the time of slaughter.
5. Tissue residue kinetics may be quite different to the PK observed in plasma, especially the elimination half-life and rate of residue depletion. The most comprehensive source of data on residue PK is that of Craigmill and colleagues.[5]
6. WHP studies undertaken to establish label WHP recommendations are generally performed in healthy animals.
7. Animals with infections may have a longer elimination half-life.
8. There are many factors that influence variability in the PK of a drug preparation, including the formulation, the route of administration, the target species, age, physiology, pathology and diet.
9. Note that the export slaughter interval (ESI) is frequently longer than the domestic WHP due to lower MRL's in export destinations. These lower MRL's must be used to determine an ESI for any cattle where any part of the slaughtered animal is to be exported. In effect, this means an ESI must be determined for any off-label drug use due to the dissemination of the various parts of the slaughtered animal to a range of varying export markets.

The following figure provides a summary of typical effects on elimination rates associated with drug use at higher than labelled rates and in animals with infections.



An example of the relationship between the maximum residue limit (MRL) and tissue depletion following administration of a veterinary medicine. In a healthy animal (A), tissue depletion to the MRL often occurs at a time point shorter than the withholding period (WHP), which has been established for the upper 95th confidence limit on the 95th percentile of the residue concentration in the treated population.[4] In such an individual animal, if the dose is doubled, tissue depletion (B) should only require one more half-life and would most likely still be within the established WHP. However, if the half-life doubles due to disease or other factors, depletion (C) would now require double the normal WHP and may still result in residues exceeding the MRL (adapted from Riviere and Mason, 2011).[6]

Table 1. Active ingredients for use in feedlot cattle – ASTAG ratings and formulations

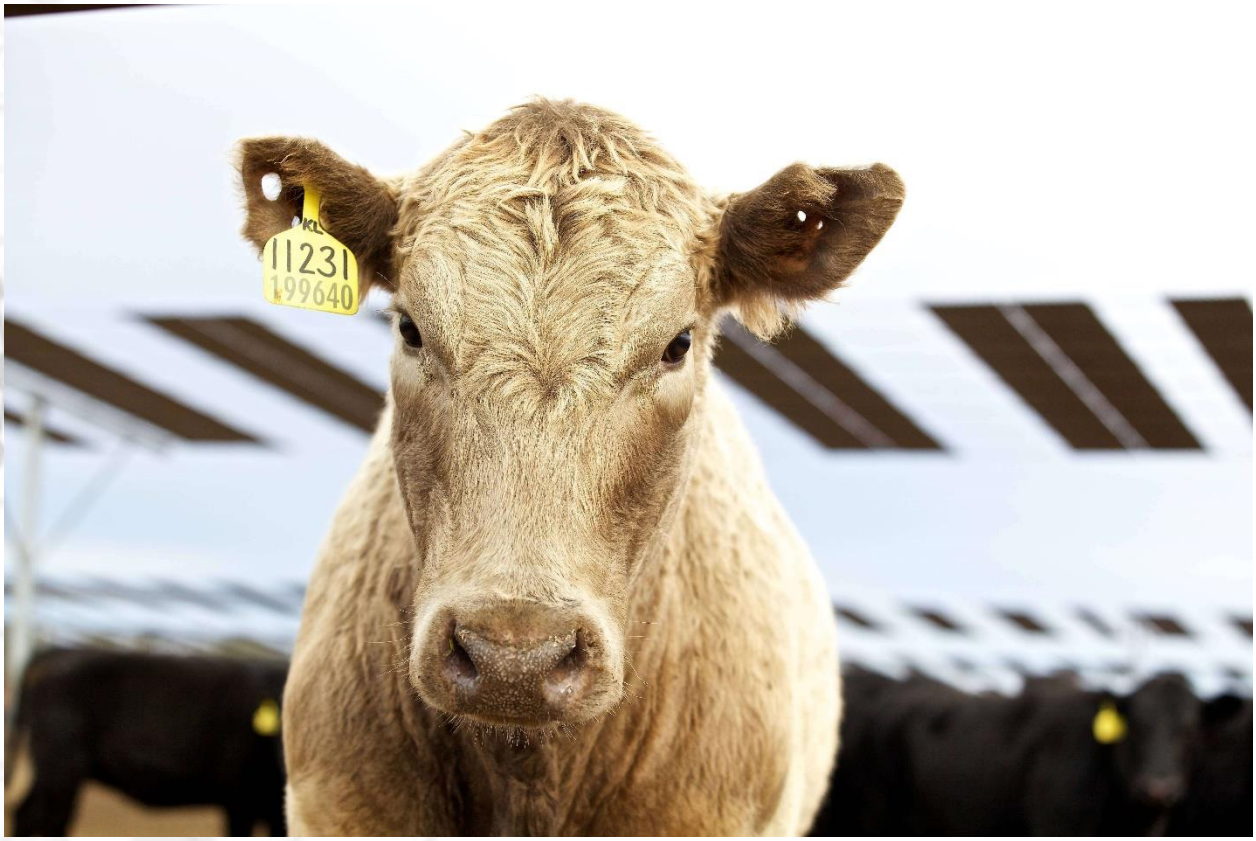
ANTIMICROBIAL AGENT	CLASS	IMPORTANCE (ASTAG 2018)	FORMULATIONS
AMOXICILLIN	Moderate-spectrum penicillins	Low	Injection
BACITRACIN | FRAMYCETIN | POLYMYXIN B | HYDROCORTISONE	Polypeptides, Aminoglycoside, Polymyxins	Low, Low, High	Eye, ear
CEFTIOFUR	3rd Generation Cephalosporins	High	Injection
CEFTIOFUR | KETOPROFEN	3rd Generation Cephalosporins	High	Injection
CEPHALONIUM	1st Generation Cephalosporins	Medium	Eye, ear
CEPHAPIRIN	1st Generation Cephalosporins	Medium	Intrauterine
CHLORTETRACYCLINE	Tetracyclines	Low	Feed
CLOXACILLIN	Antistaphylococcal penicillins	Medium	Eye
ERYTHROMYCIN	Macrolides	Low	Injection
FLAVOPHOSPHOLIPOL	Bambermycins	Low	Feed
FLORFENICOL	Amphenicols	Low	Injection
FLORFENICOL | FLUNIXIN	Amphenicols	Low	Injection
LASALOCID	Ionophores	Low	Feed
MONENSIN	Ionophores	Low	Feed
NARASIN	Ionophores	Low	Feed
NEOMYCIN	Aminoglycosides	Low	Feed
NEOMYCIN | LIGNOCAINE | HYDROCORTISONE	Aminoglycosides	Low	Topical
NEOMYCIN | PENICILLIN	Aminoglycosides	Low	Injection
OXYTETRACYCLINE	Tetracyclines	Low	Feed, injection, topical, intrauterine, water
OXYTETRACYCLINE | FLUNIXIN	Tetracyclines	Low	Injection
PENETHAMATE	Narrow-spectrum penicillins	Low	Injection
PENICILLIN PROCAINE	Narrow-spectrum penicillins	Low	Injection
PENICILLIN PROCAINE | PENICILLIN BENZATHINE	Narrow-spectrum penicillins	Low	Injection
PENICILLIN PROCAINE | PENICILLIN BENZATHINE | PROCAINE HYDROCHLORIDE	Narrow-spectrum penicillins	Low	Injection
SALINOMYCIN	Ionophores	Low	Feed
SULFADIMIDINE	Sulfonamides	Low	Water
TILMICOSIN	Macrolides	Low	Injection
TRIMETHOPRIM | SULFADIAZINE	Sulfonamides and dihydrofolate reductase inhibitors	Medium	Injection, intrauterine
TRIMETHOPRIM | SULFADIMIDINE	Sulfonamides and dihydrofolate reductase inhibitors	Medium	Injection

TRIMETHOPRIM | SULFADOXINE	Sulfonamides and dihydrofolate reductase inhibitors	Medium	Injection
TULATHROMYCIN	Macrolides	Low	Injection
TYLOSIN	Macrolides	Low	Feed, injection
TYLOSIN | OESTRADIOL | PROGESTERONE	Macrolides	Low	Subcutaneous implant
TYLOSIN | TRENBOLONE | OESTRADIOL	Macrolides	Low	Subcutaneous implant
VIRGINIAMYCIN	Streptogramins	High	Feed

For intramammary antibacterial products (containing AMPICILLIN; CEFUROXIME; CEPHALONIUM; CLOXACILLIN; DIHYDROSTREPTOMYCIN; LINCOMYCIN; NEOMYCIN; NOVOBIOCIN; OLEANDOMYCIN; or OXYTETRACYCLINE) refer to the AVA AMA Antimicrobial prescribing guidelines for dairy cattle: https://onlinelibrary.wiley.com/doi/10.1111/avj.13311 [50]

Alternatives to antimicrobials for prevention and treatment of commonly occurring feedlot diseases.

There is insufficient evidence to support the use of the following alternatives to antimicrobials for the treatment and prevention of commonly occurring feedlot diseases: nitric oxide; plant extracts; yeast or yeast products; bacterial probiotics; organic acids; bacteriophages; and non-specific immune stimulants.[7] Further research is warranted with lactate utilising bacteria, the organic acid malate, and bacteriophages. However, the use of malate has been constrained by expense and robust replicated positive results with bacteriophages have proved elusive to date.



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RESPIRATORY SYSTEM

Bovine Respiratory Disease

Body system/syndrome

Respiratory

Background/nature of infection/organisms involved

Bovine respiratory disease (BRD) is a syndrome caused by numerous stressors, viral transfer and infection, compromised immunity, and bacterial colonisation of the lower respiratory tract.[8] BRD is also referred to as undifferentiated fever in febrile feedlot animals with respiratory signs such as coughing, nasal discharge, dyspnoea/tachypnoea and non-specific signs of septicaemia. In beef feedlots, the incidence of BRD usually reaches a peak 7 to 50 days after arrival.

Principal viral agents involved include bovine herpesvirus 1, bovine viral diarrhoea virus, bovine parainfluenza (type 3) virus, bovine respiratory syncytial virus, bovine coronavirus, influenza D virus, and bovine rhinitis A and B. As these viral agents replicate in the respiratory tract, impairment of the mucociliary transport system and inflammation of the mucosal surfaces, coupled with a compromised immune system, can facilitate bacterial superinfection of the lower respiratory tract.

Bacterial agents primarily involved in pathological disease in the lower respiratory tract include commensal organisms from the nasopharynx: *Mannheimia haemolytica*, *Pasteurella multocida* and *Histophilus somni*. *Mycoplasma bovis* is not a commensal organism of the upper airways; evidence suggests that most cattle entering feedlots in Australia have not been exposed to it until induction, and that infection of most animals occurs early in the feeding period. [9, 10] Epidemiological studies indicate that it is a significant contributor to the syndrome and common in chronic presentations. *Trueperella pyogenes* and *Fusobacterium necrophorum* are commonly isolated from lung abscesses.

Bronchopneumonic lung consolidation tends to commence in the cranio-ventral lung lobes and then extends dorsally and cranially as the disease progresses.

Diagnostic Tests

Diagnosis of BRD on feedlots is routinely performed by livestock personnel, who have been trained by veterinarians on the larger scale operations, using subjective assessment of presumptive and often subtle clinical signs. The reference standard for BRD diagnosis remains autopsy and gross detection of respiratory pathology, such as bronchopneumonia, pleuritis, pleuropneumonia, pericarditis and lung abscesses.

Visual assessment of presumptive clinical signs (coughing, nasal discharge, dyspnoea/tachypnoea and non-specific signs of septicaemia) can be considered a screening test if confirmatory diagnostic tests are conducted in the (hospital) treatment crush. However, in many situations across the industry, visual assessment alone is used for both screening and confirmatory purposes. Visual assessment to diagnose BRD has been reported to have a sensitivity and specificity of less than 70% in US studies,[11] which emphasises the importance of training feedlot and livestock staff and monitoring of a range of health outcomes by the consultant veterinarian to foster constant improvement in the diagnostic accuracy of visual assessment.

