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DÉJÀ MOO

CONFERENCE

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Australian Cattle Veterinarians
6-8 April 2016
Ayers Rock Resort,
Northern Territory

Cattle Vets

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Professional development
for Australia's veterinarians

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THE CATTLE VET CONSULTANT – HERD HEALTH SOFTWARE FOR ACV MEMBERS

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Introduction

In the ideal world, farmers would have a detailed herd health plan that involved activities such as routine husbandry practices, vaccinations, parasite control, reproductive treatments and biosecurity activities that was created with the assistance of an expert who could ensure the appropriateness and correct timing for each component.

The Cattle Vet Consultant is a piece of software designed to allow veterinarians to create farm herd health management plans for farmers in the context of a veterinary consultation.

The problem

Herd Heath programs can be complex. For example, the current MIMMs IVS lists over 30 vaccines that can be used in cattle. Choosing which vaccines would be appropriate for an individual farm and when they would best be administered requires knowledge and expertise, yet many farmers do not consult a veterinarian regarding vaccines, and many only consult a vet about one vaccine at a time.

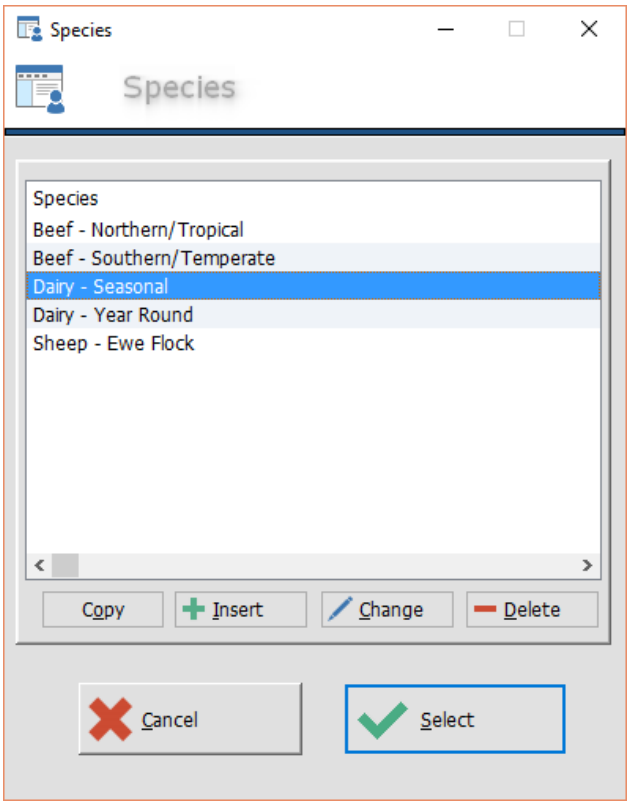
Vets are experts in vaccines, parasite control and reproductive management but anecdotally, many do not do find themselves being used as the primary consultant on many farms where they probably should be.

The “fire brigade” nature of veterinary practice promotes a tendency to look forward for the next few days or weeks

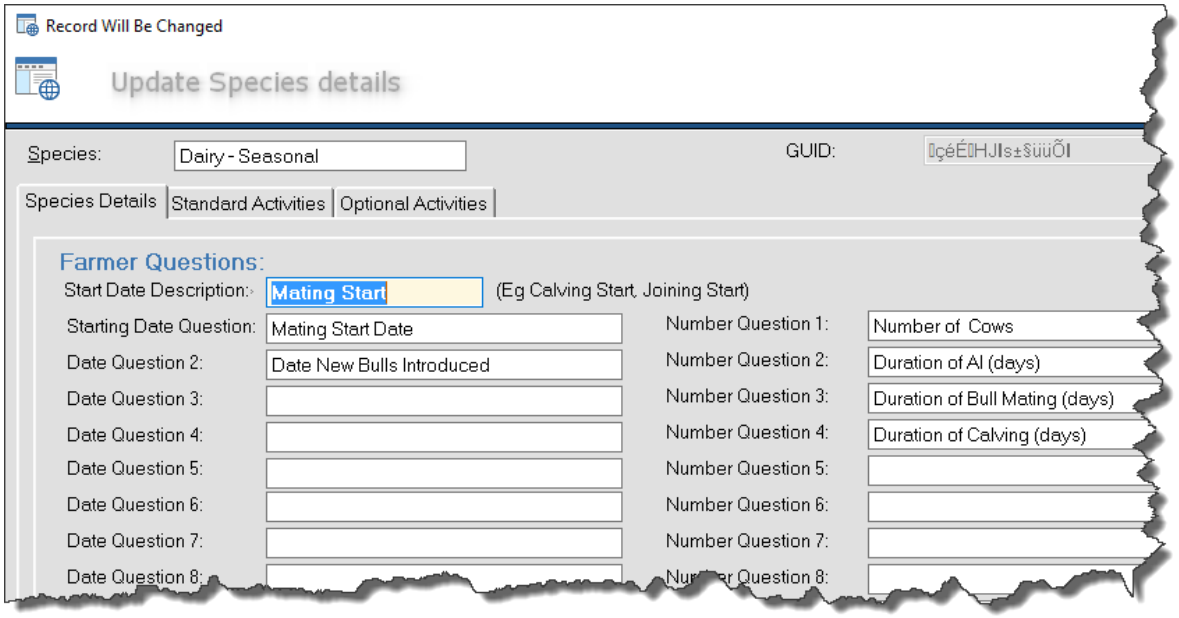
The Cattle Vet Consultant (CVC) allows vets to create personalised herd health programs for farmers, to send reminders for herd health activities, and to consolidate the information so that inventory and workload can be better managed.

Program Templates

CVC allows vets to make their own “Program Templates” for different management systems.



Different templates can be created for different species, or even different variations on the same species. Different individual vets could have their own favourite templates.



Templates allow for 10 number and 10 date questions to be specified. Users can create activities that have timings and durations based on the answers to these questions.

For example, in the Seasonal Calving Dairy template supplied with the program, a date question “Mating Start Date” and a number question “Duration of calving” is specified. An activity “Drying off” could be created that commenced at Mating Start Date +225 days, and lasted for the “Duration of Calving” days. Similarly, reproductive treatments can be specified with reference to Mating Start Date, and vaccines could be scheduled at drying off time. A further question about when bulls are purchased allows for VBBSEs and bull vaccinations to be scheduled.

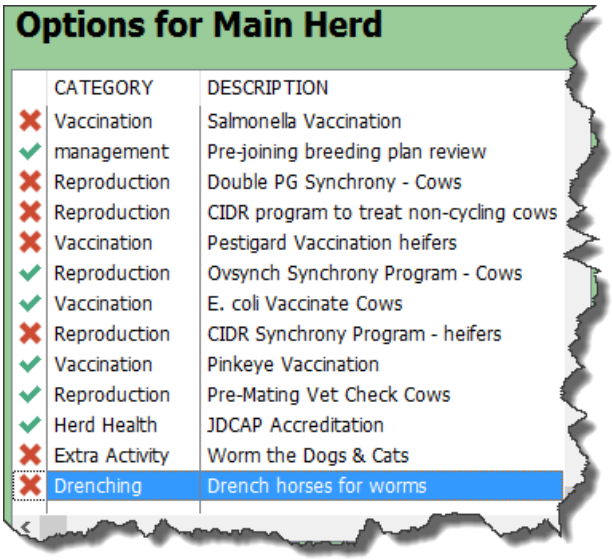
The templates allow for “Standard activities” which occur in every program, and “Optional activities” that can be selected or deselected when designing a program.

“Optional Activities” are actually collections of activities that can be added all at once. For example, an Ovsynch program might contain three injections and some AI days which can all be added with a single mouse click.

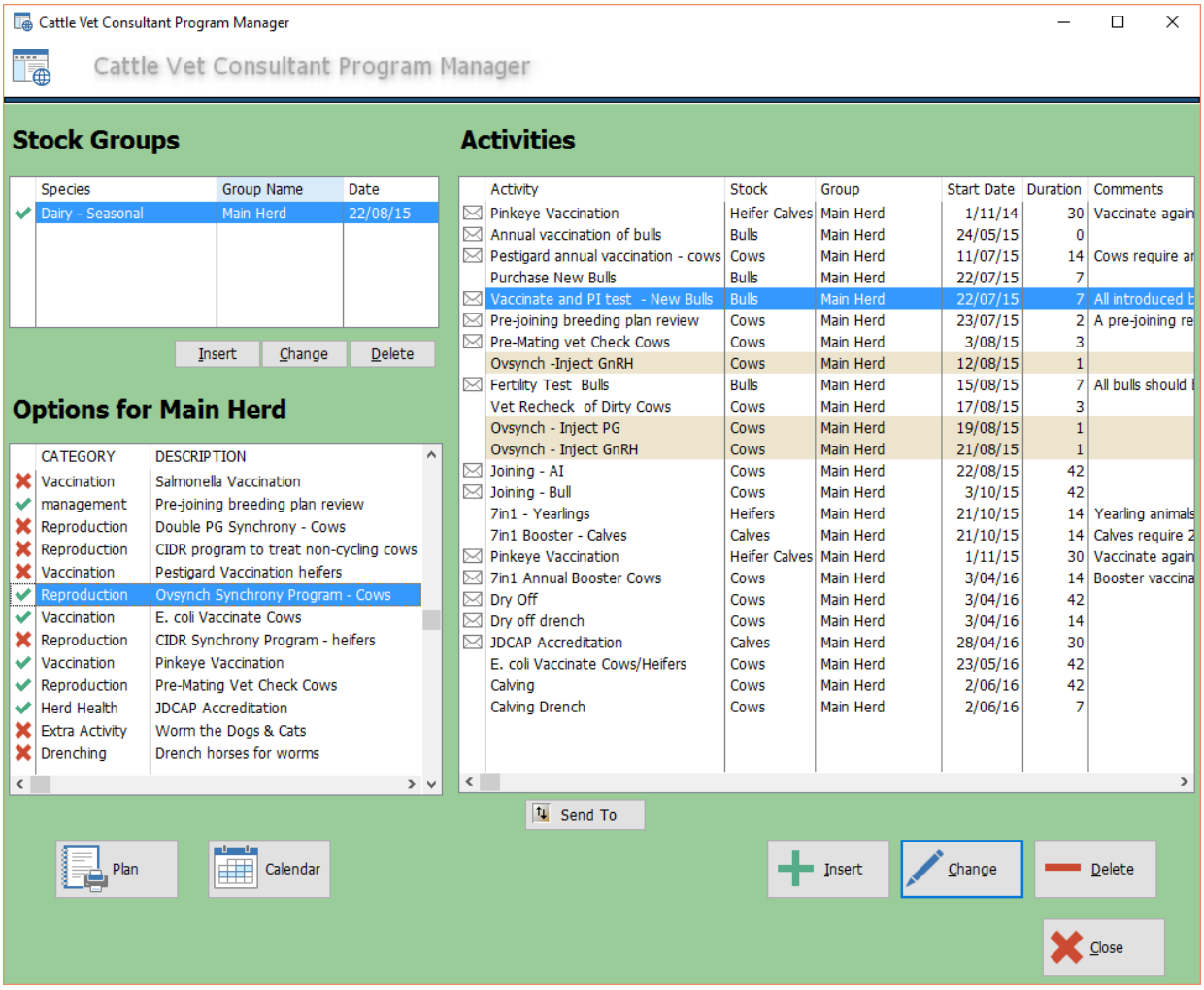
All activities can have default “email templates” associated with them, which can be sent out as reminders at pre-determined times.

Creating a program for a farmer

Creating a program for a farmer is a matter of sitting down at the computer, choosing the management groups, answering the date and number questions and then choosing the optional activities. Examples of optional activities are shown in the image below.



This framework provides the opportunity for having a sensible conversation with an individual farmer about a whole range of activities that can easily be missed, with a view to creating an individual, tailored plan.



The image above shows an individual plan under construction.

Clicking on the green tick or red cross in the bottom left box adds or removes the optional activities. In the above image, "Ovsynch" is highlighted in the bottom left box, and you can see the three activities associated with this in the template are highlighted in the box on the right.

The activities on the right with an envelope icon next to them have a reminder email associated with them.

Once an individual plan is created, any dates or emails can be manually changed.

Emails

CVC allows the planned emails to be sent, but this is a manual process which involves selecting the emails that are due and clicking the send button, which requires that someone regularly schedule CVC into their timetable.

As a general rule, software that sends emails without human intervention or oversight is dangerous!

Consolidation

Whilst it may be valuable to create plans for individual clients, there is also value in the consolidation of the data.

When a vet leaves the clinic, the program they have designed remains behind. Individual vets can see their upcoming planned workload. And practice managers can order vaccines and other drugs that may be planned.

Availability

The CVC will be available for the use of ACV members on a free, as is basis. We don't guarantee it is fault free, but we do hope it will be useful.

LISTERIA MONOCYTOGENES ABORTIONS IN DAIRY CATTLE AND CUTANEOUS LESIONS IN HUMANS – A CASE REPORT

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Introduction

Investigations of abortion outbreaks are commonly undertaken in cattle practice, however a diagnosis is obtained in only 40-50% of cases.[1] This case report describes a case investigation where an unusual diagnosis was obtained, and provides an insight into zoonotic disease in cattle practice.

Case presentation

A group of two year old Holstein cross dairy heifers in Tamworth, NSW were investigated following an outbreak of abortions in in October 2015. Seventeen out of the 44 heifers delivered a dead full term calf over a month. The heifers were four months overdue for a 7in1 booster and had not been vaccinated for pestivirus. They had been moved to the property approximately one month earlier from another property. They were being fed a lead feed partial mixed ration (PMR) and maize silage in racks. The ration had been altered by a farm worker to replace the BioChlor component (used to optimise dietary cation-anion difference) with sodium bicarbonate.

The first case seen by a veterinarian in the outbreak presented as dystocia. On vaginal examination, the heifer had a small calf in normal cranial presentation. The uterus was flaccid and the foetal membranes intact. The calf was easily delivered under epidural anaesthesia. As this was the seventh abortion in a week a post mortem examination of the calf was undertaken, which appeared grossly normal. Fresh and formalin-fixed samples of heart, lung, liver, spleen, kidney, thoracic fluid, skeletal muscle and placenta, and formalin-fixed brain were submitted the following day to the Elizabeth Macarthur Agricultural Institute Veterinary Diagnostic Laboratory (EMAI).

Histopathological examination of fixed specimens from showed autolysis with coccoid bacteria observed in almost all tissues, including brain. Pestivirus antigen was negative. The presence of bacteria within the brain was potentially a significant finding. A foetal IgG and lung culture were performed and a profuse pure growth of *Listeria monocytogenes* was isolated from the lung. The foetal IgG was <121µg/ml, suggesting peracute foetal infection.

Several of the remaining heifers were observed to be “off-colour”, and three were examined ten days after the initial case. Two were pyrexic (temperatures 39.7°C and 39.5°C) and all three had elevated heart rates (120, 100 and 116 beats per minute). The remainder of the physical exam was unremarkable and all three had live foetuses and a closed cervix. Blood samples were collected from the caudal vein and sent to EMAI for magnesium, calcium and glutathione peroxidase levels and results were normal apart from one mildly low magnesium.

Another calf was delivered twelve days after the initial case by a second veterinarian from the same practice. No post-mortem examination was performed on this calf.

Sixteen days after the first presentation, another aborted calf was presented for post mortem examination. This calf grossly had hydrocephalus. Samples of brain were submitted to EMAI and a profuse pure growth of *L monocytogenes* was isolated.

Samples of the corn silage and decomposing material from feed bunkers were submitted for culture. All samples were negative.

Overall, 17 out of the total 44 heifers (39 %) delivered a stillborn calf. No mortalities or further abnormalities were reported in the aborting heifers. Some of the live calves were reported to be "dopey" but this was mild and resolved within several days.

During the course of the investigations both vets and two farm workers who delivered calves developed a multifocal pustular dermatitis on the arms. This was accompanied by headache and malaise which persisted for several weeks. The first vet took ibuprofen (Neurofen 200mg once daily) for the headache. The second vet sought medical attention. Amoxicillin/Clavulonic acid (Augmentin forte 1000mg twice daily) was prescribed and the pustule contents cultured. The culture was not diagnostic. Both farm workers sought medical care. One was treated empirically with dicloxacillin sodium 500 mg four times daily and the symptoms resolved. The second worker was treated with antibiotics but lost to further follow-up.

Discussion

L monocytogenes are non-spore forming small cocciod rods.[2] In cattle the organism can cause encephalitis, third trimester abortions, and opthalmitis or uveitis.[3] *L monocytogenes* is ubiquitous in the environment, and commonly present in silage.[3] It can multiply in poorly fermented silage (pH>5.0-5.5), and corn silage is more commonly implicated in disease outbreaks.[4]

Ingestion is the most common route of infection for the abortion syndrome.[2] The organism crosses the gastrointestinal barrier, multiplies in the liver, and can spread haematogenously to the liver or gravid uterus.[2] Within 24 hours of bacteraemia, the placenta and foetus can become infected. The placenta becomes necrotic and oedematous and can result in abortion or stillbirth.[3]

No case reports of listeria outbreaks in cattle herds were found on a literature search. In a review of abortion investigation samples in Tunisia, *L monocytogenes* was isolated in 4.6% of cases.[5] In the United States, *L monocytogenes* was detected in <1 %-4.1 % of cases for which a diagnosis was obtained.[6]

The incidence of abortion due to listeriosis within a herd is usually considered to be sporadic.[3,7] Figures of up to 15 %[7], and very rarely up to 30 %[1] are suggested in textbooks. This case is unusual in that 39 % of the heifers delivered stillborn calves.