Crush-side confirmatory tests that can be considered include:

1. Chest auscultation – standard and electronic
    a. Crackles/wheezes/moist and dry rales – experience is needed in detection and interpretation, as well as suitable restraint. Electronic stethoscopes are of low diagnostic value.
2. Rectal and rumen temperature
    a. The value of measurement at a single point in time is limited as it can be affected by disease chronicity, the ambient temperature, temperament, and activity immediately before measurement.
    b. Detection of temperature changes over time measured using a rumen bolus could provide more meaningful information.
3. Ultrasound survey
    a. Has a high degree of diagnostic sensitivity and specificity if the operator is experienced, the machine is suitable and there is adequate animal restraint. Examination should be bilateral, with a minimum of 5 min spent examining each hemithorax.

Identification of causal organisms using transtracheal aspiration and laboratory analysis (culture and antimicrobial susceptibility testing) can provide further information to guide drug selection and preventative programmes.

Serological testing using the ID Screen *Mycoplasma bovis* Indirect ELISA (IDvet) or testing of transtracheal aspirates by PCR can yield a more rapid diagnosis of infection with *Mycoplasma bovis* than culture. This can enable assessment of the contribution of this pathogen to BRD in a feedlot.

Key issues

1. Stress factors (transport, comingling, handling, adaptation to grain diet) adversely affect immunocompetence.
2. Commensal bacteria (nasopharynx) colonise the lower respiratory tract in immunocompromised feeder cattle given the right environmental circumstances.
3. Host tolerance of the subsequent bacterial infection varies between individuals. The extent of lung consolidation does not reliably predict a clinical outcome.
4. Treatment applied early in the disease course is the most important determinant of a successful outcome.

Treatment

Systemic antimicrobial therapy is the core element of treatment. Good husbandry practices, such as low stocking density, shade or shelter, appropriate nutrition, and suitable bedding in designated “hospital” pens are likely to improve the response to treatment in more advanced or severe cases.

Antimicrobial selection can be informed by recent bacterial culture or organism identification by PCR performed on samples from cases confirmed to have BRD at necropsy, but there can also be reasonable confidence that the main bacterial agents listed above are predominant in the majority of BRD presentations, as supported by global and domestic experience and literature.[12-15] It is common practice to utilise long-acting antimicrobial preparations to avoid repeated handling of cattle for multiple treatments as this may increase the case fatality rate.

Antimicrobial selection can also be influenced by days on feed at presentation and consideration of the WHP/ESI in relation to anticipated time of slaughter.

Table 2. The characteristics of antimicrobials used in the treatment of bovine respiratory disease (adapted from Riviere and Papich, 2018).

Antimicrobial Class & Individual Antimicrobials	Mode of Action	Spectrum	Distribution	Duration of Activity (therapeutic concentration)	PK-PD Parameter#	Route of Administration	ASTAG Rating
MACROLIDES **Tulathromycin** **Tilmicosin**	Bacteriostatic or bactericidal, depending on target organism – inhibition of protein synthesis by binding to 50S ribosomal subunit	Gram negative: *Mannheimia haemolytica, Pasteurella multocida, Histophilus somni* (most other Gram negatives are not susceptible). Gram positive: *Streptococcus* spp., *Staphylococcus* spp. (including those resistant to β-lactams) Mycoplasmas *Mycoplasma bovis*	Basic drugs that concentrate in cells more acidic than plasma, particularly the lungs, where concentrations persist.	Tulathromycin 7 d with single 2.5 mg/kg dose Tilmicosin 3 d with single 10 mg/kg dose	AUC/MIC >3	Subcutaneous	Low
AMPHENICOLS **Florfenicol**	Bacteriostatic or bactericidal, depending on target organism – interferes with peptidyltransferase activity at 50S ribosomal subunit (close to site of macrolide binding so these can interfere with each other)	Gram negative: *Mannheimia haemolytica, Pasteurella multocida, Histophilus somni Fusobacterium necrophorum* and *Prevotella melaninogenica* (*Bacteroides melaninogenicus*) Gram positive: *Trueperella pyogenes* Mycoplasmas *Mycoplasma bovis*	Wide distribution with high concentration in pulmonary epithelial lining fluid, synovial fluid, kidney, urine, bile and small intestine. CNS penetration can lead to concentrations above the MIC of *Histophilus somni*.	One dose, at 40 mg/kg, provides 4d duration of activity	AUC/MIC = 18-27	Subcutaneous	Low
BETA-LACTAMS **Amoxicillin** **Ceftiofur**	Bactericidal -prevent bacterial cell wall synthesis and weaken cell wall integrity using penicillin binding proteins to attach, ultimately causing cell wall rupture.	Amoxicillin Gram negative: *Pasteurella multocida, Trueperella pyogenes, Fusobacterium*	Effective concentration achieved for susceptible bacteria in kidneys, synovial	Amoxicillin Short duration. Give 7mg/kg once daily for up to 5d Give two doses 48 hours apart at 15 mg/kg dose	T>MIC	Amoxicillin intramuscular Ceftiofur sodium intramuscular	Low High

		necrophorum, Histophilus somni Gram positive: *Streptococcus* spp., non-penicillinase producing *Staphylococcus* spp. Ceftiofur As above + *Mannheimia haemolytica,* Penicillinase-producing *S. aureus*	fluid, lung, skin and soft tissues.	Ceftiofur sodium 1 d duration.Give dose 1mgkg once daily for 3d. If response inadequate, dose on D4 and D5 Ceftiofur hydrochloride 1 d duration. Give dose 1mgkg once daily for 3d. If response inadequate, dose on D4 and D5 Ceftiofur crystalline-free acid 7 d duration with asingle dose at 6.6 mg/kg		Ceftiofur hydrochloride subcutaneous or intramuscular Ceftiofur crystalline-free acid subcutaneous (base of ear)	High High
TETRACYCLINES **Oxytetracycline (OTC)**	Bacteriostatic – inhibition of bacterial protein synthesis by binding the 30S ribosomal subunit.	Broad spectrum, including the most common bacterial BRD pathogens including *Mycoplasma bovis*	Widely distributed & can cross lipid membranes resulting in high intracellular concentration	OTC in propylene glycol & OTC in povidone: 1 d at 3mg/kg or 2 d at 10mg/kg OTC in 2-pyrrolidone (LA): 3 d at 20mg/kg OTC dihydrate: 3 d at 20mg/kg	AUC/MIC (value dependent on target pathogen)	Intravenous or intramuscular Intramuscular Intramuscular Subcutaneous	Low

NOTES[16]

Export Slaughter Interval (ESI): the ESI is product specific and subject to change. Always check the current APVMA list of cattle ESIs at https://apvma.gov.au/node/26531

PK-PD (pharmacokinetic pharmacodynamic) parameters – effectiveness of each antibacterial agent is based on both the MIC (determined in vitro) and the pharmacokinetic or concentration-time profile determined in vivo. The combination of PK and PD components provides the effectiveness parameter. Generally, the higher the value of the PK PD parameter the greater the likelihood of effectiveness.

All cases of treatment failure should be investigated to identify the cause wherever possible. It is only if the cause is known that appropriate changes can be made to improve treatment success in future cases. Possible causes of treatment failure include delayed detection of BRD leading to disease progression; intercurrent disease; inappropriate antibiotic selection; incorrect dosage regimen; incorrect diagnosis; and antibacterial resistance.

Based on field efficacy, duration of therapeutic concentration, route of administration, dose volume, and ASTAG rating, and considering that most BRD cases occur early in the feeding period, the first-choice antibiotics for the treatment of BRD are the macrolides, or florfenicol.

1. Tulathromycin (100 mg/mL) at 2.5 mg/kg by subcutaneous injection high on the neck. Single dose only.
2. Tilmicosin phosphate (300 mg/mL) at 10 mg/kg by subcutaneous injection high on the neck. Single dose only.
3. Florfenicol (300 mg/mL) at 20 mg/kg by intramuscular injection on the side of the neck. A second dose should be given 48 hours later, or florfenicol (300 mg/mL) at 40 mg/kg by subcutaneous injection high on the neck. Single dose only.

The re-treatment regimen is guided by consideration of the ASTAG antibacterial importance rankings into low-, medium- and high-importance categories (follow-up treatment could involve the use of agents more important to human health), the feedlot budget, the WHP/ESI and the microorganisms anticipated to be present in disease of longer duration. Bacterial agents present in more chronic cases include *Trueperella pyogenes, Mycoplasma bovis,* and *Fusobacterium necrophorum.*

Typical re-treatment regimens include:

- Oxytetracycline (200 mg/mL) at 20 mg/kg by intramuscular and/or subcutaneous injection (dependent on product) on the side of the neck.
- Oxytetracycline (100 mg/mL) at 10 mg/kg by intramuscular injection on the side of the neck. A second dose should be given 48 hours later.

Where the projected sale date falls inside the expiry of the WHP/ESI's for the drugs of first choice listed above, a shorter WHP/ESI is associated with the following treatments:

- Ceftiofur crystalline free acid (200 mg/mL) at 6.6 mg/kg by subcutaneous injection in the posterior aspect of the base of the ear. Single dose only.
- Ceftiofur hydrochloride (50 mg/mL) at 1 mg/kg by intramuscular or subcutaneous injection on the side of the neck. A second and third dose should be given at 24-hour intervals.

Note that as there is a low prevalence of resistance to the first-choice antimicrobials in Australian isolates of BRD pathogens, a failure to respond to treatment is caused in most cases by recognition and treatment of the disease when the pathology is too advanced (i.e. late disease recognition). It follows that prevention of antimicrobial resistance and management of treatment cost dictates that additional antimicrobial treatments should not be given until the expiration of the duration of the therapeutic concentration of the first-choice antimicrobial. Sufficient time for response to the use of an effective antimicrobial should be allowed before treatment with an additional antimicrobial. For example, it is illogical to treat a BRD case with another antimicrobial for the seven days that tulathromycin provides therapeutic concentrations. The decision to provide additional antimicrobial therapy at the expiration of the first treatment course is typically determined by assessment of the resolution of clinical signs through change in clinical illness score. No improvement in clinical illness score, without deterioration to very advanced disease and moribund state, indicates the need for further treatment.

Prognosis

Generally fair to good for cases identified and treated early in the disease course and given an optimal recovery environment (low stocking rate, bedding in designated 'hospital' pens). For late detections and advanced disease, the prognosis is guarded to poor. It should be noted that many published accounts of response to therapy are based on a subjective case definition and clinical scoring criteria and therefore are likely to be influenced by a lower diagnostic specificity. The prognosis is further complicated by differences in the immunocompetence of individual animals and possible comorbidities such as concurrent ruminal acidosis.[17]

Other more objective approaches to prognostication examined have included biomarkers, however no consistently reliable biomarkers have been established.[18] Thoracic ultrasonography has been utilised with some predictive success if objective metrics of maximal depth and area of consolidation are determined.[19] While reduced average daily gain (ADG) is associated with feedlot cattle suffering episode(s) of BRD, attempting to utilise weight change as an indicator of treatment success in the initial, short, post first treatment interval is problematic on the basis of weight change possibly influenced by hydration status. Rectal temperature used as a one-time measurement, and not a continuous monitor, has little diagnostic and prognostic potential.

Prevention

The prevention of BRD is multifaceted and complex and has been reviewed at length elsewhere.[8, 20] Discussion of BRD prevention is beyond the scope of these guidelines and the reader is referred to these publications for an extensive review of the topic.

Tracheal Oedema

Body system/syndrome

Respiratory

Background/nature of infection/organisms involved

This condition often presents later in the feeding period, typically after the main incidence period for bovine respiratory disease (i.e. > 50 days post arrival). It is characterized by dyspnoea, loud coughing and inspiratory stridor, which is the basis for the colloquial name "honker". The pathology is principally oedematous thickening of the mucosa and submucosa of the dorsal trachea, ultimately leading to tracheal stenosis. There is an appreciable degree of exercise intolerance associated with this condition and it is common to see respiratory distress worsen with movement. Severe cases are at risk of sudden, anoxic death after forced movement, especially in hotter summer months.

The aetiology is currently uncertain. However, there is significant evidence for an association with a combination of viral and bacterial agents, together with hypersensitivity reactions to common feedlot allergens, such as dust and mycotoxins.

Differentiation from cases of infectious bovine rhinotracheitis (IBR) is difficult early in the course of both conditions. However, IBR cases tend to occur earlier in the feeding period and deteriorate more frequently and rapidly to death, with typical lesions at necropsy (necrotic tracheal mucosa).

Tests for diagnosis

Diagnosis is based on clinical presentation in the home pen. Differentiation from necrotic laryngitis may be possible in the hospital crush by palpating the larynx, applying gentle pressure, and determining if this exacerbates the stridor, which would indicate that necrotic laryngitis is the more likely diagnosis. Cases of necrotic laryngitis will have significant halitosis compared to early cases of tracheal oedema.

Key Issues

- Inflammation leads to swelling of the tracheal mucosa and stenosis, which can result in severe dyspnoea and risk of sudden death.
- Onset is usually sudden, as clinical signs become apparent and the airway narrows.
- Consideration needs to be given to whether the animal is fit for any transport.
- Medical management is the most realistic option in feedlot clinical settings and the response to therapy is typically mixed.
- Bacterial involvement is less critical in the pathogenesis, but treatment should be initiated if bacterial involvement is suspected.

Treatment

This is one of the more frustrating conditions to treat in a feedlot setting, as the inciting agents, such as viruses and allergens, are not easily avoided and can complicate recovery. Oedematous mucosal swelling and airway stenosis, associated with hypersensitivity, necessitates the need for short acting corticosteroids and possibly also the use of antihistamines. Antimicrobial therapy is indicated for secondary bacterial involvement. The strict anaerobe *Fusobacterium necrophorum* is a likely contributor to cases of necrotic laryngitis and, while it may also be involved in tracheal oedema, a broader spectrum of bacterial agents may be involved. Where antimicrobials are required, long-acting formulations are preferred to minimise the handling that is required for repeat treatments, which can aggravate the dyspnoea inherent to the condition. Notwithstanding this consideration, selection of formulations with a shorter WHP will facilitate exit from the feedlot for early (or salvage) slaughter if full resolution is not expected or realised, but fit-to-load requirements can still be met.

Antimicrobials used

The following rankings of appropriate antibiotic therapies consider spectrum, efficacy and ASTAG rating.

1. Oxytetracycline (100 mg/mL) at 10 mg/kg by intramuscular injection on the side of the neck, plus 30 mg dexamethasone sodium phosphate (6 ml of the 5 mg/ml product) by intravenous or intramuscular injection high on the neck (WHP 10 days, no ESI established). A second dose of antimicrobial and dexamethasone should be given 48 hours later.
2. Amoxicillin trihydrate (150 mg/mL) at 15 mg/kg by intramuscular injection on the side of the neck, plus 30 mg dexamethasone sodium phosphate by intravenous or intramuscular injection high on the neck. A second dose of antimicrobial and dexamethasone should be given 48 hours later.

   Where the projected sale date falls inside the date of expiry of the WHP/ESI's of the antimicrobials listed above, a shorter WHP/ESI is associated with the following treatments:

3. Ceftiofur crystalline free acid (200 mg/mL) at 6.6 mg/kg by subcutaneous injection in the posterior aspect of the base of the ear. Single dose only. A dose of 30 mg dexamethasone sodium phosphate should also be given by intravenous or intramuscular injection high on the neck.
4. Ceftiofur hydrochloride (50 mg/mL) at 1 mg/kg by intramuscular or subcutaneous injection on the side of the neck. A second and third dose should be given at 24-hour intervals. A dose of 30 mg dexamethasone sodium phosphate should also be given at the first and third treatments in this regimen.[21]

The re-treatment regimen is guided by consideration of the ASTAG antibacterial importance rankings into low-, medium- and high-importance categories (follow-up treatment could involve the use of agents more important to human health), the feedlot budget and the WHP. For all treatments subsequent to the first dose in these regimens, consideration must be given to whether the clinical presentation is unchanged (and therefore whether continued treatment is necessary) and whether the dyspnoea after movement, handling and restraint is manageable.

Typical re-treatment regimens include:

- Oxytetracycline (200 mg/mL) at 20 mg/kg by intramuscular and/or subcutaneous injection on the side of the neck.
- Oxytetracycline (100 mg/mL) at 10 mg/kg by intramuscular injection on the side of the neck. A second dose should be given 48 hours later.

Prognosis
Fair, if intervention is early in the disease process, but poor if there is significant dyspnoea and stridor on presentation.

Necrotic Laryngitis

Body system/syndrome
Respiratory

Background/nature of infection/organisms involved
Necrotic laryngitis presents similarly to tracheal oedema, with cases showing dyspnoea and inspiratory stridor. The aetiology is distinct from that of tracheal oedema, in that the primary lesion is ulceration of the laryngeal mucosa, secondary to inflammation from viral infection or possibly mechanical trauma. Subsequent colonization by and proliferation of the anaerobe *Fusobacterium necrophorum* (a commensal of the oral cavity and gastrointestinal tract) leads to further inflammation and ultimately necrosis of the mucosa and stenosis of the larynx. Alternatively, laryngeal abscesses may form after haematogenous delivery of *F. necrophorum*, which is capable of colonising various tissues once blood-borne.[22, 23] This aetiological pathway probably explains those lesions in which there is no ulceration or tissue damage on the epithelial surface.

Differentiation from infectious bovine rhinotracheitis (IBR) cases is difficult early in the course of both conditions, but IBR cases tend to deteriorate more frequently to rapid death and have typical lesions at necropsy (principally a necrotic tracheal mucosa).

These cases are often, but not necessarily, febrile and, as for tracheal oedema, clinical signs can worsen after movement or transport, especially in hotter summer months.

Tests for Diagnosis

Diagnosis is typically based on the clinical presentation. Diagnosis of necrotic laryngitis is supported by palpating the larynx, applying gentle pressure, and determining if this exacerbates the stridor. Cases of necrotic laryngitis may present with significant halitosis.

Key Issues

- Inflammation leads to swelling of the laryngeal mucosa and stenosis, which can result in severe dyspnoea and risk of rapid death.
- The onset is usually sudden as clinical signs become apparent and the airway narrows
- Medical management is the most realistic option in feedlot settings and the response to therapy can be variable.
- An unresolved, active lesion can make an animal unfit for transport.
- Residual swelling after treatment (particularly with a laryngeal abscess) can result in stridor until slaughter in animals that are otherwise apparently healthy.

Treatment

Fusobacterium necrophorum is sensitive to penicillin and should be the main bacterial agent suspected in clinical cases. The associated inflammation and stenosis of the larynx necessitate complementary therapy with short acting corticosteroids or non-steroidal anti-inflammatories (NSAIDs). Cases with severe dyspnoea and respiratory distress are likely to require tracheotomy, tracheostomy tube placement and supplemental oxygen, which is not a practical option in feedlots. These cases are usually best euthanased immediately.

Necrosis of the laryngeal cartilage can also occur as a sequela of initial ulceration and subsequent viral and/or bacterial infection, and irritation by dust and other allergens in the feedlot environment. This necrosis is typically slow to heal and requires more prolonged treatment and antimicrobial courses, which are often not practical in feedlots. Constant monitoring of the response to therapy should be employed, with early exit for salvage slaughter an important option to consider.

Antimicrobials used

For mild cases where movement and restraint pose a lower risk to the animal:

- Procaine penicillin (300 mg/mL) at 12 mg/kg by intramuscular injection on the side of the neck every 24 hours for 3-5 days, depending on the response to therapy.
  [N.B. The label dose rate for procaine penicillin is 12 mg/kg. However, the recommended effective dose rate is 22 mg/kg (22,000 IU/kg).[24, 25] Off-label use is problematic due to lack of established WHP and ESI, hence routine use of procaine penicillin is not recommended for feedlot cattle until an appropriate WHP and ESI are available for use at a dose rate likely to be effective in cattle].
- Ancillary anti-inflammatory therapy should be applied at the start and end of the treatment course with 30 mg dexamethasone sodium phosphate, or a single dose of the non-steroidal anti-inflammatory drug (NSAID) meloxicam at 0.5 mg/kg by subcutaneous or intravenous injection (WHP 8 days, ESI not established).

For moderate cases, after considering the potential adverse effects of movement and restraint for treatment, as well as fitness-to-load, the suitability of the case for early (or salvage) slaughter and the ASTAG ratings, treatment can be attempted with:

1. Oxytetracycline (200 mg/ml) at 20 mg/kg by subcutaneous injection on the side of the neck with ancillary therapy with a short acting corticosteroid (dexamethasone) or NSAID. If necessary, an additional dose of oxytetracycline can be given 72 h later.
2. Oxytetracycline (100 mg/mL) at 10 mg/kg by intramuscular injection on the side of the neck, plus a short acting corticosteroid or NSAID. A second dose should be given 48 hours later, accompanied by a second dose of corticosteroids, if the clinical presentation is unchanged and dyspnoea on movement, handling and restraint is manageable.
3. Ceftiofur crystalline free acid (200 mg/mL) at 6.6 mg/kg by subcutaneous injection in the posterior aspect of the base of the ear, single dose only, plus a single dose of the NSAID meloxicam at 0.5 mg/kg by subcutaneous or intravenous injection.

For all treatments subsequent to the first dose in these regimens, consideration must be given to assessment of whether there has been any change in the clinical presentation (and therefore whether continued treatment is necessary) and whether any dyspnoea on movement, handling and restraint is manageable.

Typical re-treatment regimens include:

- Oxytetracycline (200 mg/mL) at 20 mg/kg by intramuscular and/or subcutaneous injection on the side of the neck.
- Oxytetracycline (100 mg/mL) at 10 mg/kg by intramuscular injection on the side of the neck. A second dose should be given 48 hours later.

Prognosis

Fair if intervention is early in the disease process, but poor if there is significant dyspnoea and stridor on presentation.

ALIMENTARY SYSTEM

Actinobacillosis (wooden tongue)

Body system/syndrome

Alimentary

Background/nature of infection/organisms involved

The clinical presentation is usually firm swelling of the tongue, dysphagia, drooling, and varying degrees of tongue protrusion due to granulomatous (or pyogranulomatous) lesions. Normal commensal bacteria of the buccal cavity, specifically *Actinobacillus lignieresii*, gain entry to the submucosal layers of the tongue via lacerations secondary to abrasive, rough feedstuffs, establishing infections that progress to the typical pathology. The condition is not common in feedlots, given the provision of a processed ration. However, new feeder cattle can be placed in the feedlot with pre-existing lesions.

Tests for diagnosis

Actinobacillosis is generally diagnosed by clinical presentation and oral inspection and detection of the typical lesion(s). A pre-feedlot history of access to coarse, rough feedstuffs can support the presumptive diagnosis.

Key issues

- The diagnosis is best confirmed by oral examination using suitable restraint facilities.
- Differentiation from neurological disorders such as botulism is important – sporadic cases of actinobacillosis are significantly easier to manage and result in much lower economic or population impact than outbreaks of flaccid paralysis due to botulism.

Treatment

Concurrent antimicrobial treatment and sodium iodide generally improves the treatment outcome.

Antimicrobials used

- Oxytetracycline dihydrate (300 mg/mL) at 30 mg/kg by intramuscular injection on the side of the neck. Repeat in 7 days (and check for resolution of the lesions to determine whether subsequent treatments are required) or,
- Oxytetracycline (200 mg/mL) at 20 mg/kg by subcutaneous and/or deep intramuscular injection on the side of the neck. Repeat in 3 days (and check for resolution of the lesions to determine whether subsequent treatments are required).

combined with

- Sodium iodide (500 mg/mL) at 40 mL/300 kg, diluted in 60 mL water for Injection BP, by slow intravenous infusion or subcutaneous injection. Repeat at the time of the second dose of oxytetracycline (note this is an off-label treatment).[26, 27]

Prognosis

Response to therapy is generally good and there is usually full resolution if the severity is mild to moderate at the time of treatment.

Actinomycosis (lumpy jaw)

Body system / syndrome

Alimentary

Background/nature of infection/organisms involved

The clinical presentation is usually a localised mass due to proliferative osteomyelitis of the mandible or maxilla, with some cases showing diffuse ulceration of the overlying skin with a serous exudate ("honeycomb" appearance). Normal commensal bacteria of the buccal cavity, specifically *Actinomyces bovis*, gain entry to the submucosal layers of the oral mucosa via lacerations secondary to abrasive, rough feedstuffs, establishing infections that progress to the typical pathology. The condition is not common in feedlots given the provision of a processed ration. However, new feeder cattle can be placed in the feedlot with pre-existing lesions.