L monocytogenes was only cultured from two of the stillborn animals, and was not cultured from the feed samples or affected humans. The animal's clinical signs were consistent with listeriosis, and, following discussions with pathologists at EMAI, it was felt that there was enough evidence to make a diagnosis for clinical purposes. Immunohistochemistry, and further cultures of calf tissue and human lesions may have provided further supportive evidence, but finances and resolution of human lesions were prohibitive.

The source of the outbreak was not known, however the alteration in the partial mixed ration may have contributed to the outbreak. The addition of sodium bicarbonate may have increased the pH of the PMR, facilitating growth of *L monocytogenes*. The inadequate removal of uneaten ration may also have allowed the organism to proliferate. The mouldy silage fed in racks was another possible source of the outbreak.

L monocytogenes is an uncommon cause of disease in humans[8]. In a review of cases reported worldwide from 1990-2000, approximately one-third were perinatal[8]. Of the non-perinatal cases, the mean age was 50-67 years, and 74% of patients were immunocompromised. Just under half (47%) of patients involved infection of the central

nervous system (CNS) while another 48% were bacteraemic without CNS involvement. Mortality for non-perinatal cases in the reviewed case series varied from 24 to 52 %.[8]

There was no mention of human cutaneous lesions in the above referenced review, however many case reports exist in the wider literature. The pustular lesions on skin that has come into contact with infected ruminant foetal material are the most obvious sign of the disease. Intermittent chills and fever,[9,10,] myalgia, pyrexia and intermittent rigors[11], and fever, myalgia and headache[12], are other reported symptoms. Cutaneous listeriosis without a history of contact with animals has been reported.[13]

Although cutaneous listeriosis was not confirmed by culture in the affected humans, the circumstantial evidence is reasonably compelling. Pustular dermatitis developed within a day on the arms of all four people who delivered calves during the outbreak. They also experienced headaches and lethargy. The clinical symptoms reported correspond closely with those reported for cutaneous listeriosis previously described in the literature.

Previously reported cases were treated with antibiotics, and in one case, fluid resuscitation. All cases resolved with no ongoing complications. Antibiotic choices included oral amoxicillin followed by intravenous benzyl penicillin and gentamicin[13], erythromycin[9] , co-amoxiclav[12], and doxycycline followed by amoxicillin-clavulanic acid[10]. For the cases in this series where antibiotic information was available, use of the penicillin class produced a favourable outcome.

Zoonotic transmission of cutaneous listeriosis has demonstrated in a case report where pulsed-field gel electrophoresis was used to genotype the aetological isolates[10].

This case report illustrates the importance of using personal protective equipment (PPE) during cattle obstetric work. The Australian Veterinary Association recommends the use of impervious outer wear, shoulder length gloves, face mask or respirator and protective goggles during such procedures[14.]

Conclusion

This case report describes an unusual outbreak of abortions due to *L monocytogenes* in dairy cattle. Listeriosis should included on the list of differentials in the investigation of abortion outbreaks. It also illustrates the need to wear PPE to reduce exposure to zoonotic pathogens when handling animals.

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BOVINE FAMILIAL CONVULSIONS AND ATAXIA

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Introduction

Bovine Familial Convulsions and Ataxia (BFCA) is considered a rare heritable condition in cattle. It has been reported in Angus[1], crossbred Angus[2], Charolais[3], and cross bred Polled Herefords[4]. This condition is autosomal dominant incomplete penetrance of approximately 20-30%.[2,5] The syndrome may be underdiagnosed, and animals are being euthanized or culled. Cerebellar abiotrophy is a hallmark finding of this condition. Abiotrophy was first described as premature degeneration or aging of certain tissues, especially the nervous system.[6,7] The term is used when the degeneration is due to an intrinsic abnormality as opposed to and external insult. The first report of BCFA was in 1968 in Angus calves.[5] I have a unique case series I would like to share to encourage a better understanding of this condition.

Clinical exam

Cerebellar abiotrophy is the trade mark finding with this condition. Calves are usually born normal though have been reported to be born abnormal. The individuals will present with a variety of clinical signs that are attributable to cerebellar pathology. Characteristic clinical signs of cerebellar dysfunction include ataxia with normal strength, hypermetria, wide base stance and fine head tremors. These signs are common findings with this disease. Recurrent convulsions and seizures associated with BCFA are characterized by rigid limb and neck extension from a few minutes to 10 hours. Fine head tremors are also reported. Corneal ulcers may be seen, though are likely due to trauma experienced during convulsions and consistent with exposure keratitis. Various signs of trauma including abrasions, contusions or fractures may be identified experienced from traumatic events. Animals may have pneumonia for aspiration or may have been found dead from drowning. Complete blood cell counts and serum biochemistries rarely demonstrate significant findings. Biochemical examinations offer little benefit to making a diagnosis.[8]

Disease progression

If calves survive clinical signs may subside. Seizure activity may subside by 12 months of age, and ataxia by 2 years of age. Though there has been one animal reported to be sold at 25 months of age failed to become normal.[4] Calves may present with clinical signs from birth to eight months of age. Clinical incidence has been reported to be approximately 30% and up to 39%.[5,8]

Post mortem exam

Remove the brain as carefully as possible. The brain will be grossly normal. The whole brain can be place in formalin. It is important to place the brain in formalin within 1-2 hours of death to identify the histologic changes. If you are concerned about an infectious cause such as bacterial meningitis, a swab of the brains base may be taken if the basal attachments were incised with sterile technique. A complete necropsy should be performed. Secondary lesions may be found to include trauma or aspiration pneumonia obtained due to ataxia or convulsions.

Histopathology

Characteristic histological lesions were seen in the lingula, uvula, or both. Other parts of the vermis may be affected in individuals more severely affected.[9] Lesions include segmental degeneration and loss of Purkinje cells, and axonal swellings. Axonal swelling may develop "Torpedo"-shaped structures in the outer granular layer.[9]
Electron microscopy may also be a beneficial tool. Genetic and biochemical markers have

This case series

History

Approximately 200 calves were born on a property in South East Queensland. One recently purchased bull sired 19 offspring. 17 of which were abnormal. The dams represent several breeds and crossbreds to include Charolais, Charolais Angus cross, Santa Gertrudis, and Milking Short Horn. All dams were multiparous and previous offspring with different sires resulted in normal calves. Vaccination on the property consisted of 5 in 1 as juviniles.15 of the 17 abnormal calves were born on the primary farm, the site of breeding

along with the two normal calves. The other two abnormal calves were born on a second property. 6 of the 17 abnormal calves died; 4 from drowning, one stuck in mud, and one from suspected dehydration as a result of scours. Of the drownings, two occurred at the secondary farm at 3.5 and 4.5 months of age. The other two were and 1 and 6 weeks of age. Two calves were euthanized at 2 months and 5 months of age due to animal welfare concerns. The one at 2 months of age received a full necropsy. Three were harvested at a local abattoir. The brains were collected and examined grossly and with histopathology. A captive bolt was used damaging the cerebrum, though no gross or surrounding histologic abnormalities were identified in the cerebrum. The cerebellum was undamaged. The owner reported abnormal calves as abnormal movement (ataxia) though able to navigate (normal mentation) with occasional seizures. Clinical signs may be minimal in the paddock, though are exacerbated when stressed. Age of onset varied from a few day of age to 2 months of age. Most were 2-3 weeks of age at onset. The bull had already been sold for meat when we were contacted for investigation.

Clinical Exams

Eight calves were examined on initial visit. The calves all appeared in healthy state and well-muscled. All were experiencing ataxia of various degrees with normal mentation and vision. All were able to appropriately navigate their environment. No cranial nerve deficits or proprioceptive deficits including knuckling or difficulty navigating terrain changes were observed. Two of the 8 were experiencing corneal ulcers on one eye. No additional ophthalmic abnormalities were observed. It was deemed these were secondary to seizure activity and were traumatic in nature resulting in exposure keratitis. One was unable to fully rise on its front feet and walked on its carpi. No, musculoskeletal or peripheral nerve deficits could be identified. The owner reports that this calf becomes this way when stressed though becomes normal becomes normal in 2 days of unstressed environment. This has happened twice previously and was able to rise within 2 days of the first visit. This calf was harvested at an abattoir and was able to walk in normally. On the calf euthanized on the first visit, righting reflex from both lateral recumbences, hopping and wheelbarrow tests and all reflexes were normal.

On the second visit 3 calves were examined at the abattoir. All were affected with ataxia with normal mentation and no CP deficits. No other abnormalities were noted. No gross abnormalities were noted post mortem. The brains were collected for further examination. Three calves that were examined at the first visit were reexamined. A corneal ulcer previously examined was still present, though had healed significantly. Ataxia was of similar severity in the three individuals and seizures were still reported to occur. Mentation, cranial nerves and conscious proprioception was normal. A very interesting find was that all calves including two “normal” were exceptionally well muscled for the sire, even a calf with a milking short horn as a dam. This has not been reported previously. The closest thing is an affected heifer that became supreme champion at a fat stock show.[5]

Differential diagnosis

There have been other forms of cerebellar abiotrophy reported without seizures[10,11] or non hereditatary.[12] Bovine Viral Diarrhea Virus must be on your differential list and should be tested for if you observe cerebellar signs in calves. Due to the significant impact of this disease on a herd, it would be imprudent to not test for this disease. The absence of brain stem or cerebral clinical or pathologic exam signs rules out many common neurologic conditions resulting from inflammation, deficiencies or toxins to include; polioencephalomalacia, listeriosis, encephalomyelitis, thromboembolic meningoencephalitis, meningitis, malignant catarrhal fever, water intoxications, lead poisoning, mercury poisoning, tick paralysis and rabies (not in Australia). A cervical spinal cord lesion with ataxia is possible. Though decreased conscious proprioception would be present and seizures would not be expected. Bilateral otitis interna could be possible, though deafness would be expected, and convulsions would not be expected. Alpha mannosidosis was considered though this affects all portions of the nervous system and cerebral signs would be expected including obtundation. GM1 gangliosidosis may produce ataxia, though reluctance to move and obtundation are also present. Progressive ataxia has been described in cattle, though with this syndrome difficulty posturing for micturition and is due to leukodystrophy. Meningoencephalitis has been reported to produce cerebellar signs, were differentiated on gross necropsy and histologic exam. Cerebellar neoplasia was considered, though ruled out on histologic exam. Various

toxins have been reported to cause cerebellar signs, though no history of pasture or management changes were made, and no differences in the paddocks were observed.

Diagnostic tests

1st visit

Clinical and neurologic examinations: consistent with cerebellar disease

Plasma obtained from 8 affected individuals. All negative for BVDV by PCR

Serum biochemistry and Complete blood cell count: no significant findings: mild increase in CK.

Gross necropsy: no significant findings. Focal lung consolidation

Brain Histopathology: consistent with cerebellar abiotrophy and BFCA.

2nd Visit

Clinical and Neurologic examinations: consistent with cerebellar disease

Post mortem exams: no significant findings

Brain histopathology: pending

Conclusion

Clinical signs consistent with previous reports of BFCA in addition to supporting histopathologic findings support the diagnosis of an unusual presentation of BFCA. Strong evidence of a dominant inheritance pattern with incomplete penetrance is present. A high percentage of the offspring of one bull were affected while no other calves on the premises were affected. Two significant findings were present in this case series. Previously unreported high incidence of almost 90% (17/19), and a disproportionately well-muscled presentation of the calves. These add some interesting aspects to this previously diagnosed condition. This condition may be more common than currently known. The problem does not respond to common pharmaceuticals, is sporadic and presents with unusual neurologic signs. Owners may conclude that euthanasia or salvage are the only viable options. I encourage veterinarians and owners to contact me or submit samples if they suspect they encounter this condition.

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PERFORMING FACILITATED ANKYLOSIS OR DIGIT AMPUTATION

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Introduction

This paper is not intended to be an exhaustive resource for digit disease in cattle. This paper will address two options for treating severe digit disease of the bovine. Severe digit disease can be debilitating to the individual animal. When more conservative therapies are not sufficient, surgical intervention may become the best option.

Bovine Digit Analgesia

History: August Bier, a German surgeon first introduced intravenous regional anesthesia in 1908 thus creating the procedure name "Bier block". Procaine was the local anaesthetic of choice. This block lost is popularity, though gained new popularity in 1963 by C. McK. Holmes with the novel use of lidocaine (lignocaine). A bier block is a regional perfusion of local anesthetic. This is a highly effective method of providing local anesthetic. Lidocaine is the most commonly used drug for this purpose at the University of Queensland. A tourniquet is applied dorsal to the fetlock joint. The most common vein that I use for the bovine foot is the dorsal common digital vein, or the palmar/plantar common digital vein. This vein is located on the dorsal or palmar/plantar midline and is most often a blind stick. The best place I find is approximately 5-8 cm dorsal to the interdigital cleft. The second most commonly used vein of the bovine foot is the abaxial palmar digital vein. This vein can typically be identified distal and slightly dorsal to the dewclaw running in a proximal plantar/palmar to distal dorsal fashion. The chosen area should be clipped and aseptically prepared. A Bier block is accomplished easily with a butterfly catheter placed into one of the two veins. A flash of blood is identified and 20 to 30 ml of 2% Lidocaine is typically injected. This is the preferred anesthesia for digit amputation or distal interphalangeal joint resection.

Disease States

There are several disease states that may benefit from more aggressive surgical therapy. The most common disease state that I encounter that benefits from a facilitated ankyloses or digit amputation is a septic distal interphalangeal (DIP) joint. The primary aetiology of septic DIP in beef cattle is unknown. In dairy cattle it is believed to derive commonly from sole ulcers. Additional causes may include sole abscesses, white line disease, penetrating foreign body, and chronic foot rot. The classic presentation of a septic DIP joint is that of swelling and pain unilaterally on the coronary band. Most often there are draining tracks present on the coronary band or sole. A common history is that the animal has exhibited chronic lameness and was unresponsive to antibiotics as it was believed to be foot rot. Foot rot classically presents as symmetrical swelling. Various other structures may be affected as well. These include the distal sesamoid bone and its bursa, the superficial and deep digital flexor tendons and their tendon sheath. If there is pain to palpation of the heel consider that there may be involvement of the sesamoid bone and its bursa. If swelling extends proximally on the palmar or plantar aspects of the foot, consider that there may be tendon involvement. The toe may turn upwards as well due to tendon injury or pathology.

Treatment

Antibiotics alone will rarely result in a cure of a septic DIP. Flushing with a balanced polyionic solution is the least invasive process. We will typically utilize LRS for this procedure. We have seen occasional success with this procedure, though there is no literature to suggest a success rate.

The most invasive procedure is that of a claw amputation. This may be accomplished in two ways, foetotomy wire method or a disarticulation method. With either method a foot block should be applied to the non-surgical digit to allow more clearance and decrease the incidence of injury to the surgical site. The production life of cows with digit amputation has been estimated at 10-27 months in a study of 85 cows.[1]

(Foetotomy wire method)

A sharp circumferential incision is made with a scalpel blade approximately 1-2 cm dorsal to the coronary band. The P1-P2 joint space is

approximately 2 cm dorsal to the coronary band on adult cattle. The aim of your amputation is just proximal to this joint, the proximal interphalangeal (PIP) joint. Apply the foetotomy wire interdigitally and cut through the soft tissue with a scalpel blade up to the level of the P1 P2 joint space. You want to stay close to the digit you are amputating and go no farther dorsal than necessary. Anecdotal evidence suggests that preservation of the interdigital ligaments may improve the longevity of the patient. With wire in the interdigital space, saw with an angle of 45 degrees to the proximal digit abaxially. The cut should go through the distal portion of P1. Once the digit is removed, apply a very tight bandage to control haemorrhage. Multiple layers of gauze and an adhesive bandage is applied. The bandage should be replaced at 24 hours and again every 3-5 days as needed until the wound is covered by granulation tissue. Appropriate broad spectrum antibiotics may be administered for 5 to 10 days post-surgery. The animal must be maintained with appropriate hygiene.

(Disarticulation method)
An alternative surgical approach is to perform a disarticulation of the PIP joint. A sharp vertical skin incision is made on the abaxial digit surface from the coronary band 4 to 6 cm above the coronary band. A horizontal incision is made circumferentially just proximal to the coronary band. You want to preserve as much skin as possible. The joint PIP joint is exposed on the abaxial surfaces with a scalpel blade and it followed dorsally to start the disarticulation process. The disarticulation is continued caudally until reaching the flexor tendons. The major vessels medial to the flexor tendons may be ligated or the digit may be twisted as you sharply cut through the deep and superficial digital flexors. The incision may be sutured in a T fashion with a continuous or interrupted pattern. A drainage site is recommended to keep open to allow sufficient drainage. A bandage is still applied.

Facilitated ankyloses
Remember an arthrodesis is performed essentially when you place foreign material to help stabilize the joint. Moreover the procedures herein are ankyloses rather than arthrodesis. There are four main methods of performing a resecting the DIP joint in cattle resulting in a facilitated ankyloses of the joint. A foot block is applied to the non-diseased digit. The approaches are the abaxial, solar, bulbar and proximal. Breeding bull production life has been estimated to be a mean of 21.2 months for joint resection and 27.2 months for digit amputation.[2] Though there are many factors that may go into production life and this was a small study of 21 bulls.

Abaxial approach
An imaginary point is made with two intersecting lines. A parallel line is drawn one third the distance ventral and parallel to the coronary band. A second line is drawn vertically equidistance from the heel bulb to the dorsal hoof wall. A 6 to 12 mm drill bit is used to drill through the same location on the medial aspect of the digit.[3] A Penrose drain is placed for 3 to 5 days. Flushing with dilute “weak tea” betadine once to twice a day is ideal. A bandage is placed over the digit between flushing. This technique may be easier to perform though drainage will not be as good as other techniques and a hoof wall defect will be present. This technique may require less aftercare than others.