Tests for diagnosis

Actinomycosis is generally diagnosed based on the clinical presentation and detection of the typical lesion(s) of localised swelling and distension of the mandible or maxilla with advanced cases showing ulceration of the overlying skin and the development of draining fistulae. A history of access to coarse, rough feedstuffs can support the presumptive diagnosis.

Key issues

- Lesions are typically slow growing, non-painful and integrated with the bony tissue of the mandible or maxilla
- Immediate exit for slaughter can be considered instead of treatment because the slowly developing lesions are localised to the head, which is separated from the marketable carcase in the abattoir, provided there is no skin surface ulceration and/or discharge
- Lesions with extensive bony involvement are usually refractory to any treatments.

Treatment

Antimicrobial treatment is usually complicated by the issue of penetration into the bony mass, and, for full resolution of the lesion, surgical debridement would be necessary, but this is usually not practical in the feedlot operating environment. Concurrent sodium iodide and antimicrobial treatment, as for wooden tongue, is considered appropriate.

Antimicrobials used

Oxytetracycline has better bone tissue penetration than penicillin.

- Oxytetracycline dihydrate (300 mg/mL) at 30 mg/kg by intramuscular injection on the side of the neck. Repeat in 7 days (and check for resolution of the lesions to determine whether subsequent treatments are required), or
- Oxytetracycline (200 mg/mL) at 20 mg/kg by subcutaneous and/or deep intramuscular injection on the side of the neck. Repeat in 3 days (and check for resolution of the lesions to determine whether subsequent treatments are required), used concurrently with
- Sodium iodide (500 mg/mL) at 40 mL/300 kg, diluted in 60 mL water for Injection BP, by slow intravenous infusion or subcutaneous injection. Repeat at 10 days after the initial sodium iodide dose if required.[28]

Ruminal/Lactic Acidosis

Body system / syndrome

Alimentary

Background/nature of infection/organisms involved

Ingestion of excess readily fermentable carbohydrate results in the proliferation and increased fermentation output of all bacteria. This results in a decrease in pH occurring initially due to the production of a greater quantity of volatile fatty acids than can be absorbed by the rumen papillae per unit time.[29, 30] The pH drop to approximately 5.5 (with sub-acute ruminal acidosis defined as a rumen pH of less than 5.6 for greater than 3 hours a day, in the absence of clinical signs requiring individual animal treatment;[31] favours the growth of lactate producers, chiefly *Streptococcus bovis.* [32-34] *Streptococcus bovis* is a mixed acid fermenter (acetate, formate and ethanol from glucose), but can shift to homolactic fermentation if there is excessive substrate and the pH is less than 5.6.

Additionally, the activity of lactate dehydrogenase (LDH), which converts pyruvate to lactate, is enhanced at lower pH, with maximum activity at a pH of 5.5. As pH drops further due to the overwhelming of buffering systems, the production of lactate by *S. bovis* is reduced, but there is a concurrent rise in *Lactobacillus* spp. populations, which are acid tolerant.[35] These become the predominant species in the rumen at a pH of less than 5.6. As *S. bovis* initiates the fall in pH and the resulting acidosis, lactic acidosis prevention measures are often aimed at controlling the growth of this species.

In healthy ruminal microbe populations, lactate is eliminated by lactate-utilising bacteria and does not exceed concentrations of 5 mmol/L. Lactate (pKa=3.9) is approximately a 10 times stronger acid than acetate (pKa=4.9), the primary VFA found in the rumen, and therefore, accumulates in the rumen and decreases pH further. L-lactate is the predominant lactate isomer in the rumen and the proportion of D-lactate usually increases as pH decreases. Cattle do not produce D-lactate dehydrogenase, so D-lactate causes greater problems if absorbed, due to its slower metabolism.

The most important physiological counters to depression of rumen pH in cattle are the bicarbonate and phosphate buffers supplied to the rumen in saliva in response to mastication, which is governed by the particle size of the ingested feed, with increased particle size a greater stimulant of saliva production. Approximately half the bicarbonate in the rumen comes from saliva, while the other half is absorbed from the blood through the exchange of ionised acids. Bicarbonate is the primary buffer in the blood responsible for decreasing an acid load. However, an excessive rumen derived acid insult can overcome the buffering capacity of the blood. High concentrate diets are inherently lower in roughage. Therefore, feeding high concentrate diets reduces the contribution of bicarbonate from saliva, consequently increasing the amount absorbed from the blood, which then further reduces the buffering capacity of the blood. In summary, lactic acidosis occurs in response to the feeding of excess rumen-available readily fermentable carbohydrate and insufficient effective roughage to provide adequate salivary buffers to counter a pH drop that occurs in response to an initial excess production of volatile fatty acids. If rumen pH is low enough for long enough, overgrowth of *S. bovis* followed by an overgrowth of *Lactobacillus* spp. can occur. This transition accompanies the move from sub-acute ruminal acidosis to clinical ruminal acidosis, resulting in dehydration and toxaemia of sufficient severity to warrant individual animal treatment. There are a number of ways we can cause ingestion of an excess of rumen-available, readily fermentable carbohydrate accompanied by insufficient salivary buffering: too high a dietary cereal grain inclusion in a mixed ration fed to an unadapted rumen; provision of insufficient effective roughage; over-processed cereal grains in a mixed ration; and

engorgement of a mixed ration as a result of hunger. Grains vary in their potential to cause ruminal acidosis (from greatest to least: wheat, barley, sorghum, corn, oats).

We can adapt the rumen to cereal grain by: using an introductory diet including a low proportion of grain (< 50% as fed); increasing the grain inclusion for each diet in increments no greater than 10%; allowing sufficient time for the cattle to achieve stable feed intakes and target energy intakes on each diet without showing clinical signs of ruminal acidosis before moving to the next higher grain inclusion in the diet (this varies, but usually takes approximately 5 days).

Effective roughage is essential for the stimulation of salivation and the delivery to the rumen of salivary buffers, and it also cleans the papillae (important to the absorption of VFAs) and stimulates ruminal motility. Unfortunately, targets for the readily available laboratory assessments of fibre (neutral detergent fibre [NDF], and acid detergent fibre [ADF]) to maintain optimal fermentative efficiency, and therefore to achieve the highest possible growth rates in beef cattle, have not been defined.[36] The fibre requirement for optimal ruminal fermentation efficiency in beef cattle is also complicated by: the post-ruminal energetic efficiencies achieved when the rumen is flooded with small particles of starch, resulting in some passing intact to the abomasum and small intestine for direct digestion and absorption as glucose; and, the buffering effect of high rumen degradable protein, resulting in elevated rumen ammonia concentration. We do have some guidance with beef cattle fibre requirements if we consider that the molar proportion of propionate is maximised at a rumen pH of 5.6 to 6.2.[37] For beef cattle, the higher the propionate yield the more energetically efficient the rumen fermentation. Whilst more research is required to define NDF targets in beef cattle, currently we manage the effective roughage to maintain adequate delivery of salivary buffers for rumen health (i.e. prevent an unacceptable incidence of the clinical signs of ruminal acidosis) for a given diet and stage of ruminal adaptation to concentrates. The target length for effective roughage in mixed rations is 5 to 10 cm.

The appropriate degree of processing of cereal grains depends on the dietary inclusion of grain in the diet. As the grain inclusion in the diet is increased beyond 50%, we must reduce the degree of milling to prevent ruminal acidosis due to excessive availability of the starch. Thus, with high grain rations, cereal grains should only be bruised into an average of two fragments with dry rolling and tempered huskless grains, or squashed and crimped with tempered barley. With steam flaked grain we target processing indices (flake density/parent grain bulk density, kg/hL) of 58 to 60 for barley and wheat, approximately 50 for corn, and 47 for sorghum.[38] We must also consider the fermentation rate of the grains when we consider potential inclusions and milling. The faster the grain is fermented, the more it predisposes the cattle to ruminal acidosis, and therefore, the lower the safe grain inclusion is and the less aggressively the grain should be milled. Relative fermentation rates of the cereal grains from fastest to slowest are: wheat, triticale, barley, oats, sorghum, and corn.

Once a grain-based diet has been provided to cattle and they have learned to eat it, we can create a relative excess of readily fermentable carbohydrate simply by allowing the grain diet to run out and then suddenly providing unlimited access. The resultant engorgement can cause ruminal acidosis, even if the milling of the grain and roughage is within the target specifications, and is more severe the hungrier the cattle are.

It has been suggested that the early feedlot feeding period, during adaptation of the rumen to a diet high in starch, is the period of greatest risk of ruminal acidosis.[39] However, ruminal acidosis can occur at any time during the feeding period and might be more likely during the finishing phase, when consumption is high[40, 41] found a slight downward trend in rumen pH for the duration of a 148-day feeding period (r^2 = 19% for pH versus days on feed; $P < 0.0001$), suggesting that feedlot cattle are potentially susceptible to ruminal acidosis at any time during the feeding period, not only during the adaptation period.

Pathogenesis

Elevated concentrations of glucose and VFAs in the rumen increase rumen fluid osmolality, which exacerbates the increasing acidity of the rumen by inhibiting VFA absorption. As ruminal fluid osmolality increases further, it can exceed that of blood (285 to 310 mOsm/L), resulting in the movement of water into the rumen. This water flux through the rumen wall causes swelling of the rumen papillae and damage to the rumen wall itself. In combination with the superficial chemical rumentitis caused by the acidic rumen contents, toxaemia and bacteraemia, leading to the seeding of infections in various organs, including the lungs, can result.[42] Repair of the damaged rumen wall can lead to parakeratosis, which then inhibits the rate of VFA absorption in animals that have recovered and results in suboptimal performance. In a very small proportion of cases, secondary fungal rumenitis can occur. Cattle suffering from secondary parakeratosis or fungal rumenitis show unresponsive ill thrift and are best culled.

The toxaemia that occurs due to the compromised rumen wall is exacerbated by the dehydration that occurs due to the osmotic pressure exerted by the acidic rumen, which draws water out of the blood stream. Thus, in acute cases, the animal dies from a combination of dehydration, acidaemia, and toxaemia. Initially, rumenitis induced by mild ruminal acidosis increases the rumen outflow rate. This reaction, combined with the increased passage of fermentable carbohydrate out of the carbohydrate flooded rumen, is expressed as the clinical sign of bubbly diarrhoea. The bubbles in the faeces are evidence of fermentation that continues even beyond the hindgut, and the incidence of bubbly scours is used as a proxy for the incidence of ruminal acidosis in intensive feeding systems. At the individual animal level, the occurrence of bubbly diarrhoea is accompanied by inappetence, and if the incidence of this clinical sign of ruminal acidosis is high enough, depressed feed intake can occur at the pen level.

Other sequelae include polioencephalomalacia, laminitis and bloat. Polioencephalomalacia is an induced thiamine deficiency arising from the release of thiaminase from acid-shocked bacteria (such as *Bacillus thiaminolyticus*), and/or the destruction of thiamine by sulphites associated with high dietary sulphur intake, exacerbated by a lower ruminal pH. Laminitis occurs either due to the local effects of endotoxins in the sluggish capillary beds of the hoof laminae or the effects of increased circulating blood concentrations of histamine absorbed through the inflamed rumen wall. Feedlot bloat does not involve chloroplast proteins, although the presence of these could logically exacerbate the condition. In feedlot bloat, the slimy capsule of *S. bovis* contributes to the formation of a stable foam that prevents the expulsion of gas in eructation. Further, the viscosity of the ruminal contents is greater at lower pH.

Anecdotally, it appears that feedlot bloat is seen more commonly when feed delivery and intake are erratic, and it is perhaps a more common expression of disease resulting from engorgement. Logically, there are variations in the severity of ruminal acidosis and therefore variation in the extent of pathological changes and production effects.

The perception that there is a positive relationship between clinical signs of ruminal acidosis and greater production is not only contrary to a high standard of animal welfare, it has also been shown to be incorrect.[41] Cusack and colleagues[41] found that, whilst there was a positive relationship between lower ruminal pH and greater growth rate in cattle that had a ruminal pH < 5.6 for < 3 h/d, this relationship disappeared in those that had a ruminal pH < 5.6 for > 3 h/d (= sub-acute ruminal acidosis). Therefore, inducing even sub-acute ruminal acidosis does not improve production, and there is logically a ruminal pH depression value more marked than sub-acute ruminal acidosis, where the lack of a relationship between ruminal pH and production becomes a negative effect.

Tests for diagnosis

Where a mob or pen has a ruminal acidosis problem there will be a range in the severity of the clinical signs depending on feed intake and feeding activity over the previous few days.

- Greyish watery diarrhoea, often with bubbles, sometimes containing undigested grain particles, depending on disease progression
- Inappetence
- Loss of ruminal stratification (sometimes with sloshing rumen sounds when affected cattle are moved)
- Altered ruminal fluid characteristics
    - Depressed ruminal pH (< 5.5)
    - Reduced sedimentation time (< 4 minutes)
    - Prolonged methylene blue reduction (decolourisation) time (> 6 minutes)
    - Loss of protozoa (seen at 10 to 40 x magnification)
    - Predominance of Gram +ve cocci and rods on a Gram-stained smear
- Elevated serum D-lactate
- Lethargy and depression
- Dehydration
- Recumbency and death, which can occur within 12 to 24 hours.

Key issues

Any cattle showing clinical signs of ruminal acidosis, such as watery, bubbly diarrhoea, will have a ruminal pH that is below the range associated with maximum propionate and total VFA production .[37] This will reduce the growth rate of that individual animal. Further, more severe clinical signs of ruminal acidosis, where cattle are obviously depressed, lethargic and dehydrated, constitute poor animal welfare. In addition, the incidence of ruminal acidosis is associated with a greater incidence of bovine respiratory disease (BRD)[43, 44]; and greater severity in the cases of BRD that occur,[43] and the attendant poor animal welfare that accompanies the occurrence of BRD. We therefore have an obligation, in terms maximising production for our clients and maximising the welfare of the cattle for which we are responsible, to prevent ruminal acidosis.

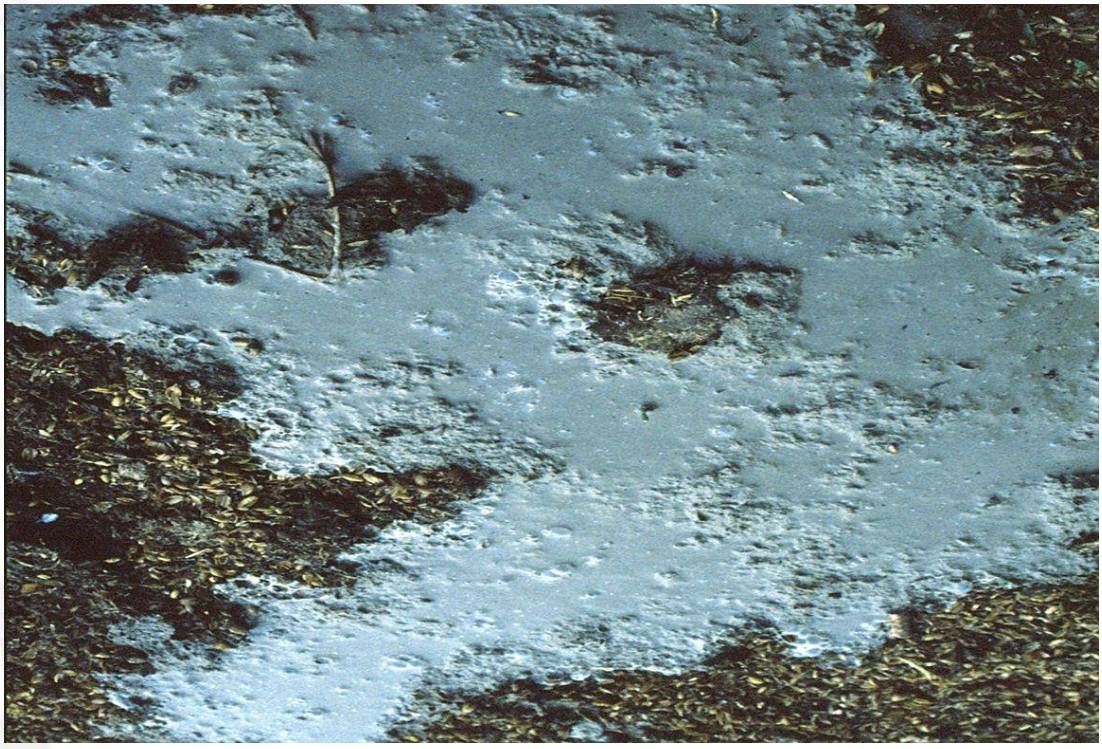

Image: watery, bubbly diarrhoea typical of ruminal acidosis (Source: P Cusack).

Treatment

Treatment of individual clinical cases represents a failure of feed management. It is based on correction of the acidity in the rumen and parenteral antibiotics to prevent secondary liver abscessation. Affected cattle are drenched with 500 g $NaHCO_3$, 500 g MgO and 500 ml paraffin all mixed into 5 to 8 L of water. Procaine penicillin is given intramuscularly once per day for 3 days to reduce the risk of organ abscessation.

Valuable cattle can also benefit from the administration of non-steroidal anti-inflammatory drugs at labelled dose rates,[45] but the most effective therapy for high value cattle is rumenotomy with lavage and transfaunation and intravenous fluids, including $NaHCO_3$, to address the acidaemia. Heart rate is a useful prognostic indicator: < 120 bpm = fair; 120 to 150 bpm = guarded; > 150 bpm = grave.

Mob or pen ruminal acidosis problems are assessed on the basis of the incidence of bubbly diarrhoea. If the incidence of bubbly diarrhoea exceeds 3% on a pen check (i.e. more than 3 bubbly scours observed on the pen floor per 100 head), this alerts us to check the milling and delivery of the ration. In the face of a more widespread and severe problem, with an incidence of bubbly scours of 10% or more, additional roughage should be supplied. With a mixed ration this will involve dropping the grain inclusion and raising the roughage inclusion to those of the previous ration. The higher roughage intake is maintained until the faecal scores of the group return to normal limits (≤ 3% bubbly scours).

Prevention

The prevention of ruminal acidosis is based on maintaining a stable ruminal microbial ecosystem and allowing sufficient time for rumen papillae development to match the VFA production from nutrient dense feeds during the adaptation period. Based on the aetiology of ruminal acidosis, the following preventative strategies should be employed:

- Filling the rumens of newly received cattle with effective roughage only for the first 24 to 48 hours if they are empty or full of lush forage.
- Gradual adaptation of the ruminal microbial ecosystem and development of the rumen papillae by introducing cereal grains gradually (i.e. starter diets with no more than 50% grain and increases in grain inclusion of no more than 10% with each subsequent diet up to the final diet).
- Disciplined feed deliveries to each pen based on:
    - the feed residual target of a scattering of feed particles in the bunk (not licked shiny but with the bunk floor exposed) at the time of feed delivery
    - the cattle behaviour target of 70% of the animals at the bunk or on the apron and 30% in the remainder of the pen at the time of feed delivery
    - feed allocation increases of no more than 10% each day and on no more than two consecutive days.
- Provision of adequate effective roughage (5 to 10 cm in length).
- Appropriate milling of grains to maximise utilisation, but also prevent excessive starch surface area, which presents an excess of rapidly fermentable carbohydrate for ruminal microbes. This will depend on the grain inclusion in the diet, the grain type, and the processing method.

Antimicrobials used

- Therapeutic agents (ionophores or antibiotics) that inhibit lactate producers (e.g. the ionophores monensin, salinomycin or lasalocid, narasin or the antibiotic virginiamycin). These agents are much more effective than buffers such as $NaHCO_3$.The cheapest source of $NaHCO_3$ is saliva from the provision of effective roughage, which provides approximately 10 times the amount provided in a mixed diet at a 1% inclusion.
- Note that ionophores have no use in human medicine and there is no evidence of the development of cross-resistance to antibiotics in cattle where they are fed. In addition to reducing the risk of ruminal acidosis and its sequelae, ionophores reduce methanogenesis, have modest effects on increasing the proportion of undegradable dietary protein, and reduce the metabolic requirements of the intestine, thereby increasing growth rate and improving feed conversion efficiency. Ionophores are routinely included in feedlot diets for their established benefits.[46-49]
- By contrast with the ionophores, virginiamycin, a streptogramin class antibiotic, is rated by the Australian Strategic and Technical Advisory Group (ASTAG) on AMR as highly important for human use (Australian Government, 2018).[2] Therefore, the selection and use of virginiamycin should always be undertaken only after all other measures have been considered and, wherever possible, implemented. If there is no alternative, then

virginiamycin treatment may be implemented, but only for the shortest duration that is consistent with achieving acceptable animal welfare and health.

- With appropriately formulated, milled, mixed, and delivered diets, the inclusion of virginiamycin is not necessary for the prevention of ruminal acidosis. Circumstances such as prolonged drought or flooding can limit access to roughage. Under these extraordinary circumstances, where feedstuff availability dictates the formulation of high grain diets, particularly high wheat diets, and where all the aforementioned feed management practices have been implemented, and an ionophore is being fed, if clinical signs of ruminal acidosis are still occurring, there is an animal welfare requirement to take the additional step of including virginiamycin at the labelled dose rate to prevent illness and deaths from ruminal acidosis. This antibiotic must not be used as a substitute for sound feed milling and delivery management. As ruminal pH depression can occur at any time during the feeding period[41] it may be necessary to include these agents in the feed until such time as a change in the grain inclusion and/or the type of grain makes the diet less likely to cause ruminal acidosis. A reversion to the use of diets that do not predispose cattle to ruminal acidosis and therefore do not require the inclusion of virginiamycin to prevent ruminal acidosis must occur at the earliest opportunity. The use of virginiamycin is therefore limited to these extraordinary circumstances that prevent the formulation of diets compatible with the maintenance of rumen health.

Bloat

Body system / syndrome

Alimentary

Pathogenesis

Bloat is a metabolic syndrome caused by an excess of gas produced in the rumen compared to gas released via normal eructation. The imbalance can be caused by physical impairment of eructation, entrapment of gas in foam or slime, and/or rapid fermentation of grain due to variable feed intake, ration changes, feed mixing errors, or the grain particle size being too fine.

In the feedlot, bloat is more commonly observed later in the feeding period when cattle are being maintained on the highest intakes of rations with a high proportion of readily fermentable carbohydrates. Interruptions to feeding operations, or feeding errors, at this stage in the feeding period run a higher risk of producing bloat. Sporadic cases of bloat may occur if eructation is restricted due to recumbency, such as in cases of feeder cattle who are physically cast. Bloat cases occurring earlier in the feeding period can be associated with other co-morbidities such as ruminal acidosis and secondary decrease in rumen motility/ eructation. Vagal nerve damage and indigestion can be associated with chronic pneumonia, pleuritis or oesophageal obstructions or lesions.

Accumulated gas from fermentation distends the rumen, with the left paralumbar fossa more obviously affected, which greatly increases intra-abdominal pressure. Consequently, the thorax becomes compressed while caudal circulation is impeded, resulting in dyspnea. Without timely treatment, death by asphyxiation is common.

Prevention

Preventative management of bloat involves providing a well-formulated diet with consistent feed calling, mixing, and delivery. Grain to roughage ratio is an important consideration, as well as the coarseness and degree of processing of the grain and roughage. Some supplements, such as

ionophores, can also reduce the risk of bloat through regulation of feed intake and alteration of the rumen microenvironment.

Treatment

For treatment, the main aim is to relieve the accumulated rumen gas and avoid asphyxiation. In many cases, insertion of a stomach tube is sufficient, if animals can be moved to and handled in a facility to allow this. If the animal is recumbent or at immediate risk of asphyxiation, emergency rumen trocarisation can be a necessary lifesaving procedure. Animals who repeatedly bloat (possibly due to vagal indigestion) can be treated surgically by performing a rumenostomy.

Enteritis - scours

Body system / syndrome

Alimentary

Background/nature of infection/organisms involved

Typically, in the feedlot clinical setting, cattle present with acute diarrhoea, which may be seen sporadically or involve a larger cohort in a single pen. Differential diagnoses include gastrointestinal parasitism, salmonellosis, yersiniosis, acute bovine viral diarrhoea virus infection, ruminal acidosis, monensin toxicity or other toxaemias. Conditions such as ruminal acidosis and monensin toxicity have the potential to present in multiple pens. Cases of chronic diarrhoea are generally confined to compromised hospital feeder cattle and the causes include Johne's Disease and permanent gastrointestinal tract damage due to prior severe parasitism.