Solar approach
This approach is designed for an animal with a severe solar wound or lesion. An elliptical incision is made at the sole heel junction around the necrotic area. The deep digital flexor tendon may be resected off of the distal phalanx and thus exposing the distal sesamoid bone. This bone may be removed to expose the DIP joint.[3] A drill bit is used and is aimed to exit 1 cm distal to the dorsal coronary band. This technique provides great drainage and visualization of the DIP joint, but this technique may result in an upturned toe and abnormal hoof growth.

Bulbar approach
A horizontal incision is made around the circumference of the heel bulb 1 cm below the coronary band. A wedge of subcutaneous tissue and tendon is resected to expose the sesamoid bone. The sesamoid bone is thus removed exposing the DIP joint.[3] The joint is thoroughly lavaged and the foot can be bandaged. This technique provides good visualization of the DIP joint without invading the tendon sheath when it is not necessary. This technique may result in an upturned toe and abnormal hoof growth.

Proximal Approach
This approach is used if the infected joint has tracked to the skin surface or “gravelled out”. This usually occurs near the coronary band. A probe can be used to evaluate the lesion to assure it is headed to the joint. Sometimes the

probe can be pushed all the way through the joint. A scalpel is used to increase the size of the opening, and a drill is used to debride the joint. You want to exit 1-2 cm below the axial coronary band midway from dorsal and palmar/plantar. Copious lavage is used and a drain is placed. The foot can be bandaged and daily lavages may be used for 3-5 days. Healing is by second intention. This is the most common method I use because they are most often presented to me with an existing exit point of the infected DIP.

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PRACTICE TIPS FOR AN EASIER CALF OMPHALECTOMY

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Introduction

Do to the time limit of this presentation this paper is not intended to be an exhaustive resource for umbilical surgery in calves. This paper will address a method that I prefer to use, and may give you some different tips. This is a common surgery performed for a variety of reasons that may become more fun with small tips. If you desire more in depth discussion of the procedure please see a reference.[1]

Nasotracheal or orotracheal intubation

Calves may experience profound hypercapnia, hypoxemia, and glottis stenosis. By intubating may allow improved ease of breathing and thus less resistance to conscious surgery. I find it easiest to place a nasotracheal tube in a calf while standing and before sedation. Some individuals find it easier to complete this task with the calf in dorsal recumbency and the head extended. You place the tube in the ventral medial meatus of the nose similar to passing a nasogastric tube in a horse. The tube is advance through the nares to the larynx. This next part can be a bit of an art. Wait until the calf takes a deep breath in and advance the tube at the same time. The tube may go in to the esophogus when you are first attempting this technique, though with practice it becomes easier but still not always easy. I do prefer a nasotracheal tube over an orogastric tube do to the minimal risk of chewing in the still conscious patient. This step is not essential though we do find the calf struggles less with this in place. This task can be challenging, and I had to do a few calves before I became competent with the procedure. It is worth practicing, and giving it a go to see if it can fit well with your surgical plan. You may use 8 to 10 mm OD tubes that are 30 cm long for this purpose. A nasogastric tube can be used in other parts of your bovine practice as well. I like to place one in adult cows that are experiencing a difficult dystocia. A mammal needs to close its glottis to create an abdominal push. With the tube in place the cow cannot push against what you are trying to accomplish with your manipulations. Once you want her to help more, you can remove the tube.

Sedation

I prefer to use a ketamine stun.[2] This allows nice multimodal sedation with small pharmaceutical volumes. A typical standard is butorphanol 0.025 mg/kg, Xylazine 0.05mg/kg and ketamine 0.1mg/kg. This may be administered IV, SQ or IM. If the IV route is chosen I will only give half IV and the other half IM. A half of a full dose can be near by if more sedation is needed. This is a common sedation cocktail I like to use in a variety of cases. This is typically a recumbent dose. A standing stun may be done with half of the total dose. This should only be administered when you are close to surgery. Most of the clipping and a “rough surgical prep” can be performed before placing the calf in dorsal Recumbency for final scrub and surgery.

High volume caudal epidural

Lignocaine HCL recommended dose range varies from 1 ml per 2.2 kg to 1 ml per 10 kg.[3,4] I prefer to use 1 ml per 5 kg in my high volume caudal epidurals for calves. This procedure provides anaesthesia to the abdominal wall and musculature along with paralysis of the rear limbs. This improves surgeon safety and patient cooperation. Local lignocaine will still be required on the cranial half of your incision. This is typically not anesthetized by the epidural. This technique and inhalant anaesthesia is well tolerated by calves and they maintain good cardio pulmonary and metabolic function when compared with injectable anaesthesia.[5] Once the injection has been performed, a common mistake is to place them in dorsal recumbency immediately. Keep them in sternal recumbency for 3-5 minutes to assure the ventral spinal roots have been appropriately bathed in lignocaine. This technique can be used for a variety of procedures in production animal practice. On a side note, I really like to use this for adult cattle needing a ventral midline caesarean section. This works really nice, and it becomes fun to give 100-150 ml through an epidural!

Surgical approach

This is not intended to be your primary source for this surgical procedure, as this is just

intended to give you some tips and things to consider.

An elliptical incision should be made twice the length of the base of the hernia. Dissection through the soft tissue to the body wall appears to create less haemorrhage than sharp dissection. The ideal place to enter the bovine abdomen for umbilical surgery is the cranial left aspect. You have the lowest risk of hitting the urachus or umbilical arteries that come off the caudal aspect or the umbilical vein that comes of the cranial right in the bovine. Digital exploration should be performed to evaluate for all potential structures before extending the incision. The hernia ring should be removed. This part of the abdomen is abnormal and is not ideal to anchor sutures to. The most commonly involved infected structure is urachal sepsis. Secondly is omphalophlebitis which is inflammation of umbilical vein. Thirdly is omphaloarteritis which is inflammation of the umbilical arteries. It is also common to have omentum adhered to hernia or structures in question. The components that are infected should be removed. If the urachus is involved, it is resected. The apex of the bladder may be easily removed and sewn with two inverting patterns of your choice. A Cushing's or Lembert pattern works well for this purpose. You want to make sure to not penetrate the mucosa which could allow urine leaking and degrade the suture. Umbilical arteries may be involved with the urachus or independently and can be resected in a similar fashion. If the umbilical vein is infected it may go up to the liver. If this is the case, a liver is not easily resected. I will marsupialize the vein to a new incision approximately 2-4 cm right lateral to the primary incision. I will place a sterile glove finger over the vein and redirect it to the new incision and suture it to the skin. A temporary encircling suture may be place around the vein to decrease the risk of abdominal contamination. This can be best accomplished with a pair of sponge forceps placed through the new small lateral incision. At least 1 cm of vein should be externalized. It will need to be flushed 1-3 times a day with dilute betadine until it heals by secondary intention. This may be performed with a red rubber catheter. This does take a dedicated owner or hospital staff to provide this aftercare.[1] By sewing the vein into the cranial aspect of the original incision a defect may result in the body wall requiring a 2 step surgery.

Closure

This is an area that creates a lot of debate. "Vest over pants" was a long standing popular method of closure. This has fallen out of favour in recent years because it has been shown to be significantly weaker than appositional closure and thus result in poorer healing. I prefer to close my incision with cruciate or inverted cruciate sutures. You have to make sure not to space your sutures very widely to ensure a tight apposition. To eliminate tension, tension relieving sutures can be used to close very large gaps. My favourite tension relieving pattern is a near far far near pattern.

After care

An abdominal compression wrap may be used for large defects or cases that you are concerned about. I typically prefer to keep the calves in a small area for 10-14 days without other calves. This area should also be clean, dry, and allow good opportunity for foot purchase and minimal opportunity for jumping.

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HOW TO TREAT DAIRY HERD BULLS: LIKE PERFORMANCE ATHLETES

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Introduction

In Australia and globally, dairy herd bulls ('mop-up' bulls) are often a forgotten part of the fertility picture, which is reflected by a lack of available research data. This article is adapted from recent research in Victorian seasonal and split calving dairy herds, in which dairy herd bulls underwent a pre- and post-mating bull breeding soundness evaluation (BBSE), as well as a management questionnaire. The objectives were: to describe the management practices of dairy herd bulls; describe and quantify the causes of reduced fertility in these bulls, as measured by the BBSE; describe the causes of bull breakdown during mating; and identify any possible associations between bull management practices and the BBSE results. Pertinent results from the study have been used to make practical recommendations on how to manage the dairy herd bull team. Using the analogy of the dairy bull team as an elite sporting team provides an easy framework for disseminating this information to dairy clients.

Background

The fertility of seasonal calving dairy herds in Australia has suffered a steady decline in recent decades. To quantify this decline, Dairy Australia funded a 10 year study of the reproductive performance of 74 split and seasonal calving Australian dairy herds. Over the period 2000-2009 there was a decline of approximately 1% per year in the 6-week in-calf rate, with similar declines in reproductive performance as measured by 12 and 21-week not in-calf rates (1.1% and 0.7% increase per year respectively).[1] In a search of this report, the InCalf Fertility Data Project the word 'bull' appears only 3 times in a 217 page document.[1] This is not a criticism of this substantial body of work, but it highlights that bull management is a topic which needs further investigation.

It is arguable that in a herd with good reproductive performance in the AI period, that the job of the bulls during the natural mating period is of minor importance in the overall fertility of the herd. However, given that the 'typical' 6-week in-calf rate of herds in the InCalf Fertility Data Project was 50%,[1] then in herds with an AI period of 8 weeks (the average for this study), there may still be approximately 30 – 40% of the herd remaining at the end of the AI period for the bull team to impregnate. This means that with decreasing AI period performance, the importance of the natural mating period is increasing, and subsequently so is the need to manage any risk associated with the performance of the bull team.

Whilst the BBSE has been extensively studied in beef herds, there is very little peer-reviewed published data which investigates the management and breeding soundness of dairy herd bulls. Conference proceedings have been produced discussing the topic,[2-6] including one which reports the pre-mating BBSE results of dairy herd bulls in Tasmania.[5] There have only been a handful of studies which have collected pre- and post-mating BBSE findings in beef herds[7-11] and there appears to be none in dairy herds. Whilst there are many similarities between the use of beef and dairy bulls, there are key differences in extensive dairy enterprises. The most important difference is that during the mating period most dairy herd bulls have to walk along dairy tracks daily with the milking herd, and each day spend time standing on concrete yards. This is in addition to their reproductive workload and probably contributes to bull breakdown. The research findings and recommendations described here were inspired by this need for dairy specific research into bull management and breeding soundness.

The Project

A pre-mating BBSE was performed on bulls from seasonally and split calving dairy herds in SW Victoria in the 2 months preceding a natural mating period (which was preceded by a period of AI mating in all herds). Immediately following the natural mating period, a post-mating BBSE was performed on the bulls which were used. At both visits a management questionnaire was performed to assess the management of the bulls from rearing up to mating, and then during the mating period. The BBSE was performed according to the ACV guidelines for physical, crush side semen, and sperm morphology.[12] A serving ability assessment was not performed. Disease testing was performed for bovine viral diarrhoea virus (BVDV), vibriosis, bovine herpes

virus, and trichomonosis, and the detailed results of these tests are discussed elsewhere.[13]

Statistical analyses included descriptive analyses of the BBSE results and bull management practices. Univariable analyses were used to examine associations between bull and herd-level risk factors and the results of the pre- and post-mating BBSE. A mixed effects logistic regression model was constructed to assess the relationship between bull-level risk factors and the pre-mating BBSE results. A second mixed effects logistic regression model was constructed to assess the relationship between herd-level risk factors and the post-mating BBSE results. In both models the variable "Herd" was included as a random effect.

The Line Up
Pre-mating, a total of 256 bulls were tested from 32 herds (17 seasonal and 15 split calving), and 200 of these bulls were presented to be re-tested at the post-mating BBSE. The herd size on average was 425 cows (154-910). The breed composition of the bulls was: Holstein – 171 (66.8%), Jersey – 54 (21.1%), Other (Angus, Murray Grey, Swedish Red, Aussie Red, Guernsey, Hereford, Shorthorn, Ayrshire, Jersey x Holstein) – 31 (12.1%). The average length of the preceding AI period was 8.0 weeks (3-16), and the average length of the natural mating period was 8.3 weeks (2.5-19).

1st Half Highlights: Pre-mating BBSE results
At the pre-mating BBSE 20% of bulls were classified as 'High risk', 17% were classified as 'Qualified', and 64% were classified as 'Low risk'. Of the 'High risk' classifications, approximately half were due to poor semen quality and the other half due to physical defects (mostly lameness and penis problems).

2nd Half Highlights: Post-Mating BBSE results
At the post-mating BBSE 37% of bulls were classified as 'High risk', 22% were classified as 'Qualified', and 42% were classified as 'Low risk'. The most common reason for a 'High risk' classification post-mating was lameness (67% of all 'High risk'), and 25% of all bulls were lame post-mating. Of the bulls used 16% were reported to have been removed from the normal mating rotation by the herd manager, and lameness and injuries were the cause of almost all of these breakdowns.

"The bull team is an elite sporting team"

1. Select your squad
2. Pre-season training
3. Pre-season fitness test
4. Manage the game/season

Select your squad
In any competitive team sport it makes sense to select the best players in the team to get the best results. In the same way as selection criteria such as skills, experience, and injury status may weigh into sporting selection decisions, there are important criteria to use when selecting the right bull team. Based on this research, some of the factors to consider when selecting the bull team include:

- **Age:** In the final pre-mating mixed effects regression model, bulls which were 4 years of age and older had twice the risk of failing the pre-mating BBSE than bulls younger than 4. These findings concur with industry resources which recommend using bulls between 15 months and 4 years old[14]. Although not making it into the final model, in the univariable analysis virgin bulls had half the odds of failing the pre-mating BBSE when compared with experienced bulls.

- **Breed:** Overall there was no difference in the overall proportion of Holstein or Jersey bulls which failed the pre-mating BBSE, although there were some differences between the breeds. Jersey bulls had significantly poorer sperm morphology and Holstein bulls had significantly straighter legs and were more likely to have abnormal leg joints (predominantly swollen hocks). Whilst these differences are of interest, ultimately the final decision on which breed to use is probably going to be influenced by factors such as cow breed, genetics (if keeping replacement calves from natural mating), ease of calving, and whether heifers born from the natural mating period are intended for export.

- **Bull Power:** The average proportion of bulls which failed the post-mating BBSE per herd dropped from above 40% at mating ratios below 3% (3 bulls per 100 cows to be mated at the mating start date for AI) to below 20% at ratios 3% or greater. These results appear to support

the current InCalf recommendations[14] of at least 3% (or 4% if herd synchrony is being used). Despite industry recommendations to simply increase bull power,[14] it is important to use better bulls, not just more bulls. Increasing the mating ratio will not necessarily compensate for high levels of sub-fertile bulls. Socially dominant bulls serve more cows and sire more calves than less dominant bulls,[15] and if these bulls have abnormalities such as lameness, penile injuries, or poor semen quality, then having higher numbers of sound bulls may not fully compensate. Even if similar numbers of pregnancies are achieved, they may occur later in the mating period, resulting in late calving cows which are less likely to conceive in the subsequent mating.[16] Therefore our recommendation is to use a ratio of at least 3% of bulls which have passed a pre-mating BBSE.

- **Conformation:** It is probably impractical for farmers to be selecting mop-up bulls based on conformation alone, but there were some findings of interest to report with respect to hind leg score. The higher the bull's hind leg score (i.e. the straighter the hind leg) the more likely a bull was to have swollen hock joints. Furthermore, swollen hock joints were associated with a higher risk of lameness. As mentioned previously, Holstein bulls were predisposed to this problem. Unfortunately for logistical reasons we didn't lift the bull's feet up and differentiate foot and leg lameness, so we can't say for sure if these swellings were causing lameness. In my opinion, straighter hock angles may have resulted in abnormal weight bearing angles and increased concussive forces on the leg joints, contributing in part to the lesions and lameness reported. If possible, when selecting your squad, avoid excessively straight legged bulls (leg score > 4).

- **Disease status:** When purchasing bulls, obtain a vaccination history for the bulls if it is known. Bulls should be vaccinated for the reproductive diseases vibriosis, BVDV, and leptospirosis. If bulls have not already had an antigen test for BVDV to determine if they are persistently infected, this should happen before they are introduced to existing stock on farm. To demonstrate that vibriosis is still a risk in dairy herds, we found bulls post-mating that returned a positive culture for *Campylobacter fetus venerealis* or *Campylobacter fetus fetus* in 2 of the 7 herds which didn't vaccinate for vibriosis.[13]

- **Safety:** The temperament and size of the bulls to be used are important selection criteria to consider. Primarily to ensure the safety of dairy staff, but also to prevent injury to other animals. Poor temperament and size (large bulls) were often quoted by farmers in the study as reasons for culling bulls.

Pre-season training

No world class sporting team would consider playing the first game of the competitive season without at least having a few months of training to prepare their bodies for the gruelling season to come. Recommendations to ensure that your bull team is in peak physical condition for the mating period include:

- **Have bulls on farm and grouped at least 2-3 months before joining:** In most seasonal or split calving herds this means that farmers need to consider preparing their bull team before the mating start date for AI. In my experience, it is not always easy to source mop-up bulls in seasonal calving areas and therefore it is important to think ahead. As seen in the results of the pre-mating BBSE a certain percentage of bulls (expect 20%) will fail, and this loss should be accounted for in advance (and not at the time of the BBSE) when putting the team together. In the aforementioned Tasmanian study, a large proportion of bulls were kept on agistment and only brought to the home farm within a month of bull mating start date, and this was considered an important risk factor for bull failure at the BBSE.[5] Grouping bulls together results in stress and fighting due to social hierarchical restructuring, the consequences of which may take bulls 2-3 months to recover from.

- **Aim to have them in moderate BCS:** Over-conditioned bulls may have reduced libido[17] and have more scrotal fat which can hinder testicular

thermoregulation and result in reduced semen quality.[18] Bulls in this study were generally in moderate body condition pre-mating and they maintained this condition during the mating period. This contrasts with beef bulls that have been shown to lose condition during mating.[10] The reason for the observed maintenance of condition in the study bulls may be related to workload, or possibly the fact that during mating dairy bulls are fed the same high quality pasture and roughage as the milking herd, and in some cases grain as well.

- **Vaccinate/BVDV antigen test:** As discussed earlier, it is important to protect bulls from reproductive diseases by vaccinating for vibriosis, BVDV, and leptospirosis prior to mating. If not already done so when purchasing or rearing bulls, they should have a BVDV antigen test to ensure that they are not persistently infected.

- **Nutrition:** In the final post-mating mixed effects regression model, bulls in herds which fed their bulls grain in the 3 months pre-mating were nearly 4 times as likely to fail the post-mating BBSE. Concentrate feeding can lead to laminitis,[19] which is a risk factor for many of the common foot lesions causing lameness in dairy cows.[20] Grain can be high in phosphorus, and diets with low calcium to phosphorus ratios have been shown to predispose to osteoarthritis and lameness.[21] Other vitamin and mineral imbalances have also been associated with osteochondrosis dissecans,[22] which if exacerbated by other factors such as walking on tracks, standing on concrete, and conformation may have contributed to the lesions and lameness seen in the study. Whilst further research is needed to understand the dietary factors affecting the soundness of dairy herd bulls, it is reasonable to recommend that bulls are fed a consistent diet, without excessive feeding of grain.

- **Footy boots:** Preventative 'blocking' is the practice of placing rubber blocks on the claws of all feet pre-mating to prevent lameness. Anecdotally farmers have claimed that they have fewer problems with lameness in their herd bulls after 'blocking'. We didn't observe a difference in lameness prevalence post-mating between 'blocked' and 'unblocked' bulls. These results may be due to small sample size and selection bias, given that the herds which 'blocked' bulls had a historically high prevalence of bull lameness. There was a trend for bulls that had been 'blocked' to have swollen joints post-mating, and for that reason, since the trial the clinic has changed to using thinner blocks. It might be the case that the presence of the block alters the normal weight bearing angles of the legs. The blocks had all fallen off by the end of the mating period, and would invariably have fallen off at different times, which may have resulted in a period of imbalance in the foot whilst there was only a block on one claw, resulting in abnormal stresses on the leg joints. In my opinion, these thinner blocks replicate a more normal hoof anatomy whilst still protecting the sole of the foot from damage. I think that 'blocking' has merit, and more and more herds in our region are taking up the practice despite its significant cost. A randomised controlled trial is required to properly assess the efficacy of preventative 'blocking' in preventing lameness.

Pre-season fitness test

A sporting coach can select a team of superstars, and train them well for the upcoming season, but they still need to be put through their paces to ensure that they are up to the challenge of the big game. Bulls have an equivalent to a pre-season fitness test: the BBSE. All bulls should undergo a thorough BBSE (+/- serving ability test, depending on facilities and willingness of farmer/veterinarian to do so) prior to each mating period. The results of this study and numerous others in beef bulls would suggest that a proportion of bulls will fail, many for reasons which are only detected during a BBSE.

Manage the game/season

Now that the season has begun, don't take your eye off the ball. You've done the hard work in preparing the team, now bring the season home and win the cup!

- **Manage the bench:** It is logical that bulls which are rested periodically during the mating period will last longer before

breaking down. Consequently, the InCalf bull resource recommends the following: “Run half the bull team with the herd at any one time, spell the rest, and rotate the group weekly”.[14] Our research did not obtain sufficient evidence to assess the strength of these recommendations, and further research in this area is warranted.

- **Watch the game:** As lameness and injuries are the most common cause of bull breakdown, early identification is critical to remove and replace bulls which may be underperforming. As a serving ability test is not routinely performed in the dairy industry, it is important that farmers observe bulls mating to ensure that they have adequate libido and are able to achieve intromission. Dairy farmers have an advantage over beef farmers in this respect, as most have close access to their herds twice daily. Most farmers in the study reported a high level of monitoring (88% watched the bulls mate and 100% observed the bulls for lameness and injuries).

Post-season analysis – Take-home messages:

- Dairy herd bulls should undergo a BBSE prior to mating
- Use young bulls (less than 4 years old)
- Consistency of nutrition is important
- Lameness prevention measures for dairy bulls (and cows) will increase bull longevity
- Vaccinate your bulls for vibriosis and BVDV

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TREATMENT AND MANAGEMENT OF DIGITAL DERMATITIS

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Introduction

Lameness in cattle and other intensively farmed animals is an ongoing challenge, with respect to animal welfare, the economics of agricultural production and the social license to farm. Bovine digital dermatitis (BDD) is a debilitating emerging infectious disease of the feet of cattle. Despite 40 years of research, the epidemiology of this disease is not well understood. After first being described in 1974,[i] the condition has spread all over the world and is reported as endemic in Southern and Western Europe, including the United Kingdom (UK), and the United States of America (USA). BDD is the most common cause of infectious lameness in dairy cattle in the Northern Hemisphere and represents approximately 25% of the total lameness cases in the UK.[ii] In these countries, it is an important animal health and welfare concern, as well as a significant impediment to production and profitability due to the detrimental effect on milk yield and reproductive performance and the significant costs associated with its treatment and control. Once a herd is infected, digital dermatitis appears to be impossible to eradicate where it has been studied in other countries.[iii] In countries where BDD has been established for sometime it has been found to cause non-healing lesions, termed 'toe necrosis' when it invades the hoof wall and pedal bone of cattle.[iv] In the UK, it is now believed the spirochaetes responsible for BDD are causing a new, severe, debilitating disease called Contagious Ovine Digital Dermatitis (CODD).[v]

It is believed by some researchers and veterinarians in the field, that the incidence of BDD is increasing in Australian dairy farms, and there is a need for veterinarians to become active in advising clients on early detection, prompt treatment and management of this emerging disease.

Bovine digital dermatitis

Presentation

Digital dermatitis is characterised by inflammation of the skin, mostly in the region of the bulb of the heel and coronet of the hoof, but can also cause erosion of the horn tissue in more chronic cases.[vi][vii][viii] The different presentations of digital dermatitis have led to the classification of these stages; M0, healthy skin; M1, early stage, skin defect < 2 cm diameter; M2, acute active ulcerative lesion >2cm diameter; M3, healing stage, lesion covered with scab-like material; M4, chronic stage, that may be dyskeratotic (mostly thickened epithelium) or proliferative or both.[ix][xxi]

Digital dermatitis is believed to be caused by Treponemes, a type of spirochaete[xii] after the skin barrier is weakened due to mechanical or chemical irritation.

What is known about incidence in Australia?

In Australia, a survey was reported in 2008 that did not detect any evidence of spirochaetes being involved in similar looking lesions in Australia.[xiii] However, since then, a single case has been diagnosed via laboratory testing in the Macalister Irrigation District in Victoria.[xiv] Many anecdotal reports exist and it is a high priority to study the prevalence of the disease.

Individual cow treatment

Different treatment strategies have been reviewed but the most favoured is topical antibiotic treatments on individual cows after cleaning the skin with water and/or functional foot trim.[xv] The use of topical oxytetracycline has been found to be effective at reducing lesion and pain scores in active lesions.[xvi] [xvii]A foot trim is important in removing eroded heel that may reveal more digital dermatitis and may improve the penetration of the treatment.[xviii] Early detection and treatment is essential for limiting the spread of the disease. Establishing a periodic assessment of milking cows such as BDD pen walks or whilst milking have been suggested to speed diagnosis and reduce the spread from these active lesions.[xix] Oxytetracycline in an aerosol form (Norbrook Alamycin Aerosol 33mg/g oxytetracycline (as hydrochloride)) is registered for topical use and if used daily for at least 5 days can be effective at reducing the clinical signs. Many lesions are

chronic and although treatment may seem effective, research overseas has suggested that these recur due to the causative organism becoming encysted, deeper in the skin. More aggressive treatment of BDD lesions is now not supported, as it will provoke the cows' immune system and ensure spirochaetes become encysted.

In a well-designed BDD treatment study, cows with active digital dermatitis were assigned to different treatment groups; one group received a tetracycline hydrochloride (TH) paste (175ml propylene glycol, 175ml vinegar, 150g of tetracycline hydrochloride) applied with a paintbrush, another group received a powdered form of TH held in place by a gauze swab and bandage along with a control group.[xx] The TH paste treatment group was as effective as TH bandage group. These results therefore eliminate the need for the use of bandages in the treatment of this condition, which can often lead to further problems if not managed well.

Management

A manageable level of BDD can be achieved through an integrated control strategy as advocated by researchers at the University of Wisconsin. The management of digital dermatitis is contentious but early topical treatment of infected animals is important along with the use of footbaths, with either formalin or copper sulfate, in both lactating, dry and youngstock groups to control the spread of infection. Replacement youngstock that are carrying an infection prior to calving have a five-fold increased chance of suffering a lameness event due to BDD compared to those that do not have the condition prior to calving.[xxi] Other aspects critical to control are improving foot hygiene through reducing sources of mud and manure, optimising lying times, improving skin integrity and increasing foot angle through the corrective foot trimming.

Use of footbaths

The aim of using a footbath for cattle is to control infectious causes of lameness and specifically to control M4 BDD lesions; M2 lesions should be treated topically. It is fortunate to have the benefit of many years of international research in design of footbaths, chemicals used and frequency required to reduce the impact of BDD in herds. A leading UK veterinarian, Roger Blowey, has termed BDD as 'mastitis of the foot' and advocates the use of twice daily footbaths with formalin 2-8% for cows as the necessary disinfectant applied to cows similar to post milking teat disinfection.[xxii] Other groups have suggested frequencies of daily or as required depending on hygiene scoring of the animals; for example, very dirty animals will require daily footbaths.

Products

Although many different products have been tested, there are two common compounds used in footbaths to reduce the spread of digital dermatitis; 1-5% formalin and 2.5-5% copper sulphate. Both have their disadvantages; formalin is a respiratory and conjunctival irritant and carcinogen and copper sulphate can have negative environmental effects if not disposed of properly.

Design

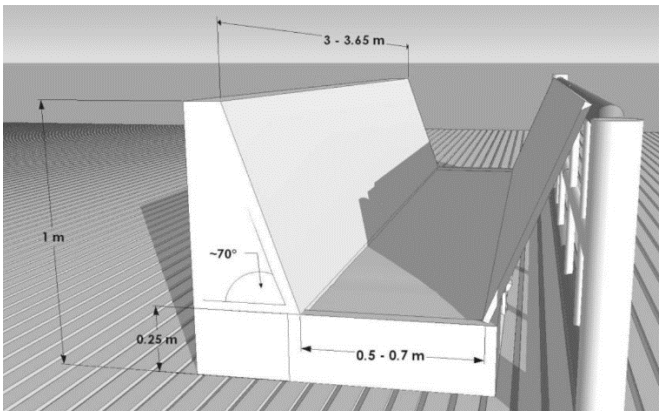
The success of a footbath depends on the transfer of the chemical to the foot and the contact time with the foot. Ideally the feet should be cleaned prior to using the footbath and afterwards allowed to dry for the chemicals to work.[xxiii] Although different baths have been described there is increasing consensus that a design described by Cook et al (2012) achieves the aims of

- not greatly affecting cow flow
- maximising the number of foot 'dunks' per pass
- reducing the total fluid volume required for medicating
- reducing the amount of manure contamination (see figure below).

Roger Blowey and Neil Chesterton however, advocate for wider design to allow two cows to pass believing that this optimises cow flow and reduces faecal contamination.[xxiv]

Footbaths are usually located at the exit of the dairy. They need to be far enough away to allow efficient cow flow and so that formalin vapour is not detected by people in the dairy. A non-slip surface within the footbath is important to minimize slipping and adequate drainage is essential. It is recommended that footbath solutions be changed every day, or after 200 cows, whichever comes first.[xxvxxvi] Innovative custom made footbaths that are a closed system

automatically delivering the right amount of chemical, counting the number of cows that pass, emptying, cleaning and refilling again are an excellent way to minimise the effects of formalin but also to ensure that emptying occurs in a timely manner.[xxvii]



Picture 1. Footbath design to optimise cow flow and the number of foot immersions while minimising bath volume[xxviii]

Other BDD management strategies

Prolonged moisture to the hoof area and reduced access to air are risk factors for disease establishment and transmission.[xxixxxxxxxi] Striving to create dry areas for cattle, reducing access to slurry and reducing standing times have been important in reducing the incidence on affected farms. There is also growing agreement between researchers of the importance of control strategies for replacement and dry stock, including the use of footbaths's in these groups.[xxxii]

Conclusion

Digital Dermatitis is a debilitating and costly disease which is endemic in many other dairying nations and is an important biosecurity risk to Australian dairy farmers. Although only an emerging disease in Australia, it is important that farmers, stock agents and veterinarians are alert to its detection and spread and are able to quickly and accurately diagnose then treat the condition. With the increasing intensity of dairying in Australia, there is a great need for further research into BDD, due to the threat of its increasing virulence, within cattle and across species, which has occurred in other countries.

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HOW TO TREAT CONSISTENTLY – ESTABLISHING FARM PROTOCOLS

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Introduction

Over the last few decades, the primary responsibility for the treatment of common diseases of individual animals in Australian livestock production systems has moved away from the veterinary practitioner to herd managers or employed farm staff. Some of the reasons that have led to this shift include a significant increase in herd size; a decrease in the value of the individual animal and an increase in the number of trained staff on both dairy and beef properties.

The use of treatment protocols for common diseases creates a consistency amongst all farm personnel and if planned correctly can result in superior animal health outcomes. Cattle operations are a perfect place for the development of treatment protocols as there are often multiple employees assigned to treating animals which could lead to inconsistency if individual preference was maintained. The veterinarian has a major role in the development of these protocols to ensure that amongst other areas: animals are being diagnosed appropriately, correct treatments are provided and withhold periods are appropriately calculated and enforced.

The following paper aims to outline some of the steps and thought processes of the author when creating on-farm treatment protocols which can be implemented on commercial operations.

How are treatment protocols useful for veterinary practices?

In the introduction I outlined some of the reasons why treatment protocols are important for livestock producers but these same protocols are also very important for veterinary practices.

Firstly, they are extremely useful for standardising the way individual veterinarians treat common ambulatory problems such as retained foetal membranes or respiratory disease. Providing a consistent treatment regime helps to eliminate conflicting treatment recommendations from different veterinarians which can be a major problem in large dairy and beef veterinary practices. Treatment protocols also provide confidence to newer graduates who may not be able to draw from previous experience in treating these common ailments and find it difficult when confronted by an opinion of what should be used by a producer.

Secondly, formulating on-farm treatment protocols combined with producer education and treatment outcome monitoring is an excellent tool for helping to establish a valid veterinary/client/patient relationship for the responsible dispensing and administration of scheduled therapeutics.

Lastly, the provision of treatment protocols that include withholding periods and any OH&S (Occupational health and safety) recommendations can also be used to avoid any potential legal problem which may be encountered when a residue violation or inappropriate administration has occurred. A drug which should regularly be dispensed with a treatment warning are the synthetic prostaglandins due to their potential to cause severe asthma or abortion in pregnant humans. A written treatment protocol doesn't replace the requirement to have withholding periods clearly defined on the label of the drug but it serves as an extra reminder to the client.

Determine which diseases require treatment protocols

The common treatment protocols developed for on-farm use include: mastitis, metritis, retained foetal membranes, pneumonia, lameness, neonatal calf diarrhoea and "downer cows". From the authors experience these diseases would make up greater than 90% of the medical problems that occur on dairy and beef properties. These diseases are suitable for treatment protocols as they are relatively easy to recognise (mild cases of pneumonia is probably the outlier).

Formulating the treatment protocol

The next important step in the process is identifying an individual who is willing and able to undertake the task. This individual may be in charge of a team of veterinarians who are given individual disease protocols to develop but ultimately this individual will be required to have

a final say when consensus doesn't occur. Creating treatment protocols can be a lengthy process which is often not cash generating for a business thus there is the potential for the task to be a seen as less important than clinical work.

Creating consensus amongst all clinicians is often the most difficult part of determining what therapeutics are included in each of the treatment protocols. The reality is that there is often not one plan which is perfectly agreeable to all clinicians. A common disagreement often occurs with which antibiotic is most suitable for each disease. This can be further complicated by the fact that certain producers will prefer to use a certain antibiotic due to previous perceived success or failure. An example of this is the treatment of respiratory disease in young calves. Suitable antimicrobials for this disease include oxytetracycline, tilmicosin, tulathromycin or ceftiofur. One way to cater for the differing views of both client and veterinarian is by to have a generalised treatment protocol for the disease which has multiple antibiotic options which are then individualised for each farm. When this occurs it is important the clinician consults with the on-farm treatment protocol folder when performing ambulatory work on the property to provide the consistent treatment regime. As a general rule, avoid the use of extra-label drug usage unless withholding periods are clearly stated and there has been additional staff training.