Where acute enteritis with diarrhoea is due to salmonellosis, serovars Typhimurium and Enteritidis are usually implicated. Because of the older age and greater immunocompetence of most cattle placed in feedlots, cases tend to be sporadic. However, as with sheep, inanition is a major predisposing factor, and substantial losses (mortalities of around 5%) have been seen in cattle ready for slaughter that were without feed for 24 h as a result of transport and diversion to another feedlot due to a breakdown, and then placed on a feedlot ration until their delayed slaughter could be rescheduled. Presenting signs can range from asymptomatic (and development of a carrier state) to mild to severe diarrhoea, with marked pyrexia, depression and dehydration. Faecal material in severe cases is usually fetid and can be accompanied by (fresh) blood and shreds of necrotic mucosa, or profuse, watery, and a bright green-yellow colour. The route of transmission is faeco-oral, which can be enhanced by intensive production systems, but infection is also influenced by predisposing factors, such as individual immune status, concurrent disease (e.g. BVDV infection and/or parasite burden), and gastrointestinal tract compromise arising from transition to grain feeding.

Yersinia pseudotuberculosis infection can also cause acute enteritis with diarrhoea (yersiniosis). The risk factors are similar to those for salmonellosis and cases tend to be sporadic, but diarrhoea is usually more chronic than acute, and is associated with weight loss.

Tests for diagnosis

A definitive diagnosis is obtained by laboratory analysis of live and necropsied animals. Supportive tests include:

1. Faecal culture in live animals.
2. Necropsy, revealing haemorrhagic enteritis, necrosis of the large intestinal mucosa, and malodorous faeces containing mucous and/or blood.

3. Culture of tissues collected at necropsy: jejunum, ileum, colon and mesenteric lymph nodes.
4. Histopathological examination of jejunum, ileum, colon and mesenteric lymph nodes collected at necropsy.

Key issues

- Stressors, such as pre-existing GI parasitism or other disease, as well as transition to the feedlot environment, predispose to disease.
- Cases tend to be sporadic, except for mobs with inanition, in which substantial losses can occur.
- The yearling and older cattle that are typically placed in feedlots will often have a self-limiting version of (infectious) enteritis which does not require treatment.

Treatment

Improvements in husbandry are important to optimising outcomes. Isolating clinical cases from other cattle, especially compromised hospitalised animals, in dedicated isolation pens will limit further transmission. This has the dual benefit of reducing competition for these animals to allow adequate hydration during resolution of the clinical signs. At the time of presentation in the hospital crush, correction of dehydration with oral fluid therapy is useful as supportive therapy for moderate to severe cases. Hydration status can be assessed and corrected each time they are in the hospital crush. *Salmonella* spp. have variable antimicrobial resistance, so antimicrobial selection should be informed by culture and susceptibility testing.

For animals that are inappetent, administration of a vitamin B-complex preparation may act as an appetite stimulant.

Antimicrobials used

- Trimethoprim (80 mg/mL) at 4 mg/kg and sulfadiazine (400 mg/mL) at 20 mg/kg by intramuscular injection on the side of the neck. Daily doses should be given until clinical signs have resolved.
- Oxytetracycline (100 mg/mL) at 10 mg/kg by intramuscular injection on the side of the neck. A second dose should be given 48 hours later.

Prognosis

Fortunately, strains of *Salmonella* with multidrug resistance are currently much less common in Australia than in other countries. Therefore, treatment with appropriate antimicrobials early in the course of disease is usually sufficient to resolve clinical signs in yearling and feeder cattle.

CARDIOVASCULAR SYSTEM

Traumatic reticulopericarditis

Endocarditis

- See AVA-AMA Antimicrobial prescribing guidelines for dairy cattle. https://onlinelibrary.wiley.com/doi/10.1111/avj.13311 [50]

Image source: MLA Image Gallery: https://www.mla.com.au/news-and-events/media-hub/image-gallery/feedlot/

MUSCULOSKELETAL SYSTEM

Interdigital necrobacillosis – footrot

Body system / syndrome

Musculoskeletal

Background/nature of infection/organisms involved

Interdigital necrobacillosis usually presents acutely in feedlots and is one of the principal causes of lameness across the industry. The bacteria responsible are most commonly *Fusobacterium necrophorum,* and less commonly *Dichelobacter nodosus, Prevotella* spp. and *Porphyromonas spp.* Predisposing factors include any trauma of the interdigital skin, which may occur during transport and handling during placement in the feedlot, and wet/muddy/deep faecal litter pen surfaces harbouring *F. necrophorum* and other anaerobic bacteria. *F. necrophorum* is a normal commensal of the gastrointestinal tract.

Cellulitis of the digital area commonly accompanies infection and is prominent at the coronary band. As disease progresses, the interdigital lesion swells further, spreading the digits wider, and possibly resulting in the integument separating and cracking. Severe infections present with significant lameness, heat in the region, fetid exudate from the interdigital fissure(s) and sloughing of necrotic tissue from the interdigital space. Extension of a severe lesion to deeper structures, such as the interphalangeal joint and/or flexor tendons, can result in joint sepsis and tenosynovitis.

Tests for diagnosis

Diagnosis is typically based on assessment of the presenting clinical signs. More robust confirmation of the diagnosis can be achieved by close inspection of the digit using suitable restraint apparatus, such as a tilt table, vertical crush or leg rope.

Key issues

- Wet, muddy conditions, with significant faecal contamination, increases the risk of disease, particularly if feeder cattle spend prolonged periods with their feet exposed to such conditions.
- Trauma to the interdigital space, by gravel in lanes and pens, and/or jagged points in handling facilities, will also increase the risk of disease.

Treatment

Treatment consists of systemic antimicrobials, possibly supplemented by topical therapy using foot baths (e.g. 5% copper sulfate solution), particularly for large outbreaks affecting larger cohorts in residential (home) pens. In cases that are presented in appropriate restraint apparatus, regional intravenous infusion can also be considered for severe cases that risk extending to joint sepsis or tenosynovitis (David Anderson, *pers. comm*.)

Antimicrobials used

- Procaine penicillin (300 mg/mL) at 12 mg/kg by intramuscular injection on the side of the neck every 24 hours for 3-5 days, depending on the response to therapy.
  [N.B. The label dose rate for procaine penicillin is 12 mg/kg. However, the recommended effective dose rate is 22 mg/kg (22,000 IU/kg)[24, 25]. Off-label use is problematic due to lack of established WHP and ESI, hence routine use of procaine penicillin is not

recommended for feedlot cattle until an appropriate WHP and ESI are available for use at a dose rate likely to be effective in cattle].

- Oxytetracycline (100 mg/mL) at 10 mg/kg by intramuscular injection on the side of the neck. A second dose should be given 48 hours later.
- Combination of 1 ml of procaine penicillin (150 mg/ml), benzathine penicillin (150 mg/ml) and procaine hydrochloride (20 mg/ml) at 1 ml/25 kg as a single intramuscular injection on the side of the neck.
- Oxytetracycline (200 mg/mL) at 20 mg/kg by intramuscular and/or subcutaneous injection on the side of the neck. A second dose should be given 3 days later.

Prognosis

If intervention with systemic antimicrobials is instituted early, the prognosis is generally very good. Multiple cases in single pens may necessitate group foot bathing, if the infrastructure is available, to manage the mild cases and mitigate the requirement for managing large numbers of cattle in the hospital facility and use of injectable antimicrobials. Routine pen cleaning, and possibly bedding, should be part of the preventative programme, as well as transfer to dry ground where feasible. Unresponsive cases with extension into the deeper sub-dermal tissues, including the flexor tendon sheath and distal digital joints, inevitably require salvage procedures, such as claw amputation, to arrest the disease.

Digital dermatitis (Hairy heel warts)

- See AVA-AMA Antimicrobial prescribing guidelines for dairy cattle: https://onlinelibrary.wiley.com/doi/10.1111/avj.13311 [50]

Septic arthritis

Body system / syndrome

Musculoskeletal

Background/nature of infection/organisms involved

Septic arthritis develops in feedlot cattle after haematogenous spread, typically from primary respiratory tract infections, or arises from traumatic wounds that penetrate the joint capsule. Primary respiratory tract infections involving *Histophilus somni*, *Trueperella pyogenes* and *Mycoplasma bovis* are commonly associated with joint infection sequelae after haematogenous spread. *T. pyogenes* and *Fusobacterium necrophorum* can reach the joints haematogenously as a result of bacteraemia secondary to ruminal acidosis.[42]

Tests for diagnosis

The clinical presentation is readily recognizable, with profound lameness to the level of non-weight bearing in one or more limbs. The affected joint, typically the stifle, hock, carpus, or elbow, is acutely swollen, and swelling is limited to the affected joint. On palpation and examination, there is acute pain and a reduced range of motion on joint flexion. A fluid-filled effusion and heat may also be detectable.

Confirmation of the diagnosis is possible by laboratory analysis of a joint fluid aspirate. an aspirate with a total protein concentration of greater than 45 g/L, a polymorphonuclear (PMN) cell count greater than 20,000 cells/mL and/or with more than 80% of the cells polymorphonuclear is consistent with septic arthritis. Bacterial isolation and/or cytological visualisation further confirms

the diagnosis, but a failure to detect bacteria on cytology does not exclude the diagnosis. The clinical history and fluid analysis is considered the basis of a definitive diagnosis.[51]

Key issues

- In feedlot production systems septic arthritis is associated with significant cartilage destruction and joint pathology and treatment is unrewarding. These animals do not qualify as fit-to-load for transport and slaughter. Typically, for the only appropriate approach is immediate euthanasia
- An increasing incidence, compared to the usually infrequent and sporadic incidence of cases, can indicate a substandard therapeutic response and poor management of BRD. In this scenario, a rising number of chronic cases, associated with treatment failure, and with a history of multiple treatments, begin filling hospital treatment pens. In such situations, all aspects of BRD management require review. An elevated incidence can also occur when clinical ruminal acidosis is observed and merits closer attention to feed management practices to prevent ruminal acidosis.

Treatment

Antimicrobial treatment can be guided by the laboratory analysis of the joint aspirate, but this is rarely practiced because of the lack of suitable infrastructure for treatment and the poor prognosis. In addition, for any hope of resolution to fitness-to-load, an extended course of antimicrobial therapy would be required, which is also not feasible in the feedlot setting and could result in residues in the finished carcase.

Treatment of septic arthritis should only be considered if regional limb perfusion can be performed, for a minimum of 20 minutes, and for two separate sessions approximately 48 hours apart. This requires a safe restraining tilt table that allows access to the diseased joint. Often, cases are advanced at presentation, complicating the process of joint lavage because of accumulations of fibrin obstructing lavage flow through the joint, necessitating arthrotomy to successfully irrigate the joint. Supportive therapy after lavage and/or arthrotomy includes anti-inflammatory treatment and sterile bandaging.

Antimicrobials used

Systemic antimicrobials that can be considered for treatment of joint infections are same as for bovine respiratory disease.

1. Tulathromycin (100 mg/mL) at 2.5 mg/kg by subcutaneous injection high on the neck. Single dose only.
2. Tilmicosin phosphate (300 mg/mL) at 10 mg/kg by subcutaneous injection high on the neck. Single dose only.
3. Florfenicol (300 mg/mL) at 20 mg/kg by intramuscular injection on the side of the neck. A second dose should be given 48 hours later, or florfenicol (300 mg/mL) at 40 mg/kg by subcutaneous injection high on the neck. Single dose only.

The re-treatment regimen is guided by consideration of the ASTAG antibacterial importance rankings into low-, medium- and high-importance categories (follow-up treatment could involve the use of agents more important to human health),, the feedlot budget, the WHP and the microorganisms expected to be present in infections of longer duration.

Typical re-treatment regimens include:

- Oxytetracycline (200 mg/mL) at 20 mg/kg by intramuscular and/or subcutaneous injection on the side of the neck.
- Oxytetracycline (100 mg/mL) at 10 mg/kg by intramuscular injection on the side of the neck. A second dose should be given 48 hours later.

Where the projected date of sale makes a short WHP/ESI necessary,

- Ceftiofur crystalline free acid (200 mg/mL) at 6.6 mg/kg by subcutaneous injection in the posterior aspect of the base of the ear. Single dose only.
- Ceftiofur hydrochloride (50 mg/mL) at 1 mg/kg by intramuscular or subcutaneous injection on the side of the neck. A second and third dose should be given at 24 h intervals.
- Antimicrobials that can be used for intra-articular infusion include the following:
  - Up to 500 mg ceftiofur sodium.
  - Up to 1 x 10^6 units of crystalline penicillin.