In recent years there have been several journal articles dedicated to the treatment of various cattle diseases which rely on evidence based medicine (e.g. "Evidence Based Medicine for the Bovine Veterinarian" *Veterinary Clinics of North America-Food Animal Practice* vol. 28 issue 1 Mar 2012). One of the difficulties in relying on evidence based medicine for the formulation of treatment protocols is that often the evidence does not exist to include or exclude all aspects of a proposed treatment regime. Where evidence is available, the dosages used are often significantly higher than recommended label dosage thus requiring the veterinarian to alter the withholding period. In the author's opinion, when formulating treatment protocols for livestock, science and research is mainly used for exclusion of inappropriate therapeutics rather than choosing the most appropriate. Basic pharmacology can be used to identify when a certain therapeutic is inappropriate for a particular condition, for example the use of penicillin in the treatment of bovine mastitis due to the inability of the antibiotic to cross the blood/milk barrier. The factors which guide the choice of a suitable (or more poignantly an unsuitable) therapeutic agent from a pharmacological point of view are beyond the scope of this paper.

More experienced clinicians often play an important role in deciding which treatments are recommended by a veterinary practice. In many cases, these decisions are extremely sound but experience should not necessarily outweigh the ideas of newer graduates who may be more up-to-date with enhance treatment regimes. An example from the clinic that the author works in is the use of non-steroidal anti-inflammatory as an ancillary treatment to many common diseases. From a review of records from the author's clinic, there is a greater propensity for newer graduates to use anti-inflammatories for diseases such as metritis and pneumonia when compared to the more experienced clinicians. Therefore, if one was to formulate protocols for these two diseases in the author's practice it is likely that the more experienced veterinarians would advise that it was not necessary to use non-steroidal anti-inflammatories.

A significant factor which often guides which therapeutics are included in a treatment protocol is the cost of the protocol. The direct costs of the treatment protocol are the cost of the therapeutics themselves. Indirect costs can relate to the length of withhold required, staff time to perform the task and equipment required to administer them (for instance, oral pumps for administration of large volume drenches). An example of using simple economics to decide which treatment to use is shown in the table below. The table shows two products which could be used for the treatment of a mild cases of clinical mastitis. If we assume that both products give equal cure rates than it is clear that Product B would be a better choice.

Intramammary	**Treatment regime**	**WHP**	**Drug cost**	**Withhold cost** (25L per day @ 40c/L)	**Total Cost**
Product A	1 tube every 48hrs for 3 treatments	96hrs	$14.70	$100.00	$114.70
Product B	1 tube every 12hrs for 3 treatments	72hrs	$17.85	$45.00	$62.85

The key piece of information in the previous example is that there was an assumption that cure rates were equal. For some of the antibiotics, cure rates can be obtained from the manufacturer or from published research in order to compare cure rates between products but for many diseases this information is not available. A cost-benefit analysis is often used in agriculture to determine if it is economically viable to perform a particular action. Using this form of analysis when deciding on what should be included in treatment protocols (particular when ancillary therapeutics are considered) is very difficult as there is simply not enough real world or research data to make this decision. On larger properties which have a large number of clinical cases, one can compare the treatment outcomes using different combinations of therapeutics to determine the success of different treatment protocols and thus compare which is more economical (see section "Review of protocols").

What are the key components of a treatment protocol?

The document needs to include the following:

- Clinical signs of the disease to be treated (clear and concise).
- Name of the drug(s) to be used
- Site of administration
- Volume or dose required
- Frequency of administration
- Duration
- How treated animals will be identified
- Withholding periods

It is essential that a treatment protocol is clear and where possible does not allow for any grey areas.

How to present the protocol?

The presentation of the protocol will vary dependant on the level of information that needs to be supplied and the autonomy of the individual treating the cattle. For instance, it is appropriate to have significant amount of detail in a training manual that is supplied to all new employees and is mainly used as a reference guide in an office. This same type of document would be completely unsuitable in a working milking parlour for a junior milker who quickly needs to know which intramammary tube is required to treat a case of mastitis.

A common form of treatment protocol is a Standard Operating Procedure (SOP) document. An SOP is a clear step by step set of instructions that are designed to provide a detailed description on how a task should be performed. These documents are very common in many industries and are perfectly suited to both dairy and intensive beef operations. The detail provided in SOP's are specifically designed to eliminate confusion or indecision on why or how a specific disease is to be treated. SOP's should be provided to all new employees and should be accessible at all times.

The most common form in most dairies is a simplified version which is often no more than one page and on display in the milking parlour. The wall chart will often only include the name of the disease, the therapeutics to be used and the assigned withholding periods.

In recent years there has been considerable change in the workforce on many dairy and beef operations. There has been a significant increase in the number of overseas labour units from multiple different countries who often have limited English skills. In the United States it is common to find treatment protocols translated into Spanish but this is unlikely to occur here due to the number of languages required. Overcoming the language barrier can be difficult and where possible visual explanations should be used.

Implementing the protocols on commercial operations

Recognition of disease

It is important that a case definition is created for each of the common diseases for which a treatment protocol has been established. The case definition needs to be clear and unambiguous as possible. For instance, the primary clinical signs associated with retained foetal membranes that would require treatment could include:

- Membranes still present 24 hours after calving
- Foul, fetid discharge from vulva
- Temperature greater than 39.5
- Other secondary clinical signs could include: poor rumen fill, low milk production, inappetence

The skill level of each of the employees should be assessed in order to establish the level of training required for each of the treatment protocols. The ability to quickly detect common diseases on beef and dairy properties differs significantly between diseases.

Training staff in the responsible use of drugs

Training staff members in how to responsibly administer, handle and dispose of therapeutics is often a step which is neglected by employers and advisors. There is an assumption that staff know what they are doing which in many cases the author has found to be incorrect. The two major areas where training is often required is the hygienic administration and correct storage of drugs. The Australian Cattle Veterinarians have developed an online resource which can be used for this purpose (http://www.ava.com.au/cattle).

Tailoring the protocol to the property – creating buy in

Even the best protocol will fail if it is not followed properly. It is important that all people who are required to follow the protocol feel that they have some ownership of the protocol. The easiest way to do this is to involve the staff members in the initial part of the process when you are deciding what treatments should be included for each of the diseases.

Review of protocols

Once a protocol has been established and is being used on a property the next part of the process is to assess the success of the treatment protocol through data collection and analysis. The most common way to assess treatment success is to look at either/or case resolution, case recurrence and case fatality. The metric which is used will vary dependant on the disease for instance case resolution and reoccurrence would both be important for mild clinical mastitis but case fatality would be unlikely. It is also important to recognise that treatment success rates don't necessarily reflect an inappropriate treatment regime has been used. A good example of this would be the treatment of an animal with chronic respiratory disease. In most of these cases all forms of treatment will fail and this can only be prevented by improved early detection and treatment. Continued poor treatment resolution should be investigated thoroughly by the herd veterinarian.

The easiest way to collect and subsequently analyse data is through the use of on-farm health and management database or software program. Complete, accurate and reliable records are the cornerstone of monitoring treatment protocols. An important part of the development of a treatment protocol is establishing how the data is going to be recorded and entered into the on-farm management software. Countless hours can be spent sifting through data which may not be useful or relevant if this doesn't occur. For example, one of the more common mistakes is recording every daily treatment of a mastitis event which when analysed will artificially elevate the treatment reoccurrence or failure rate. Whilst data collection is extremely important, it is even more important that the data is analysed and used for decision making. The quickest was to stop a producer from collecting and recording treatment data is to not perform any analysis or provide any useful feedback.

Once we are happy that a protocol is being used correctly and desired treatment outcomes are being achieve it is also important to monitor for any drift in adherence to the protocol or identify if there are areas that the protocol could be improved. One of the greatest risks for protocol drift is when new staff are hired that have pre-existing ideas on how animals should be treated from previous employment/education. It is really important that all new staff are inducted into how and why the protocols have been established and how important it is that consistency is maintained. On the flip side, new staff may have pre-existing ideas that could improve how an existing protocol is implemented or have the desire to develop new protocols for other areas. Protocol drift can either be monitored systemically via examination of clinical records and drug purchases or through random spot checks from herd management.

Discussions regarding changes to a protocol are positive for the ongoing use of treatment protocols, but altering the protocols without the mutual agreement of the stakeholders should not be tolerated. All changes to a protocol should be based on the most objective measures available.

Conclusion

The development and use of farm treatment protocols can be a valuable service for the clientele of a veterinary business. Whilst as veterinarians we tend to focus on the importance of science in regard to developing treatment protocols, in order to achieve success, it is far more important to provide a protocol that is easily followed by all staff and regularly reviewed.

TREATING CALVES BETTER – THE BENEFITS OF FEEDING MILK

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Introduction

Over the past decade a body of research has evolved investigating the effects of increasing the volume of milk or milk components available to reared calves. Traditional dairy calf rearing approaches have focused on feeding a set volume of milk per day, usually about 10-12% of bodyweight. Low milk feeding systems promote early consumption of concentrates hence maximising rumen development and allowing for earlier weaning. In contrast calves that have access to higher milk volume or milk component intakes have reported greater weight gains, exhibit more natural behaviour and are less susceptible to disease during the rearing period. It has also been suggested that milk components may have the ability to turn on genes, extending this effect into adult life with greater bodyweights and reduced age at first calving and greater milk yield in the first lactation. However not all the literature is in agreement with these findings and conclusions. As part of Dairy Australia's Rearing Health Calves program an attempt has been made to develop an overview of this rapidly changing topic in dairy calf nutrition and provide guidance to the industry on whether calves can indeed be treated better by feeding more milk.

Low level milk feeding systems

For most of this and the last century, the overriding objective for modern calf feeding programs has been to focus on strategies that facilitate early weaning and an easy transition from a liquid to solid diet. Early in the 20th century the key drivers for such an approach were identified as increasing income from the sale of whole milk and reducing labour and overall feed costs.[1,2] Even in times when costs were low, it was clear that calves needed to be reared as cost effectively as possible to maximise farm profitability.

Feeding strategies that encourage the early intake of concentrates have generally been the cornerstone of any early weaning program. The restriction of milk intake to the level of 10% which is still commonly used today was identified as a tool to encourage calves to intake greater amounts of non-milk feed stuffs as early as 1924.[2] In this study calves were weaned onto a gruel as a transition to a more substantial diet of grain and hay. Calves reportedly achieved average daily gains (ADG) of 0.58-0.68kg/day, comparable to many of our herds today. Of interest was the higher ADG was seen in calves that were fed carrots in addition to the gruel, grain and hay stimulating the discussion of the value of carrots for calf growth.

Weaning practices in the US & Australia

The ability to survive solely on a solid diet is dependent on the presence of a fully developed and functional rumen. It is now well known that the digestion of concentrates (grains/pellets/muesli) produce more of the volatile fatty acid butyrate which promotes development of the rumen wall.

One of the key indicators that weaning can be undertaken is the demonstrative ability of the calf to consistently consume sufficient levels of good quality concentrates. Other indicators commonly used by farmers for weaning such as weight, age and visual assessment may not be accurate indicators. In Australia, farmers tend to use a combination of these observations in the weaning decision making process. Calves are also regularly reared in group housing systems making it challenging to accurately predict individual concentrate intake for calves. Other factors such as surplus milk availability, space, infrastructure and weather can heavily influence the decision of age at weaning.

Although early weaning is often spoken of as a focus of our calf rearing systems, it is not commonly practiced. US calf rearing operations appear to have a greater focus on weaning calves at around 6 weeks of age. Early weaning significantly reduces the cost of calf rearing, reduces labour requirements and potentially decreases the risk of exposure to disease. Conversely it places calves at far greater risk of being weaned before they have developed sufficient ruminal function to maintain growth rates on a milk free diet. Contrast this to the Australian dairy industry where it is more common to wean at closer to 12 weeks of age. This approach clearly takes pressure off the process but it is also a significant difference between US and Australian calf rearing strategies. This is an important consideration when any comparisons are made between

Australia and the US - Australian calves are more likely to be reared on milk for twice the length of time as US calves.

Low level milk feeding vs *Mother Nature*

Although broadly achieving the objectives set out in the early 20's, low level milk feeding systems have failed to address other industry concerns in regards high growth rates, scours and calf disease. Increasing focus on animal welfare and decreasing profits drives greater attention to find solutions to these concerns. One such approach has been to reflect back on how *Mother Nature* intended calves to be reared.

Low level milk feeding systems involve feeding a maximum of four litres of milk per day regardless of bodyweight through out the rearing period and ad lib concentrates from the first week of life. US systems tend to introduce fibre at the time of weaning (5-6 weeks) whereas in Australian systems a source of fibre usually is introduced a few weeks after birth.

Low level of milk feeding, amounting to about 10-12% bodyweight, is in stark contrast to the situation under where a calf left on its mother. In his review of the topic, Khan concluded calves left to nurse on the cow consume approximately 6-12 kg/day of milk in several feds per day.[3] This amounts to at least double what is supplied through our modern calf rearing systems. Taking into consideration that multiple feedings are replaced by single or twice daily feedings, it is clearly a very different way to rear calves than what happens when calves are left on cows. Growth rates of up to 1kg have been reported on calves fed *ad lib* milk, well above growth rates seen on modern dairy farms.[4] Calves fed at lower rates struggle to met industry set targets. Low level milk feeding in practice equates to feeding 4 litres daily for a 40kg calf. The allowable weight gain from this milk diet at 20ºC is around 0.4kg/day but at 5ºC it is only 0.2kg/day - not an unfamiliar temperature in many of the South Eastern dairy regions during winter.

Fortunately, the time that calves derive their complete nutritional needs solely from a liquid diet is usually short. Most calves begin to consume concentrates by 2 weeks of age and by 3 weeks can reach levels that significantly contribute to their daily energy and protein requirements. The Australian practice of early introduction of roughage in the form of hay may also contribute to the higher growth rates seen in practice. The highest starter intake was achieved in calves when offered a variety of chopped roughage and silage from 2 weeks of age.[5] Interestingly this effect was not seen when chopped lucerne was offered. Presumably its high palatability contributed to its preferential intake over concentrates. These findings challenge US concerns that early roughage introduction reduces the intake of concentrates and extends the weaning period.

Clearly milk alone, fed at 10-12% of bodyweight will not achieve the growth rates needed to met industry targets. High levels of growth seen on calves fed *ad lib* has lead to research investigating the role higher volumes of milk or milk components can play to achieve better health and growth outcomes. Newborn calves are clearly physiologically designed to grow on milk based proteins and carbohydrates. Achieving high average daily gains is the key to higher weaning rates and a greater likelihood of producing heifers capable of reaching target weights. The benefits of well grown heifers are widely known in relation to gains in fertility and lifetime production. This approach to calf rearing has become known as accelerated calf rearing but terms such as enhanced and intensified are also commonly used to describe this "new" concept in calf rearing.

Fatty udder syndrome

A long standing concern about increasing growth rates in heifer calves is the potential for excess fat deposition at the cost of udder parenchymal tissue. This condition, sometimes referred to as *fatty udder syndrome* can decrease lifetime milk production in affected animals. The udder is considered to undertake three separate growth periods consisting of two distinct types of growth, isometric and allometric. Isometric growth is where the udder tissue is developing at the same rate as general body growth and allometric growth is where the growth rate is faster than the rest of the body. The isometric phases of growth occur twice, once from birth to 12 weeks and lastly from about puberty (1 year of age) to the end of development. The allometric growth phase occurs in the period between the two isometric phases.

Nutrition can stimulate greater rates of lean gain during the pre-weaning period, without impairing future mammary development. Hence high average daily gains through the first isometric

growth phase do not seem to have a detrimental effect on subsequent lactation performance. This is in contrast to findings associated with high growth rates occurring during the allometric phase from weaning to puberty.[6] A meta-analysis study reported that milk and protein yield can be maximised at growth rates of around 0.8kg/day for Holstein heifers.[7] There would appear to be great scope to encourage high average daily gains during this period of birth to weaning or the first isometric phase of udder development without adversely risking potential lifetime milk production.

Epigenetic effects of milk feeding

It has also been suggested that the practice of accelerated calf rearing may also be doing more than just improving average daily weight gains. It is believed by some that enhanced nutrient intake from either milk or milk replacer during the pre-weaning phase can alter milk yield potential of the calf through an epigenetic interaction.[8] It is postulated that components in milk may upregulate genes that influence milk production, in effect turning them on and setting up the heifer for a potential high lifetime milk production. The thinking behind this idea is supported by the *Lactocrine Hypothesis* where colostrum or milk borne factors have been postulated to influence tissue development through epigenetic regulation in a variety of species including humans.[9]

The idea of well grown out heifers doing better in the herd is not new. InCalf research has clearly demonstrated this in Australian herds. This study has shown that heifers with higher weights pre-calving calve sooner than lighter heifers. It has also identified that liveweight at first calving is positively correlated with better subsequent fertility. Larger heifers also produce more milk as they can consume greater quantities of dry matter, maintain body condition by use of body reserves and compete better with mature animals in the herd. What is being suggested by the supporters of the accelerated calf rearing approach is that by the feeding of greater volumes of milk or milk components, particularly in the pre-weaning period, heifers are genetically primed to do better in the herd and this effect is not just related to greater bodyweight.

A large scale study was undertaken in the US to investigate the relationship between milk replacer, growth rates pre- and post-weaning and subsequent lactation performance.[8] They found a linear relationship with average daily gain (ADG) and first lactation milk yield where for every 1kg of pre-weaning ADG, milk yield increased by 1113kg in the first lactation and this effect was nearly 3x greater if just prepubertal ADG was studied. It was concluded that this pre-weaning effect was ultimately the result of epigenetic programing. The same authors subsequently analysed a total of 12 data sets by meta-analysis to assess the effect of pre-weaning nutrient intake from milk or milk replacer on first lactation and lifetime productivity.[10] Many of these published studies have found positive but not significant gains from enhanced pre-weaning nutrition. They found that treated calves (i.e. received higher milk or milk replacer volumes) had an overall milk yield response of 435+/- 117kg/lactation over non-treated calves. Similarly, they showed for every kg of pre-weaning ADG, first lactation yield increased by 1550kg and calves fed for greater pre-weaning ADG were 2 times more likely to have greater first lactation milk yields. They concluded that these findings indicate a relationship with pre-weaning nutrition and the phenotypic expression of first and most likely life-time lactation milk yield, speculating an epigenetic or programming effect.

Increased pre-weaning nutrition via liquid means can be achieved via feeding larger volumes of milk, milk replacer or milk components per feed or by increasing the number of feeds permitted by the calf over the day. In Australia, there is a longstanding industry concern about increased rates of diarrhoea associated with high volume milk feeding. Such concerns have been difficult to support scientifically despite the “in-practice” reality suggesting that high volume feeding diarrhoea is likely multifactorial in its causation. In environments where pathogen counts are significant, high volume feeding may simply introduce more pathogens. It may also provide more suitable intestinal environmental conditions for diarrhoea to occur, or pathogens to multiple due to changes in digestive characteristics such as transit time, volume of liquid contents or pH alterations. It is not unexpected though that the water content of faeces of calves fed greater amounts of liquid feeds will increase yet the dry matter component of the faeces may actually remain the same so maybe the definition of diarrhoea needs to be addressed. Larger volumes over the day can be successfully delivered using automatic calf feeding systems with no noticeable increase in

scours replicating the situation when calves are left on cows.

A recent Australian studied investigated the effect of feeding calves milk with added milk replacer powder commonly referred to as "fortified milk" versus the common low level milk feeding approach.[11] This enabled no change in milk feeding volumes (i.e. 2 litres twice daily) but allowed a higher intake of energy and protein. A total of 150gms of milk replacer powder (25% protein, 20% fat) was added to 2 litres of whole milk and fed twice daily. Calves feed this mixture from birth to weaning had significantly higher ADG over the duration of the trial when compared to the control group (0.82kg/day vs. 0.71kg/day), no detrimental health outcomes but at a cost of nearly 3 times the conventional low volume rearing system ($93.27 vs $33.97). The average total concentrate consumption over the entire trial period for the fortified group was about half of the control calves. It is important that prior to implementing such an approach in Australia consideration should be given to determining the optimum milk supplementation rate and regulating the intake to achieve growth rates without incurring poor health outcomes. Over fortification of milk can lead to osmotic diarrhoea as can over consumption. Regulating intake of fortified milk in group housing, mass feeding systems is not easily achieved and such an approach is more suited to an automated or individual feeding setup.

The great unknown at present is whether the lactation response is a function of total nutrient intake or if factors in milk are responsible for the enhanced milk yield, the so called epigenetic effect. Is milk necessary to achieve this potential increase in productivity or can it be obtained by just better management of solid feed intake? There have been some very public scientific criticisms of this belief that milk borne factors are responsible for the lactation effects seen.[12] The critics claim calves on accelerated feeding programs must continue to be fed and managed well to maintain the growth advantage gained before weaning and to capitalise on that growth by conceiving and calving at an earlier age. In a peer reviewed report[13] that scrutinised the 12 reported studies used by Soberon and Van Amburgh [10] in their meta-analysis they identified that 5 of the databases used were from non-peer reviewed studies putting at question the scientific value of the findings. Of the remaining 7 peer reviewed studies, 3 were comparing the effect of whole milk at high feeding levels versus poor quality milk replacers so were not truly investigating a single variable and none of the 4 remaining studies of milk replacer versus milk replacer individually showed significant effects on first lactation milk yield. A recently published study further supports their views. In this prospective cohort study, 152 Holstein calves were either fed a total of 4 or 8 l of whole milk daily (twice daily feedings). As expected the average daily gain up to day 56 was greater for the calves on the higher volumes of whole milk but the difference between the two groups was no longer present at 3 and 12 months. They reported no effects of whole milk feeding on age of first calving or first lactation production.

The ability for calves reared on accelerated programs to be safely early weaned at 56 days is also in question. Due to lower intakes of concentrates, rumen development can be compromised leaving these calves vulnerable to growth slumps following weaning. A recently published study has shown that post-weaning digestion is lower than optimal and contributes to lower post-weaning growth in calves fed aggressive compared with conventional or moderate milk replacer programs.[14] Obviously this finding has a much greater impact on early weaning then later weaning programs.

Milk feeding levels and disease

The intestinal tract of the newborn calf is highly permeable during the first two weeks of life. This occurs as a consequence of the process of passive transfer of immunity required to provide the calf with immunoglobulins for the first 4-6 weeks of its life. Farms with poor calf milk hygiene practices run the risk of increasing disease rates in calves by either bacteria interfering with immunoglobulin G absorption and/or exposing calves to greater numbers of potential pathogens. Clearly high volume milk feeding practices can increase disease rates by amplifying this risk if farms are not carefully assessed for suitability for implementation.

To offset the concern of increased cost of rearing calves with enhanced nutrition, benefits in health are often promoted. Conventional rearing systems are often described as "controlled starving" of calves with poor growth rates and associated poor health outcomes. In defence of conventional systems, calves are still able to gain weight despite the lower volume of milk on offer. Clearly this gain may be lower in thermo-negative environments and prior to

developing the ability to derive significant nutritional support from concentrates but are there clearly defined health benefits from higher planes of nutrition pre-weaning? Unfortunately, the answer is unclear.

A few studies have investigated the role of level of nutrition on immune function or more specifically leukocyte function and though some have shown an effect others have not, so no clear conclusions can be drawn.[15-16] There are only 2 published studies that investigate the interaction of plane of nutrition and an enteric disease challenge during the first few weeks of life. The first found that calves fed higher amounts of milk replacer and exposed to a challenge with coronavirus before weaning had greater average daily gain but also an increased incidence of diarrhoea and required more veterinary attention compared to calves on a lower plane of nutrition.[17] In contrast the other challenge study using *Cryptosporidium parvum* concluded that calves fed a higher plane of nutrition maintained hydration and had faster resolution of diarrhoea in additional to better growth.[18] Clearly, high plane of nutrition calves had better average daily gains in both studies as expected but differed in how they responded to an enteric disease challenge.

A series of recent reports from researchers at Texas Tech University have started to add more information to this puzzle. In the first of series of studies they found that neither neutrophil oxidative burst nor whole blood bactericidal capacity was influenced by level of milk replacer fed (high versus low) during the pre-weaned period among either Jersey or Holstein calves.[19] A subsequent study reported that Holstein calves fed the low level of nutrition had elevated neutrophil oxidative burst and increased activity of the neutrophil surface adhesion molecule L-selectin compared to calves fed a high level of milk replacer but only during the pre-weaned period.[20] No significant differences were seen after weaning commenced at 45 days. They hypothesised that the heightened immune ability may relate to behaviour associated with low volume feeding. Calves fed low levels of liquid feed show increased non-nutritive suckling behaviour due to hunger and non-satiety. Microorganisms consumed due to this behaviour may lead to an early priming of the gut. Non-nutritive suckling clearly places any calf at risk of pathogen exposure and is a negative outcome of low volume feeding systems.

A third study looked at Jersey calves fed high and low levels of milk replacer and challenged with an opportunistic bacteria pre-weaning. It found that despite the high level fed calves maintaining an average daily gain differential, they had higher rectal temperatures and more significant inflammatory responses.[21] Examination of distal ilium at day 24 of life showed these same calves tended to have greater, though not significant, villus heights and total mucosa mass. They hypothesised that this trend to have greater intestinal surface area may increase the likelihood of pathogen attack purely by there being more available sites for pathogens to attach.

Investigating the longer term effects of high volume feeding, a final study has been reported that looked at a post-weaning respiratory challenge in calves either feed high or low levels of milk replacer.[22] It found that calves fed low levels of milk replacer mounted more significant inflammatory reactions and were more greatly affected by the disease than calves on high levels of milk replacer feeding.

From this work, it appears that pre weaning calves fed higher levels of milk or milk replacer may be at increased risk of enteric disease due to greater surface area of the gastro-intestinal tract but show better immune function and lesser susceptibility to infectious disease post-weaning. Calves on low level feeding may also be at greater risk due to non-nutritive suckling behaviour. In summary any immune function benefit from high level of milk or milk component feeding may only be realised post-weaning.

Can calves be treated better by feeding more milk?

Clearly any calf management practice that translates to high average daily weight gains from birth to first calving will ultimately be of benefit to both the calf and the producer. The goal to improving average daily gains is understanding that it is clearly a multifactorial process. Addressing pre-calving care of the dam, provision of a clean, comfortable environment and optimum management of colostrum nutrition, health and weaning should be the foundation of any calf management system. Even regular, gentle handling has recently been shown to result in a small but significant increase in average daily gain,[23] so we shouldn't discount how we interact with calves either.

Any attempt at improving average daily gains should take into account the cost benefit of such an approach and the ability of the enterprise to successfully implement the change successfully. Accelerated calf rearing approaches are not suited to every farm. Farms most suitable for implementation would be those with proven hygienic calf milk handling and an ability to regulate individual liquid intake (i.e. individual pens or locking head bails).

The period of growth that is most suitable for manipulation appears to be the first 12 weeks of life. Increasing the intake of milk solids either by increasing milk volumes or fortification of milk will clearly stimulate high average daily weight gains though just focusing on early uptake and ongoing consumption of concentrates may produce a similar effect. Caution will need to be exercised to ensure that daily concentrate intake levels do not suffer as a result of any increased milk or milk components consumption. There may be a window of opportunity for using an approach such as fortified milk in the first 2-4 weeks of life to initiate good growth but then still allow early transition to a concentrate diet, minimising costs and ensuring a successful weaning process. Clearly this advice would alter if some large prospective cohort studies are published that clearly indicate feeding of higher levels of milk or milk components alone is the key to changing the phenotypic outcome of a calf by switching on her genes.

Taking into consideration current scientific research it is not currently possible to claim that high levels of milk feeding during the pre-weaning period will reduce either the incidence of disease or significantly improve the long-term productivity and fertility of a calf through just an epigenetic-type effect. Calves can be treated better by managing them to ensure that they grow. The focus should be on optimising growth during the pre-weaning period. Any growth advantages achieved need to be maintained throughout the post weaning period by close attention to nutritional demands and disease management. Only by ensuring that heifers meet industry targets will long term gains be realised.

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PERCUTANEOUS FETOTOMY TECHNIQUES – A CUT ABOVE THE REST

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If vaginal delivery by traction is not possible without danger to the dam or fetus, the options of caesarean section or fetotomy need to be considered. Two basic types of fetotomy have been described in the veterinary literature. Subcutaneous fetotomy is performed with chisels and hooks with fetal parts being removed while leaving the fetal skin to protect the genital tract and serve as points of traction. Percutaneous fetotomy is performed with a fetotome and wire saw with sections of the fetus progressively removed. While the techniques of subcutaneous fetotomy have been in development since the early 18th century, the technique of percutaneous fetotomy has only been in development since the 1930's. The main driver to the progress of the percutaneous fetotomy technique was the development of a suitable fetotome and cutting wire. This document will describe the process of percutaneous fetotomy.

EQUIPMENT REQUIRED FOR PERCUTANEOUS FETOTOMY

Training and proper equipment are essential for performing a fetotomy. The basic equipment required is shown in Figure 1 and described subsequently.

Figure 1 - Basic equipment required to complete a percutaneous fetotomy. From bottom left in clockwise direction is the Krey hook with attached rope, Utrecht fetotome with wire threader inserted through one barrel, calving chains, a roll of fetotomy wire, fetotomy handles and pliers. A wire introducer can be included in the kit, although a calving chain can be used for this purpose in most cases.

Fetotome. A two-barrel fetotome with a smooth head made from hardened steel is used. A hardened head is required to prevent the saw wire from cutting into the head during cuts parallel to, or at right angles to, the fetotome head. The fetotome also has a notched plate near the handle that allows fixation of an obstetrical chain under tension to maintain proper position of the instrument relative to the fetus. The ability to attach the chain to the fetotome is considered **essential** for efficient completion of the percutaneous fetotomy procedure. Utrecht and Thygussen fetotomes are recommended.

Threader and Brush. A flexible shaft with an eye on one end is used to thread the saw wire through the barrels of the fetotome. Many threaders will have a brush on the opposite end, which is necessary to clear the barrels of lubricant and debris.

Wire Saw Handles. Several types are available and the choice depends on the preference of the operator. Those that incorporate a spool for saw wire are convenient, but the type selected should provide a positive grip on the saw wire, be easy to attach, not kink the wire, and be comfortable for the assistant while sawing.

Krey Hook. A Krey hook with an obstetric chain or rope attached is necessary to anchor the fetotome to the fetus while making several cuts and to extract fetal parts after they have been separated.

Saw Wire Introducer. A curved wire introducer facilitates passage of the saw wire around fetal parts. If a wire introducer is not available, the fetotomy wire can be attached to an obstetric chain to assist in passing the wire around the fetal hindquarters.

Saw Wire. A good grade of saw wire (fetotomy wire) should be selected. Poorly manufactured or improperly maintained (rusted) saw wire breaks easily. Approximately 5 m of wire is required to thread the fetotome. The Utrecht fetotome is approximately one metre long and serves as a good field guide to drawing out a suitable amount of wire. To reduce the chance of breaking the wire while sawing, it is advantageous to rotate the wire through the fetotome as each cut is completed and replace the wire after each fetotomy procedure.

RESTRAINT AND ANAESTHESIA FOR FETOTOMY

Clinicians develop personal preference as to whether they prefer to perform a fetotomy in the standing or laterally recumbent animal. Both positions have advantages and disadvantages and sometimes there is no choice due to available facilities or the condition of the cow. Initially, many operators find fetotomy is most easily performed with the cow standing in an area that allows adequate space behind for manipulation of the instrument and saw wire. If the dam is recumbent and cannot be induced to rise, elevation of the hindquarters may be helpful initially. But as the procedure progresses, access to the fetus becomes easier with the cow in complete lateral recumbency.

Epidural anesthesia is indicated in nearly all cases to relieve pain as well as straining. Where available, clenbuterol should also be administered to relax the myometrium and reduce the risk of uterine rupture. Tranquilisation may be indicated in some cases, but general anaesthesia is only rarely necessary. The administration of antibiotics should be considered prior to the commencement of the procedure.

LUBRICATION

A generous amount of lubricant (~ 5 litres) is required during a fetotomy to protect the genital tract of the dam as well as the hands and arms of the operator. Petroleum-based lubricants are preferred to water-soluble types because of their proclivity to cling to tissues and resist dilution by fetal fluids. However, water-based lubricants may be utilised provided high volumes are regularly applied. A stomach-tube and pump aid the application.

ASSISTANCE

At least one, and preferably two, assistants are required to perform a fetotomy. If one assistant is available, the clinician covers the head of the fetotome with one hand and maintains the position of the fetotome with the other hand while the assistant saws. If two assistants are available, the clinician covers the fetotome head while one assistant maintains position of the instrument and the other saws. Sawing should begin with slow, short strokes with only light pressure on the wire. After the wire is seated beneath the fetal skin, strokes of the wire can be lengthened with more pressure applied. Tension on the saw wire should not be relaxed during the cutting procedure as the saw wire may tangle and break. It is also preferable not to stop the cutting procedure until it is complete, otherwise the wire may become trapped and damaged.

INDICATIONS FOR FETOTOMY

Percutaneous fetotomy is not a substitute for cesarean section but is indicated in certain cases of dystocia. The decision guidelines based on “diagnostic traction” (whether the fetlocks can be delivered a hands-breadth outside the vulva for cranial presentation, or the hocks to the level of the vulva for caudal presentation) are a useful aid to determining when fetotomy is indicated. Importantly, fetotomy should not be used as a last resort after the application of excessive traction when the dam and operator are exhausted and the birth canal traumatised. A decision to perform a fetotomy should be made promptly after it becomes obvious that delivery by traction is not justified. In general, fetotomy is useful to relieve dystocia caused by fetopelvic disproportion, pathologic enlargement of the fetus (fetal giantism), incomplete cervical dilatation, fetal malposture and malpresentation, and fetal monsters. Fetotomy is not useful when the birth canal is obstructed or reduced in size, as in uterine torsion.

Percutaneous fetotomy is given primary consideration to relieve dystocia when the fetus is dead and cesarean section is given primary consideration when the fetus is living. Exceptions may be indicated when the value of the dam and her future reproductive ability and milk production are greater than the value of the fetus.

Either partial or complete fetotomy may be required to relieve dystocia. A complete fetotomy is usually required to deliver oversized fetuses. A complete fetotomy following the Utrecht guidelines requires a maximum of seven (but usually five) cuts in cranial presentation and six or seven cuts in caudal presentation. The amputated fetal parts should be no larger than can be extracted using only light traction.

Partial fetotomy is indicated in cases of fetal malposture. The offending appendage can be quickly amputated, after which the fetus can be delivered by traction. It is necessary to be aware that in protracted cases, the uterus can be tightly contracted around the fetus and the repulsion necessary to allow mutation may result in uterine rupture. Partial fetotomy is also useful in cases of a dead fetus in hiplock that cannot be relieved without excessive traction.

Amputation of the Head

Removal of the head allows easier access to the forelimbs for amputation or mutation. A loop of saw wire is passed over the head until it rests immediately caudal to the ears. The head of the fetotome is introduced alongside the head and positioned between the mandibles and caudal to their posterior borders. If this area is not accessible, the fetotome can be positioned on the lateral surface of the face with the fetotome head caudal to the ramus of the mandible (Figure 2). After the head has been amputated, it is extracted. Subsequent traction to the fetus can be applied via chains on the legs. In addition, the Krey hook can be fixed to the exposed cervical vertebrae when needed.