Prognosis

The prognosis is generally poor. In the feedlot setting, immediate euthanasia is more appropriate than therapy.

Image source: MLA Image Gallery: https://www.mla.com.au/news-and-events/media-hub/image-gallery/feedlot/

NEUROLOGICAL SYSTEM

Thromboembolic Meningoencephalitis (TEME)

Body system/syndrome
Neurological

Background/nature of infection/organisms involved
Thromboembolic meningoencephalitis (TEME) is an uncommon, sporadic condition. Its prevalence is lower than that of polioencephalomalacia (PEM) in the feedlot setting. The disease is characterized by acute onset and rapid progression of neurological signs from ataxia to recumbency and seizures to coma. Once neurological signs are apparent, there is a very high mortality rate. *Histophilus somni* is the principal bacterial agent involved and results from haematogenous dissemination from a primary respiratory tract infection.

Tests for diagnosis
TEME can realistically only be diagnosed definitively at necropsy after submission of brain and cerebrospinal fluid for histopathology and bacterial culture. The presenting clinical signs, if observed, are suggestive and can be differentiated from PEM by the more rapid progression of signs and lack of “star gazing”, which is more common in PEM, as well as a marked initial pyrexia, which is usually absent with PEM.

Key issues
- Low incidence, but high mortality rate.
- Often associated with a higher prevalence of BRD across the feedlot.
- Rapid progression of neurological signs, which is often not observed. More typically animals are discovered as sudden deaths in home pens and TEME is diagnosed subsequently at necropsy.

Treatment
Histophilus somni is sensitive to a wide range of antimicrobials used for respiratory disease in feedlots, including tulathromycin, florfenicol, penicillin, amoxicillin, oxytetracycline and cephalosporins, and treatment can be successful if cases are identified very early in the disease process, whilst still bacteraemic and pyrexic. More widely distributed antibiotics capable of crossing lipid barriers, such as oxytetracycline and florfenicol, are preferred to enhance penetration across the blood-brain barrier. These are given at the dose rates listed for BRD. However, treatment is frequently not possible because the neurological signs can prevent movement to a crush, and at this advanced stage cases usually fail to respond to treatment.

Prognosis
Poor.

(Cervical) Vertebral body abscess

Body system/syndrome

Neurological

Background/nature of infection/organisms involved

Abscesses in the vertebral body may occur secondary to bacteraemia and sepsis from primary respiratory tract infections or ruminal wall compromise as a result of ruminal acidosis. They may also occur as a result of inappropriate needle placement when medications are administered in the neck, the standard site for injection in feedlot cattle. *Trueperella pyogene*s[52, 53] and *Fusobacterium necrophorum*[54] have frequently been isolated from vertebral body abscesses, indicative of haematogenous spread from chronic respiratory tract infections or ruminal wall compromise.

Tests for diagnosis

If localisation is in the cervical vertebra(e), cattle can present with neck stiffness, hind limb ataxia and possibly some degree of forelimb paralysis. Invariably there is also reduced locomotion in the home pen, reduced appetite, unwillingness to groom and depression from the bacteraemia/toxaemia and neck pain.

A definitive diagnosis requires radiography and aspiration of the bony lesion for bacterial culture, along with histopathology. Haematology and blood biochemistry may be suggestive of a chronic inflammatory response.

Key issues

- A condition very rarely diagnosed ante- or post-mortem, but some calculable risk exists given the prevalence of respiratory disease.
- Treatment would be directed only on the basis of presumptive diagnosis and history given the lack of infrastructure and resources to perform definitive diagnosis.

Treatment

Treatment, if attempted, would need to be in combination with drainage of the abscess and surgical debridement of diseased bone, while maintaining structural integrity of the vertebral column. Even then, prognosis is poor. Euthanasia is a more appropriate intervention and enables a necropsy to be performed for definitive diagnosis.

Antimicrobials used

- Procaine penicillin (300 mg/mL) at 20 mg/kg every 24 hours for 14 days. Note this is an **off-label** regimen.[24]
- Amoxicillin trihydrate (150 mg/mL), with a loading dose of 20 mg/kg, followed by 10 mg/kg intramuscularly every 12 hours for 10 days. Note this is an **off-label** regimen.
- Oxytetracycline (100 mg/mL) at 10 mg/kg every 24 hours for 14 days. Note this is an **off-label** regimen.

Prognosis

Poor. Euthanasia is usually the best option.

Listeriosis

Body system/syndrome

Neurological

Background/nature of infection/organisms involved

Listeria monocytogenes can infect the central nervous system by ascending the trigeminal nerve, via a breach in the oral mucosa, and cause meningoencephalitis in cattle. The most common presentation is localized facial paralysis with a head tilt towards the affected (ipsilateral) side. Sometimes the head tilt is combined with circling and 'star gazing'. The disease is usually sudden in onset, but the incubation period is usually prolonged - over several weeks. The disease is sporadic, typically affecting only individual animals and principally adult cattle.

Listeria species are ubiquitous in soil and faeces and can be found in high numbers in spoiled straw and silage. It can persist for months to years in the environment.

Tests for diagnosis

The most useful diagnostic aid for listeriosis in feedlots is a thorough neurological examination in a home pen/laneway (allowing observation at rest and in motion), followed by restraint for a closer neurological examination. The neurological deficits are most commonly affected seen in regions supplied by the trigeminal, facial, vestibulocochlear and glossopharyngeal nerves, with lesions located at the proximal end of these cranial nerves, with the potential for extension into the brainstem, cerebellum and possibly cerebrum. Trigeminal nerve damage leading to unilateral loss of control of mastication results in accumulation of unmasticated feed material on the affected side. On physical examination, the jaw on the affected side may be dropped or easily opened. Infection and damage to the facial nerve results in unilateral facial paralysis, characterised by drooping of the ear and ptosis and a flaccid lip. This is the more common feedlot presentation. Vestibular nerve infection can lead to ataxia, a head tilt and circling, with cattle sometimes found with their head forced through a gate, or in between a bunk rail and feed bunk. Glossopharyngeal nerve damage mainly results in more severe dysphagia than is seen with trigeminal nerve damage, combined with significant drooling, accumulation of feed in the oral cavity and reflux of feed and water into the nasopharynx.

If sampling is possible, cerebrospinal fluid analysis reveals a high protein concentration (> 200 mg/L) and increased white blood cell count (> 10/µL).

Bacterial culture of feedstuffs is not a definitive diagnostic tool as many organisms are commonly present in spoiled material. Overgrowth of *L. monocytogenes* is more likely in poorly-made silage in which the pH has not dropped to below 4.5, so the feeding of such silage, in combination with the clinical signs of listeriosis, is supportive of the diagnosis and suggestive of the aetiology.

Key issues

- In feedlots, facial nerve paralysis is the more common clinical presentation, manifesting as a drooping ear, ptosis and lip flaccidity on the affected side.
- *L. monocytogenes* is widespread in the environment and can proliferate in poorly fermented silage. Control measures include the use of inoculants or additional fermentable carbohydrate when the silage is laid down to promote the proliferation of the lactobacilli necessary to drive silage pH low enough to inhibit the growth of *L. monocytogenes*. In addition, adequate compaction and cleanliness when the silage is laid, and exclusion of air from the stack, are essential to prevent aerobic spoilage.

- Mould is associated with aerobic deterioration and therefore may indicate areas with high concentrations of high *L. Monocytogenes* – this material should not be fed.
- Treatment involves an extended course of systemic antimicrobial therapy

Treatment

L. monocytogenes is susceptible to a wide range of antimicrobials, but efficacy *in vivo* can be reduced by the intracellular location of the organism. Treatment needs to be centred on administration of an high-dose, short-acting antimicrobial(s) for an extended period, continuing for at least 48 hours after the cessation of clinical signs:

- Procaine penicillin (300 mg/mL) at 30 mg/kg intramuscular every 12 hours for 7 days, followed by 20 mg/kg intramuscularly every 24 h for 7-14 days, depending on the response to therapy. (Note this is an **off-label** regimen.)
- Oxytetracycline (100 mg/mL) at 10 mg/kg intramuscular every 24 hours for 14 days minimum. Note this is an **off-label** regimen.

Prognosis

The prognosis is better with early intervention. Cases of meningoencephalitis left untreated typically don't recover and die. Supportive therapy with NSAIDs to manage inflammation, electrolyte correction by oral fluid therapy, and optimal husbandry and nursing care will improve recovery rates. Indicators of guarded to poor prognosis include recumbency, nystagmus and the absence of a menace response.

URINARY SYSTEM

Prolapsed Prepuce

Body system/syndrome

Urogenital

Background/nature of infection/organisms involved

In feedlots, the use of hormonal growth promotants (HGP) can increase the risk of eversion and, ultimately, prolapse of the prepuce. Additive risk factors include the anatomical predisposition of long pendulous sheaths in *Bos indicus* breeds, a small preputial orifice, and hypoplasia of the retractor prepuce muscles. Prolonged eversion of the preputial mucosa renders it more prone to irritation and trauma, or desiccation in hot, dry climates. Common sequelae in either of these situations is inflammation, oedema and fibrosis, which can ultimately restrict urine outflow because of stenosis of the preputial orifice and complicate the preputial lesion, resulting in retained urine and associated oedema and inflammation, known as false water belly. Left untreated this can lead to sloughing of large areas of the skin of the ventro-caudal abdomen.

Tests for diagnosis

A prolapsed prepuce is readily recognised by observation, but confirmation can be achieved by closer physical examination under appropriate restraint.

Key issues

- Use HGPs increase the risk of this condition in feedlot cattle.
- Feedlot environments can facilitate infections of the everted preputial mucosa and result in prolapse and further sequelae.
- Breeds with pendulous sheaths are at higher risk (especially *Bos indicus* derived breeds).

Treatment

Medical treatment is focused on reducing swelling and attending to secondary bacterial infection, therefore short acting corticosteroids are combined with antimicrobials to treat the condition. A wide spectrum of microorganisms, both commensal and environmental, are involved.

Under appropriate restraint, consideration can be given to combining systemic therapy with prepuce cleaning with antiseptic solution and topical therapy with emollients, antiseptic ointments or sprays and corticosteroid therapy. For complicated lesions that result in the urine retention, surgical drainage of the prepuce and establishment of alternative urine outflow are necessary.

- A combination in 1 mL of procaine penicillin (150 mg), Benzathine penicillin (150 mg), procaine hydrochloride (20 mg) at 1 mL/25 kg as a single intramuscular injection on the side of the neck
- Oxytetracycline (200 mg/mL) at 20 mg/kg by intramuscular and/or subcutaneous injection on the side of the neck. A second dose should be given 3 days later with consideration of a second dose of corticosteroids.

For cases that have been at the feedlot longer and where exit for salvage slaughter is an option:

- Procaine penicillin (300 mg/mL) at 12 mg/kg by intramuscular injection on the side of the neck every 24 hours for 3-5 days, depending on the response to therapy. Corticosteroids should be administered with first and third injections.

[N.B. The label dose rate for procaine penicillin is 12 mg/kg. However, the recommended effective dose rate is 22 mg/kg (22,000 IU/kg) [24, 25]. Off-label use is problematic due to lack of established WHP and ESI, hence routine use of procaine penicillin is not recommended for feedlot cattle until an appropriate WHP and ESI are available for use at a dose rate likely to be effective in cattle].

- Oxytetracycline (100 mg/mL) at 10 mg/kg by intramuscular injection on the side of the neck. A second dose should be given 48 h later, combined with a second dose of corticosteroids.

Prognosis

Early intervention typically results in a good response, in that restriction to urine outflow is avoided and the animal is fit-to-load for transport to slaughter.

Image source: MLA Image Gallery: https://www.mla.com.au/news-and-events/media-hub/image-gallery/feedlot/

REPRODUCTIVE SYSTEM

Retained Foetal Membranes – Metritis

Body system/syndrome

Urogenital

Background/nature of infection/organisms involved

Parturition in beef feedlots is typically an unwanted and unintended event. Possible sequelae are retained foetal membranes and metritis. It is not common to witness parturition as it occurs, so, if female feeder cattle present with retained foetal membranes in home pens, there is a high probability that the membranes have been retained for greater than 12 hours. In feedlots, adverse fertility effects are of little concern, so the focus is on the potential for systemic illness, with its effects on welfare, weight gain, and suitability for slaughter.