Figure 2 - One method of placing the fetotome and wire in preparation for removal of the head. In some cases, this single cut will create enough room to allow mutation and completion of a vaginal delivery.

Amputation of the Forelimbs

Before being amputated, the forelimbs must be extended and the distal portion of the limb protruded from the vulva. An obstetric chain is fixed to the limb and the chain passed through the loop of the wire saw from above to below (Figure 3).

Figure 3 - Prior to placing the fetotomy wire for removal of the forelimb, the obstetrical chain attached to the limb is passed through the wire loop from above. The fetotomy wire loop is then placed between the claws of the calf.

The saw wire loop is then placed between the claws of the forefoot to temporarily anchor it. This process helps to ensure the wire is not twisted during placement of the fetotome. The fetotome is passed alongside the lateral surface of the limb until the head rests near the middle of the scapula. After moderate traction to extend the leg, the obstetric chain is anchored to the fetotome. The saw wire loop is removed from the interdigital space and while the assistant applies slight tension on the wire handles, the loop of saw wire is moved up the medial surface of the limb until it lies medial to the scapula in the axillary space. Care should be taken to ensure the wire loop goes completely over the elbow. The calving chain is then detached from the fetotome and the instrument advanced more deeply into the uterus until the head lies 3 to 5 cm dorsal and caudal to the scapula. Traction is applied to the obstetric chain to fully extend the joints of the limb and the chain is again anchored to the fetotome. The final placement is shown in Figure 4.

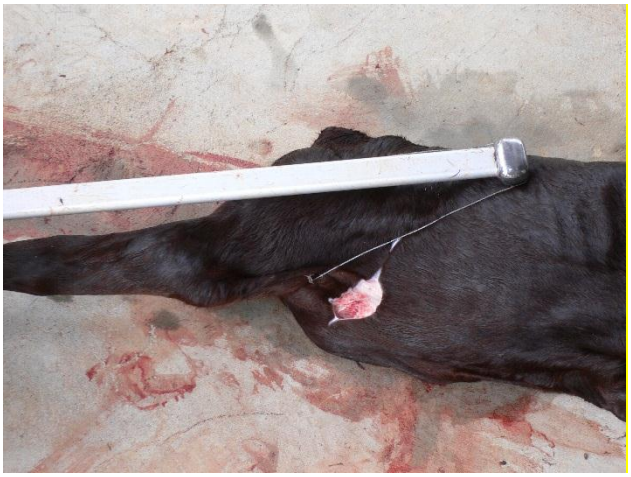

Figure 4 - Placement of the saw wire and head of the fetotome for an acute angled cut to remove the forelimb.

The obstetrician covers the head of the fetotome with a hand and the limb is amputated. When the fetotome has been positioned correctly, the forelimb along with the entire scapula is amputated and the diameter of the fetus reduced. Common errors include cutting through the shaft of the humerus or through the shoulder joint, which results in formation of sharp bony fragments and eliminates the leg as a point of traction without reducing the diameter of the fetus. The second forelimb is amputated similarly, but in most cases the procedure will be completed more easily because of the space made available by removal of the first leg. If a small portion of scapula remains after the cut, it can be manually detached from the musculature and removed.

Transverse Division of the Trunk

Two transverse cuts are made to section the fetal trunk. The first includes the neck and the cranial portion of the chest, while the second cut is made through the lumbar region. In some cases, the fetus can be partially delivered after the forelimbs have been amputated and the first transverse cut may not be necessary. In preparation for amputation of the cranial portion of the chest, the Krey hook is fixed to the exposed cervical vertebrae. The chain attached to the hook is passed from above to below through the loop of saw wire as previously described for the forelimb. While the loop of saw wire is held externally, the fetotome is passed along the dorsolateral surface of the fetal chest until the head is near the area where the scapulas were attached. The chain from the Krey hook is then anchored to the fetotome and while moderate tension is applied to the saw wire, the loop is positioned around the fetal thorax. The final position of the saw wire is approximately at the middle of the sternum and the loop is at a right angle to the fetotome. Before sawing, check the position of the saw wire to ensure that the fetotome was not rotated while being positioned and that the saw wires were not crossed. The fetotome tends to move up and down when sawing begins, but this motion can be minimised by pushing the fetotome against the fetus and by using short strokes with the saw wire when commencing the cut. After it has been amputated, this portion of the chest is narrow and in most cases extracted easily by traction on the Krey hook.

Amputation of the remaining portion of the chest is conducted similarly. With the Krey hook anchored to the thoracic vertebrae, the head of the fetotome is positioned on the dorsolateral surface of the fetus immediately caudal to the last rib so that the saw wire loop is at right angles to the fetotome. In the case of a large fetus, this portion of the chest may be too large to permit safe extraction. The diameter can be quickly reduced by separating the ribs from their attachment to the vertebrae. To achieve this, the saw wire is removed from one of the fetotome tubes and attached to an introducer. When attaching the wire to an introducer, care is required to ensure the loose end of the knot is long enough to prevent it from acting as a cutting device, which could occur if left too short. After puncturing the diaphragm of the fetus with a finger, the introducer and saw wire are passed through the lumen of the thorax. The hand is withdrawn from the thoracic cavity, reintroduced down the lateral aspect of the thorax, the introducer and wire retrieved, and the fetotome rethreaded. The head of the fetotome is placed near the vertebral body and the saw wire positioned to sever the ribs near their vertebral attachment. After the ribs have been separated, the thorax can be collapsed and withdrawn.

A longitudinal division of the fetus separates the hindquarters and reduces them in size for delivery as shown in Figure 5. Using an introducer, the saw wire is passed over the dorsal aspect of the pelvis and the introducer retrieved between the hindlimbs. The fetal hindpart can be divided into equal parts by placing the head of the fetotome against the lumbar vertebra or into unequal parts by placing the head of the fetotome on the lateral surface of the fetus cranial to the hip joint.

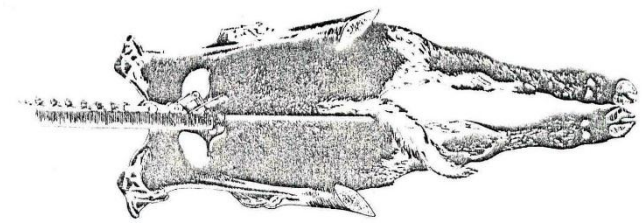

Figure 5 - Sectioning the pelvis to relieve hip-lock.

Hiplock

A partial fetotomy can be used to expeditiously relieve dystocia caused by hiplock whether the condition arises spontaneously or is the result of inadequate rotation during delivery by traction. The forepart of the fetus is first removed by a transverse cut between the last rib and the pelvis. The hindpart of the fetus is then divided and delivered as described previously.

Fetotomy in Caudal Presentation

Amputation of the Hindlimb

An obstetric chain is attached to the limb to be amputated and passed from above to below through the loop of saw wire at the head of the fetotome. In the manner similar to that described for amputation of a forelimb, the wire loop is temporarily anchored between the claws and the fetotome introduced along the lateral surface of the limb and advanced until the head rests in the area of the greater trochanter of the femur. After the obstetric chain is anchored to the fetotome, the saw wire is removed from the interdigital space and advanced up the medial surface of the limb until it lies medial and cranial to the stifle joint. Traction is then placed on the obstetric chain to extend all the joints, and the head of the fetotome is further advanced until it rests dorsal to the greater trochanter of the femur. The chain is anchored to the fetotome and the limb amputated. If the fetus is very large, the second limb can be amputated similarly or the trunk can be divided transversely with the second limb still attached if it appears that delivery of the severed portion will be possible.

Transverse Division of the Trunk

The first transverse division of the trunk is made between the pelvis and the last rib. If one hindlimb has been removed, the cut is begun by positioning the fetotome in the manner described for amputation of a hindlimb. However, the fetotome is positioned farther forward on the fetus and the saw wire loop is positioned caudal to the last rib. This cut results in delivery of the remaining hindlimb along with the fetal pelvis. If both hindlimbs have been removed, the pelvis is secured with a Krey hook, the saw wire loop is positioned transversely around the fetus, and the pelvis is removed.

The second transverse division of the trunk is made by positioning the head of the fetotome on the dorsolateral surface of the fetus immediately caudal to the scapulae. The saw wire loop is then positioned at a right angle to the fetotome so as to divide the fetus at approximately the middle of the sternum. If the rib cage cannot be safely extracted, it can be divided and collapsed as described for fetotomy in cranial presentation.

Division of the Forepart

The remaining fetal forepart can be reduced in size by amputation of each forelimb separately or by diagonal division of the forepart. Forelimbs are amputated by passing an introducer and saw wire dorsally between the neck and the limb and retrieving them ventrally, positioning the wire between the elbow joint and the chest. After the fetotome has been rethreaded, its head is positioned in a space that has been bluntly dissected between the scapula and the thorax. If diagonal division of the forepart is selected, the saw wire is positioned similarly but the head of the fetotome is placed on the lateral surface of the opposite scapula. The larger portion, consisting of the cranial thorax and one limb, is delivered first followed by delivery of the remaining forelimb and head.

Fetotomy in Abnormal Posture

Displacement of the Head

Access to the flexed neck can be facilitated by amputation of the forelimb on the side opposite that to which the head is displaced. The saw wire attached to an introducer is then positioned between the neck and the body of the fetus. After the fetotome is rethreaded, the fetotome head is positioned as close to the thorax as possible and the neck is severed.

Carpal Flexion

If dystocia due to carpal flexion cannot be corrected by repulsion and mutation, the dystocia can be easily relieved by partial fetotomy. If the head prevents easy access to the flexed carpus, it is amputated first. Then with the use of an introducer, the saw wire is placed around the flexed carpus and the fetotome is threaded. The head of the fetotome is positioned against the distal portion of the carpal joint as shown in Figure 6. The limb is amputated and

the distal portion removed. An obstetric chain can then be anchored proximal to the carpal joint for delivery by traction.

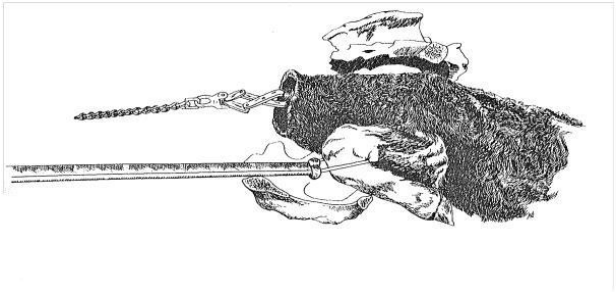

Figure 6 - Fetotomy approach for an impacted carpal flexion. Note the wire placement distal to the joint. Image courtesy Dr CJ Bierschwal

Hock Flexion

Amputation of the limb immediately distal to the hock reduces the danger of uterine rupture that may accompany attempts to repel the fetus and mutate the malposture in protracted cases. The saw wire is threaded around the limb and positioned distal to the hock joint. An obstetric chain can be anchored to the limb proximal to the hock for delivery by traction.

Hip Flexion

Using an introducer, the saw wire is passed over the dorsum of the fetus and directed between the limb and body. The introducer is retrieved ventrally and the fetotome threaded. Prior to amputating the limb, the head of the fetotome is placed against the fetal ischium. In bilateral cases, it may be necessary to amputate the second limb if the malposture cannot be safely mutated.

Fetotomy for Delivery of Abnormal Fetuses

Fetal monsters are occasionally encountered as causes of dystocia in cattle. The variety of configurations will challenge the resourcefulness of the obstetrician, but fetotomy is often preferable to cesarean section.

Schistosoma Reflexus

If the fetus is presented with its viscera exposed, the fetal organs can be removed after a thorough examination to ensure that rupture of the uterus has not resulted in prolapse of maternal organs. An attempt is made to encircle the fetus with the saw wire and divide the trunk near the point of deviation. Frequently the portions can then be delivered. If not, they can be reduced in size by further divisions. Sharp bone fragments frequently result and the dam's birth canal should be protected when the fetal segments are delivered. Some abnormal fetuses are presented with three or more limbs and the head in the maternal pelvis. In these cases, an attempt is made to amputate the most accessible limb in the most expeditious manner, followed by further section of the fetus at the discretion of the clinician.

Perosomus Elumbus

These fetuses are usually not oversized and the forepart of the fetus is delivered spontaneously. Difficulty is encountered when the operator attempts to complete the delivery because the hindlimbs are often ankylosed and distorted. Attempts to withdraw the fetus by traction may result in perforation of the uterus. Delivery can be accomplished by a partial fetotomy similar to that described for resolving cases of hiplock.

Modified Fetotomy

A modification of the Utrecht method for complete fetotomy has been described that reduces the number of cuts required and is applicable in cases in which the fetus is not excessively oversized.

Cranial Presentation

The head is first amputated by encircling the neck with the saw wire. The head of the fetotome is then positioned dorsal to the fetal scapula in a manner similar to that described for amputation of a forelimb except that the saw wire is positioned between the stump of the neck and the opposite forelimb. This diagonal cut results in amputation of one forelimb, the neck, and a portion of the thorax. The resulting opening in the thorax permits evisceration of the thoracic and abdominal cavities, which further reduces the size of the fetus. In addition, the size of emphysematous fetuses is reduced by the escape of gas following breach of the body cavities. Traction on the remaining forelimb is then continued until delivery is complete or until the size of the fetal hindpart obstructs progress. If necessary, the fetal pelvis is sectioned in the manner described for resolution of hiplock.

Caudal Presentation

The first hindlimb is amputated in the manner described by the Utrecht guidelines. The fetus is then reduced in size by evisceration. If size of the fetus is not reduced sufficiently to permit delivery, a transverse cut is made through the thorax caudal to the scapula. The ribs can be severed from their attachment to the vertebrae and the thorax collapsed if necessary. A final cut is made obliquely through the remaining forepart

of the fetus. One section is composed of a forelimb and most of the thorax and the other is composed of the head, neck, and remaining forelimb.

AFTERCARE FOLLOWING FETOTOMY

In most cases, the dam requires less care following fetotomy than following caesarean. Upon completion of a fetotomy, the uterus is routinely lavaged with warm (42 to 45□C) water to which is added a small amount of a nonirritating disinfectant or salt. Approximately 5 to 10 L are pumped into the uterine cavity through a stomach tube and then syphoned out. The procedure is repeated until fetal tissue and lubricant have been removed and the efflux is clear. Dystocia is a common antecedent to secondary uterine inertia, and treatment with an ecbolic agent such as oxytocin is often indicated. Systemic antibiotics are frequently indicated, especially in protracted cases. Other supportive therapy such as intravenous fluids or calcium should be administered as required. Anecdote suggests that the uterus will contract and involute more effectively if the cow is allowed to exercise, in contrast to being confined in a stall. Fertility and milk production are typically higher following fetotomy than following caesarean.

REPRODUCTIVE CONDITIONS OF THE PREPARTUM PERIOD IN CATTLE

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Introduction

A discussion on treating prepartum disease conditions in cattle warrants a quick overview of the main structures involved in the disease processes. Specifically, the structure and function of the fetoplacental unit. This paper will provide an overview of the fetoplacental unit, followed by the aetiology and treatment of prepartum conditions such as vaginal prolapse, hydrops amnion, hydrops allantois, mummified fetus and macerated fetus

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Fetal and placental development

Placental Development and Structure

There are **two** components to the placenta, the **fetal membranes** and the **maternal endometrium**. The structural elements of the mammalian placenta consist of the amnion, yolk sac, chorion, allantois, and umbilical vessels. Only the structures of direct significance to prepartum conditions will be discussed here.

Amnion

The amnion develops from ectoderm and avascular mesoderm within 13 to 16 days after fertilisation in ruminants. It is a double-walled sac that completely surrounds the embryo except at the umbilical ring. The amnion provides a fluid filled environment in which the embryo floats and develops in a state of weightlessness. Fluid arises from the **upper respiratory tract** of the fetus in addition to the **amniochorion** and, importantly, from fetal **urine**. During normal gestation the volume of amniotic fluid increases from around 40 mL at 53 days of gestation through to 2.5 L at about 225 days of gestation and up to about 6 L at term.

Allantois

The allantois is an outgrowth of the embryonic hindgut and is continuous with the urinary bladder. The outer allantoic layer becomes richly supplied with blood vessels connected to the fetal heart by umbilical veins. This vascular layer expands into the extra-embryonic coelom and fuses with the chorion to form the **chorioallantois**, the main fetal membrane. The inner layer, which is mainly devoid of blood vessels, is adjacent to the amnion. The urachus is a narrow tube, located within the umbilical cord which connects the allantoic sac with the fetal bladder. The allantoic cavity is filled mainly with clear, watery, amber fluid that is derived from **urine** from the fetal kidneys, **but also** fluid coming directly from the **chorioallantoic membrane** (Dickerson and McCance 1957). During normal gestation the volume of allantoic fluid increases from around 250 mL at 60 to days of gestation through to about 8 L at 225 days of gestation and about 19 L at term.

Chorion

The chorion is formed by elongation of the trophoblast, which becomes invested with a layer of endoderm followed by the lateral development of the mesoderm. The chorion is closely applied to the allantois to form the chorioallantois and a smaller connection with the amnion where it is described as the chorioamnion.

Chorioallantois

This fusion of the chorion and the allantois is a highly vascular structure that is in intimate contact with the endometrium. Fusion of the allantois to the chorion (and thus the formation of the chorioallantois) is complete by approximately the 28th day of gestation (Hafez and Rajakoski 1966). The chorioallantois is designed for metabolic interchange of gases, nutrients and waste between the fetal and maternal circulations. Importantly, the chorioallantois and the chorioamnion appear to act as filters, modifying and contributing to the fluids they contain.