Female feeders that develop metritis typically present in the three weeks following parturition. Retained foetal membranes is a risk factor for developing metritis. A wide spectrum of bacterial agents can be associated with metritis, but the organisms most commonly involved include *Trueperella pyogenes, Fusobacterium necrophorum, Escherichia coli* and *Bacteroides* spp. Female feeders with metritis are typically systemically ill and require treatment.

Key issues

- Fertility and reproductive efficiency are not important in beef feedlots.
- Retained foetal membranes are a concern for welfare, suitability for slaughter and the effects of secondary illness on weight gain.
- Traction of retained foetal membranes should not be attempted in the first 48 hours *post partum*.

Treatment

The preferred approach to management is to remove the female feeder animal and calf (if viable) to a hospital pen for ongoing monitoring and treatment, if required. Membranes should not be removed by traction, but rather resected at the level of the vulva to reduce the risk that they function as a wick for infection of the uterus. The calf at foot will be subject to the feedlot's policy for management of calves born on site, but whether this is euthanasia, transfer of ownership or slaughter, co-habitation in a hospital pen with the dam in the interim is of benefit to both the cow and the calf.

If clinical signs of systemic illness, such as inappetence, dehydration and depression, develop, combined with a foetid discharge from the vulva, the female feeder should be presented to the hospital facility for assessment, including measurement of her rectal temperature. If pyrexia is confirmed, systemic antimicrobials are indicated.

Antimicrobials used

1. Procaine penicillin (300 mg/mL) at 12 mg/kg by intramuscular injection on the side of the neck every 24 h for 3-5 days, depending on the response to therapy.
   [N.B. The label dose rate for procaine penicillin is 12 mg/kg. However, the recommended effective dose rate is 22 mg/kg (22,000 IU/kg); [24, 25]. Off-label use is problematic due to lack of established WHP and ESI, hence routine use of procaine penicillin is not recommended for feedlot cattle until an appropriate WHP and ESI are available for use at a dose rate likely to be effective in cattle].

2. Oxytetracycline (100 mg/mL) at 10 mg/kg by intramuscular injection on the side of the neck. A second dose should be given 48 h later.

Prognosis

For uncomplicated retained foetal membranes, the prognosis is very good. If secondary complications, such as metritis, occur, the prognosis is still generally good for cattle with mild to moderate disease receiving systemic antimicrobial therapy. The prognosis is guarded if there is evidence of toxaemia associated with more severe clinical signs.

INTEGUMENT & ADNEXA

Dermatophilosis – rain scald

Body system/syndrome
Integument

Background/nature of infection/organisms involved
Dermatophilosis, or "rain scald", is an infectious dermatitis caused by a combination of a wet prevailing climate, skin damage and infection of these breaks in the integument with *Dermatophilus congolensis*. Mechanical damage of the skin can be facilitated by biting flies, which can inhabit feedlots in great numbers during spring, combined with rain events, can establish the favourable conditions for *D. congolensis* to infect the integument. *D. congolensis* is an obligate parasite of the skin, but is not considered a commensal. Infection is usually confined to the living epidermis and does not penetrate into the dermis. Usually access to the epidermis is restricted by fur, sebaceous secretions and the stratum corneum, but disruption of these by wetting or trauma allows the motile zoospores of the organism to invade the skin.

The lesions produced are pustules, progressing to scabs and crusts covered in matted hair. The distribution of the lesions reflects the predisposing cause, areas where the skin becomes persistently wet. When biting flies and insects are predisposing causes, lesions are concentrated on the neck and dorsum. The lesions are typically non-pruritic.

Tests for diagnosis
A provisional diagnosis can be made based on clinical signs, the distribution of the lesions and recent history. Confirmation is by demonstration of the unique and characteristic microscopic features of *D. congolensis* in Gram-stained smears from skin lesions, or culture if required.

The lack of pruritis differentiates the condition from chorioptic mange. The distribution and characterisation of the lesions caused by chorioptic mange is inconsistent with dermatophytosis and photosensitization.

Key issues
- Damage to the skin is a prerequisite for infection, with biting flies the likely culprit in feedlots.
- A frequent occurrence during prolonged wet periods.
- The distribution of lesions is frequently along the dorsum.

Treatment
Many cases are self-limiting, especially if rain events are brief and insect control at the feedlot can be achieved. More severe, persistent cases require treatment with a combination of topical therapy (iodine detergent wash of crust lesions) and systemic antimicrobials for severe cases.

Antimicrobials used
- Procaine penicillin (300 mg/mL) at 12 mg/kg by intramuscular injection on the side of the neck every 24 hours for 3-5 days, depending on the response to therapy.
  [N.B. The label dose rate for procaine penicillin is 12 mg/kg. However, the recommended effective dose rate is 22 mg/kg (22,000 IU/kg) [24, 25] Off-label use is problematic due to lack of established WHP and ESI, hence routine use of procaine penicillin is not

recommended for feedlot cattle until an appropriate WHP and ESI are available for use at a dose rate likely to be effective in cattle].

Prognosis

The prognosis is generally very good due to the self-limiting nature of the disease when the predisposing factors are removed.

Body (subcutaneous) abscess

Body system/syndrome

Integument and adnexa

Background/nature of infection/organisms involved

Subcutaneous abscesses can occur in feedlot cattle as a result of injections, penetrating wounds or secondary infection of haematomas, which can be associated with transport, yarding and processing, or injuries in the pens. A variety of bacteria can be cultured from subcutaneous abscesses, with *Trueperella pyogenes* and *Fusobacterium necrophorum* most commonly isolated.

Tests for diagnosis

Clinical signs depend on the site and extent of the abscess – a large swelling involving a limb or both hips may cause mechanical lameness. In most cases, the walled off nature of the lesion means that there is no systemic illness.

Confirmation of an abscess, and differentiation from a subcutaneous haematoma, can initially be obtained by palpation of the mass demonstrating firm, warm swelling and is often painful that developed slowly. Abscesses are usually painful. For a haematoma, the mass would have developed rapidly and be less firm. Ultimate confirmation is by long needle aspiration to demonstrate characteristic pus from an abscess. Haematoma fluid may be at any stage of fibrin-rich organization.

Treatment

Resolution of a subcutaneous abscess is greatly enhanced by lancing at the lowest point of the mass with a large opening to facilitate drainage and exposure to air. On obtaining drainage and access to the interior of the mass, flushing with solutions such as saline or dilute chlorhexidine or iodine can expedite healing. Systemic antimicrobials are not usually required, except in cases with associated significant cellulitis, with multiple abscess sites that are not amenable to adequate drainage and/or evidence of systemic illness (e.g. pyrexia).

Antimicrobials used

- Procaine penicillin (300 mg/mL) at 12 mg/kg by intramuscular injection on the side of the neck every 24 hours for 3-5 days, dependent on the response to therapy.
  [N.B. The label dose rate for procaine penicillin is 12 mg/kg. However, the recommended effective dose rate is 22 mg/kg (22,000 IU/kg) [24, 25]. Off-label use is problematic due to lack of established WHP and ESI, hence routine use of procaine penicillin is not recommended for feedlot cattle until an appropriate WHP and ESI are available for use at a dose rate likely to be effective in cattle].

- Oxytetracycline (100 mg/mL) at 10 mg/kg by intramuscular injection on the side of the neck. A second dose should be given 48 hours later.

Prognosis

The prognosis for recovery is good.

Mastitis

Body system/syndrome

Integument and adnexa

Mastitis occurs rarely in feedlot cattle and for its treatment the reader is referred to the AVA-AMA Antimicrobial Prescribing Guidelines for Dairy Cattle:

https://onlinelibrary.wiley.com/doi/10.1111/avj.13311 [50]

OCULAR SYSTEM

Infectious bovine keratoconjunctivitis – pink eye

Body system/syndrome
Ocular

Background/nature of infection/organisms involved
Infectious bovine keratoconjunctivitis is a common and highly contagious ocular disease that is more commonly seen in cattle that have recently arrived at beef feedlots. The principal infectious agent is *Moraxella bovis;* the significance of *Moraxella bovoculi* is not fully understood [55]. The capacity of these bacteria to colonise and cause disease is enhanced by corneal trauma, which can be caused by dust, excess ultraviolet light, plant awns, a lack of eyelid pigmentation, and/or anatomically prominent eyes. Older cattle are more resistant to infection. Flies are important mechanical vectors for transmission.

Clinical signs of pink eye include corneal ulceration and oedema, photophobia, blepharospasm, excessive lacrimation and epiphora. Progression of the disease results in deepening of the corneal ulcer and sometimes corneal rupture, resulting in prolapse of the iris and permanent blindness.

Tests for diagnosis
This disease is readily recognisable by clinical examination and the characteristic lesions. Culture of ocular swabs will demonstrate the aetiological agent(s), but is of limited value in individual animals.

Key issues
- Corneal trauma allows opportunistic infection
- Younger cattle are more commonly affected

Treatment
Given that feedlots often have limited hospital pen space and opportunities to handle cattle for re-examination, all cases of pinkeye benefit from multiple treatment options and management when first presented. Topical ocular treatments and subconjunctival injections can be given, in combination with eye patch protection of the diseased eye to reduce ongoing trauma and transmission. For very severe cases, and when bilateral disease is present, systemic antimicrobial and NSAID therapy should also be considered.

Antimicrobials used
1. Topical treatment: Benzathine cloxacillin eye ointment 250 mg dose, as required.
2. Cephalonium dihydrate eye ointment 180mg repeated at 48-72 hours as required.
3. Bulbar subconjunctival injection (or superior palpebral, if a bulbar injection isn't able to be administered) of 2 mL of procaine penicillin (300 mg/mL), combined with 1 mL of dexamethasone sodium phosphate (5 mg/mL), twice over a 72-hour period.
3. Systemic antimicrobials for severe cases: oxytetracycline (100 mg/mL) at 10 mg/kg by intramuscular injection on the side of the neck. A second dose should be given 48 hours later.

Prevention
Preventative measures include fly control, weed and thistle control (on farm of origin) and vaccination. Note that cattle often arrive at the feedlot with the condition; vaccination needs to be 3-6 weeks prior to challenge, so vaccination is less relevant in a feedlot context.

Prognosis

The prognosis for recovery is good if intervention is early in the disease process and corneal irritation/risk of trauma can be mitigated.

Image source: MLA Image Gallery: https://www.mla.com.au/news-and-events/media-hub/image-gallery/feedlot/

AURAL SYSTEM

Ear infection (Otitis externa/media)

Body system/syndrome

Aural

Background/nature of infection/organisms involved

Ear infections have been observed more frequently in intensively fed cattle in Australia since the mid-2000s. Most cases of otitis media are thought to arise secondarily from respiratory disease as a result of infection ascending through the eustachian tube, although haematogenous spread or spread from otitis externa can occur. Logically, with infection primarily arising secondarily to BRD, the bacteria most commonly isolated are *Mycoplasma bovis, Mannheimia haemolytica*, *Pasteurella multocida*, *Histophilus somni*, and *Trueperella pyogenes*. Between 0 to 4% (with a mean of 1.7%) of cases of BRD are reported to have developed otitis media (Cusack, unpublished data). In most cases, infection remains localised, but spread can occur to the inner ear in a small number of cases, resulting in meningitis.

Suppurative bacterial otitis is characterised by mucosal thickening of the vestibular membranes and accumulation of thick fluid in the labyrinths. The tympanum can be intact, but if it has ruptured there can be clear, yellow, proteinaceous material in the external ear canal.

Tests for diagnosis

Otitis media is an important differential diagnosis for listeriosis because they share a number of clinical signs, including a head tilt, ptosis, a dropped ear, lip and nostril flaccidity, nystagmus, stumbling or falling towards the side of the lesion. However, unlike cases of listeriosis, animals with otitis media have a normal rectal temperature, normal appetite, awareness and responsiveness to surroundings, in most cases an aural discharge, and, when it occurs, constantly horizontal nystagmus.

Treatment

Based on the aetiological association of ear infection with BRD, the treatment regimens listed for BRD are appropriate.

Prognosis

Most cases of otitis media recover uneventfully.

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