Fetal Membrane Fluid Dynamics

Fetal urine is an important input into both amniotic and allantoic fluid. Yet, both amniotic and allantoic fluids differ substantially in composition from that of fetal urine. Specifically, the membranes seem to be processing the fluid. One major variation is the low osmotic pressure of the fetal fluid, in the vicinity of 150 mOsm/kg. This suggests the fluids are being hydrated (water added) by the membranes. This and other changes suggest that the allantoic or amniotic cavities should not be exclusively regarded as a reservoir for fetal urine. One interesting finding is that if fetal urine is drained to the exterior by fetal cystocentesis, the volumes of both the amniotic and allantoic fluids decline to virtually zero over a period of several days to a week – that is – quite rapidly. High osmotic pressure (particularly high sodium concentrations) within the allantoic fluid has been associated with hydrops allantois, with the maternal blood in these cases showing hyponatraemia. This suggests sodium sequestration within the allantoic fluid in hydrops allantois cases. There is suggestion that fetal adrenal insufficiency may play a role in some hydrops cases (Wintour *et al.* 1986), possibly in association with perturbations to brain development. Also, in diabetic humans, the increase in blood glucose translates into increased glucose concentrations within the amnion, and interestingly there is an increased occurrence of hydrops amnion in human diabetics. In animal models, increases in amniotic fluid glucose concentration have led to increased amniotic fluid volumes.

Placental Development

The fetal side of the bovine placenta forms from the fusion of the allantois and chorion. This is represented diagrammatically in **Error! Reference source not found.**.

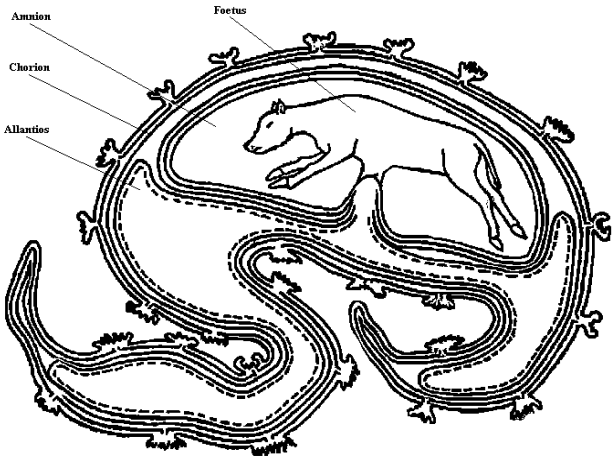

Figure 1 - A diagrammatic representation of the bovine fetal membranes. Where the chorion and allantois are apposed, it is referred to as the chorioallantois. Where the chorion and amnion are apposed, it is referred to as the chorioamnion.

The placenta reaches physiological maturity between 90 and 120 days of gestation and near maximal size is reached by mid gestation.

Further increases in physiologic function of the placenta are achieved by enhanced interdigitating of the microvilli within the placentome. Importantly, if there is attachment of villi to areas other than at caruncular sites, a condition known as **adventitial placentation** occurs, which can lead to an inflammatory response and increased fluid production.

There is evidence to suggest that many factors prior to and during gestation can influence the ultimate size and function of the placenta (Robinson *et al.* 1995). These include genetic influences, maternal nutrition prior to and after conception, the dam body size, ambient temperature and season.

Oestrogens During Gestation

Oestrogens are necessary to enhance the effects of progesterone in the early stages of gestation. Later on, they help to produce udder development, relaxation of pelvic ligaments, cervical relaxation, and to sensitise the uterus to oxytocin and prostaglandins (Roberts 1986). Oestrone, oestradiol 17-β and oestradiol 17-α are the major oestrogens synthesised by the fetoplacental unit. The combined plasma concentration of these three oestrogens is referred to as total plasma oestrogen (Erb *et al.* 1982).

Significant oestrone sulfate secretion from the feto-placental unit commences from about day 50 of gestation. The secretion of total oestrogens increases steadily from 30 to 500 pg/mL between 60 and 100 days of gestation, then increases rapidly between 100 and 150 days to reach concentrations of around 3000 pg/mL. From 150 to 240 days there is only a slight increase, followed by a further sharp rise in the last week of pregnancy (Eissa and El-Belely 1990).

Concentrations of plasma total oestrogens start to increase sharply from one week before calving until concentrations of over 4000 pg/mL are reached on the day of parturition. Within 12 hours after parturition, and removal of the fetoplacental unit, concentrations have returned to less than 200 pg/mL (Eissa and El-Belely 1990).

The **concentration of plasma total oestrogens** during late gestation has been noted to **vary with** calf birth weight, heifer nutrition, cotyledonary weight and the season of the year (Rasby *et al.* 1990; Erb *et al.* 1982). These studies have found oestrogen concentrations to be higher with increased calf birth weight, twins and summer calvings. Heifers fed to maintain a thin body condition during mid-gestation had higher plasma oestrogen concentrations when measured at 8.5 months of gestation compared to heifers in good condition (Rasby *et al.* 1990). This was supported by another study where it was found that the plasma concentrations of oestradiol in fat cows were significantly lower than that of normal-conditioned cows during weeks 4 to 10 prior to calving (Zhang 1989).

In summary, increased plasma oestrogen concentrations during the last half of gestation tends to be associated with higher calf birth weights, twins, lower body condition scores during gestation, higher ambient temperatures and increased cotyledonary weights.

The significance? – Oestrogens cause hydration of collagen and interstitial oedema. Anything causing increased plasma oestrogen concentrations may increase the chance of vaginal prolapse in susceptible animals.

Corticosteroids During Gestation

Plasma concentrations of corticosteroids peak approximately six days prior to calving and again on the day of parturition (Eissa and El-Belely 1990). These authors believe the abrupt increase in plasma corticosteroids six days prior to parturition is significant in the mechanism of initiation of parturition. The increase in corticosteroid levels is also significant in **increasing** the production of placental **oestrogens** due to the activation of placental enzymes. The peak in plasma oestrogens approximately one week prior to parturition reflects this corticosteroid peak.

Placental Lactogen and Prolactin During Gestation

Ruminant placental lactogens are members of the somatotrophin-prolactin gene family. They are synthesised by trophectodermal binucleate cells and secreted into both the fetal and maternal circulations. Information on the synthesis and secretion of placental lactogen is limited, but evidence suggests that nutrition and/or body condition can influence synthesis or secretion (Rasby *et al.* 1990; Gluckman and Barry 1988).

The plasma concentration of placental lactogen in the fetus peaks at approximately 25 ng/mL in mid-gestation then declines to low levels of approximately 5 ng/mL one to two weeks prior to parturition. In the maternal circulation placental lactogen is detectable from the fourth month of gestation, but in cattle the levels remain low (compared to sheep and goats) at around one ng/mL for the duration of pregnancy (Rasby et al 1990).

Placental lactogen can bind to fetal somatotrophin (growth hormone) receptors and has been shown to stimulate glycogen synthesis and IGF production in fetal rat tissue. It is also possible that it may stimulate fetal growth by redirecting maternal metabolism in favour of transplacental glucose transport to the fetus. Concentrations of placental lactogen in maternal plasma have been found to be greater in thin cows compared to moderate body condition cows (Rasby et al 1990). However, the effect of nutritional influence at specific times during gestation is still unknown.

In summary, increased concentrations of maternal placental lactogen tend to be associated with higher calf birth weights, lower body condition scores during gestation, twins, and higher placental weights. As noted above, some of these parameters affected by placental lactogen may subsequently influence plasma oestrogen concentrations.

VAGINAL PROLAPSE

Aetiology/Pathogenesis

Vaginal prolapse can be considered a toxic effect of long term exposure to oestrogen….but with genetic and environmental influences. High concentrations of oestrogens are produced by the feto-placental unit during mid to late gestation in the bovine. Oestrogens affect the reproductive tract by causing **interstitial oedema** and the **hydration of collagen**. This results in slackening of the pelvic ligaments, which support the caudal reproductive tract, and also causes thickening of vaginal and vestibular tissue due to the oedema. The combination of relaxed pelvic ligaments, interstitial oedema, and increasing abdominal pressure from the growing fetus, lead to vaginal prolapse in **susceptible** individuals. From the preceding discussion on oestrogen, corticosteroids and

placental lactogen it is useful to note the effects of nutrition and the environment on plasma oestrogen concentrations during gestation. There is also the potential effect of the **bull genetics** on **fetal membrane** endocrinology. Oestrogen production from the fetoplacental unit, coupled with the increasing size of the gravid uterus explains why the majority of vaginal prolapse cases will be in mid to late gestation cows. However, the physical effects of oestrogens also explain why cows ingesting phyto-oestrogens, cows with cystic ovarian disease, or embryo transfer donors that are repeatedly super-stimulated, may also succumb to vaginal prolapse. Additionally, there tends to be a breed predisposition and within any breed some individuals are more susceptible than others. This indicates that there is an **hereditary component** to the disease, with boss Indicus X boss Taurus breeds being implicated as having a high susceptibility. Interestingly, there are suggestions in the literature that bulls with excessive preputial skin may sire females more susceptible to vaginal prolapse (Smit 1994). Importantly, the heritable and repeatable nature of vaginal prolapse strongly favours a treat-then-cull approach to management of this disease. While there is varying anecdote regarding other factors that contribute to seasonal increases in the occurrence of vaginal prolapse, there is theoretical support (see earlier discussion on endocrinology during gestation) to suggest there would be increased risk of vaginal prolapse in cows of lower body condition, fed high roughage diets, entering the third trimester of gestation in the warmer months.

Initially, a small amount of vaginal tissue is exposed at the vulval lips. As this tissue becomes sunburned, dried and irritated, further swelling will occur and eventually the cow will feel uncomfortable and start using abdominal muscles to strain and push further vaginal tissue outside the vulval lips. The prolapse can be graded and this process can assist with informing treatment and management considerations:

Grade 1 - the prolapse is still intermittent, occurring most commonly when the cow is lying down. There is little secondary damage visible beyond mucosal erythema and oedema. Reduction and fixation is usually readily performed with little concern for complications other than recurrence.

Grade 2 - the vaginal prolapse is continuous with the urinary bladder sometimes inverted into the prolapse cavity. There may be some superficial mucosal erosion but no exposure of the cervix. Reduction and fixation is usually readily performed with little concern for complications other than recurrence.

Grade 3 - there is continuous vaginal prolapse with exposure of the cervix. Often the urinary bladder is inverted into the prolapse cavity. Commonly there is a varying amount of liquefaction of the cervical mucus seal, leading to possible contamination of the uterus and fetal membranes. If the gestation is left to progress there is risk of placentitis, fetal death, abortion and metritis, masceration, or fetal emphysema. There may be varying degrees of superficial mucosal erosion. In these cases, initial treatment followed by a managed (usually induced) parturition is recommended. The delivery can be via assisted traction, or elective caesarean. In closely monitored cows (for example embryo transfer recipients), parenteral antibiotics, non-steroidal anti-inflammatories and progesterone supplementation (for example 150 mg of progesterone in oil every 2nd day) may be used to assist with maintaining the pregnancy to term.

Grade 4 - this grading covers the criteria of grades 2 and 3, but also includes the presence of infection or necrosis of the vaginal wall. In the earlier stages of this grading (as a guide, from approximately 36 to 72 hours of continuous exposure), the condition is described as subacute and generally with careful cleansing and lubrication the prolapse can be replaced into the vaginal vault. In more chronic cases as fibrosis progresses the stage will be reached where the vagina can no longer be replaced within the vaginal vault.

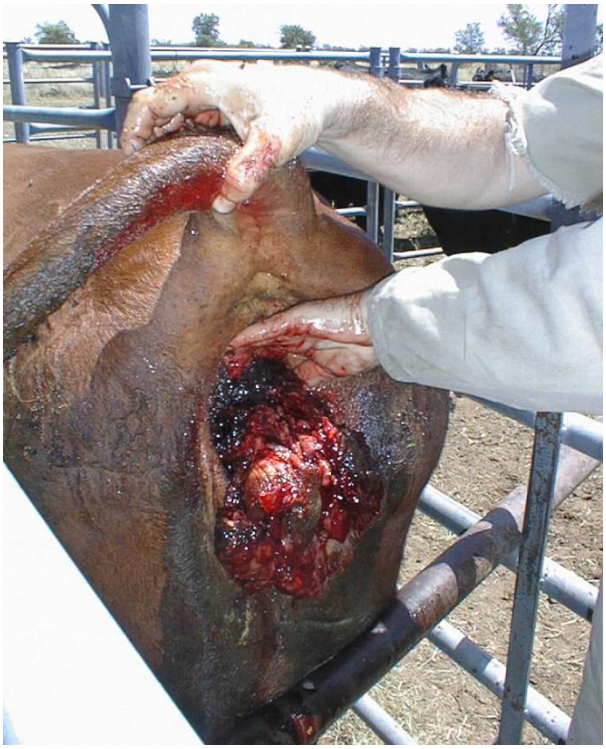

Figure 2 - An example of a Grade 3 vaginal prolapse that is continuously exposed, with the cervix involved.

Treatment

1. If there will be a delay before the prolapse receives veterinary attention, the owner can be instructed to gently clean the exposed tissue and spread a layer of sugar or salt over it. The sugar or salt may help reduce some of the oedema over a period of time. Placing the cow in shade and covering the prolapse with a moist towel, or plastic cling-wrap, can help protect it from further soiling and damage until it can be replaced.
2. Perform a rectal examination of the cow to confirm pregnancy and to estimate gestational age. This procedure also aids in emptying the rectum which helps reduce faecal contamination of the prolapse once treatment is under way.
3. Clean the prolapse thoroughly with dilute chlorhexidine or iodine scrub. While cleaning, inspect the prolapse for tears or penetrating wounds. Although uncommon, full-thickness injuries may need suturing if present. In severe cases where the prolapsed tissue is badly traumatised or has become necrotic, a mucosal strip or amputation may be necessary.
4. Administer a low epidural anaesthetic using 4 to 5 mL of 2% lignocaine.
5. Begin massaging the prolapse, being particularly careful not to penetrate the vaginal mucosa with fingertips. Apply obstetrical lubricant if necessary to help this process. Kneading bread dough is a good analogy for the technique used to replace the vagina. The aim of the kneading action is to reduce some of the oedema and to begin the replacement process. Eventually, firm pressure with the flats of the hands will be needed to push the prolapse in through the vulval lips and vestibulovaginal sphincter.
6. Once the prolapse is back through the vulval lips, maintain pressure on it for three or four minutes so that it is replaced in as normal a position as possible and normal blood flow returns to the tissue.
7. The prolapse needs to be retained in one of three methods. These are the Buhner technique (Figure 4 and Figure 5), closing the vulval lips with mattress sutures, or using buttons to fix the anterior vaginal wall in a manner similar to that described by Mencheve (Figure 6 and Figure 7).
8. With regard to the Buhner technique, using a number 22 scalpel blade, make a small horizontal stab incision approximately midway between the base of the anus and the dorsal commissure of the vulva. Make **one** or **two** horizontal stab incisions side-by-side under the ventral commissure of the vulva (Figure 4).
9. Insert the Buhner needle in the order as shown in Figure 4 and place the circumferential Buhner suture. Note that it is not a purse string suture, but purely a deeply-placed circumferential suture that mimics the vestibulovaginal sphincter.
10. Plan for a possible impending parturition. This may include inducing parturition with dexamethasone. A suitable protocol is to administer 30mg dexamethasone, instruct the owner to untie the suture in 24 hours, allow the cow to calve (usually in 36 to 48 hours) and then have the owner re-tie the suture after the membranes have passed.

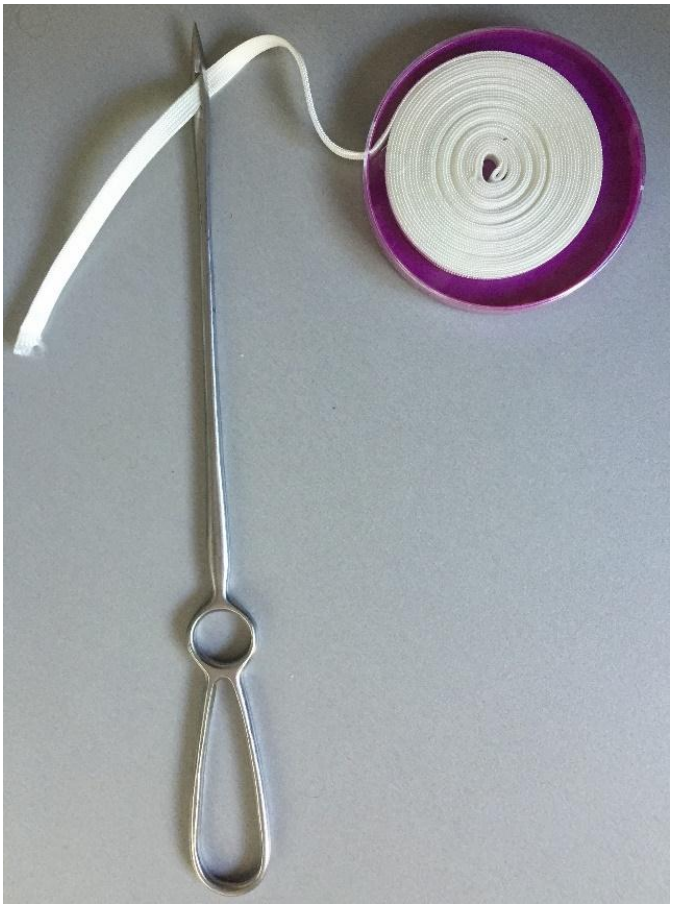

Figure 3 - The Buhner needle and Buhner tape. Utilising the correct form of tape is integral to success of this procedure. It is difficult to pull up tension with lighter cotton tape.

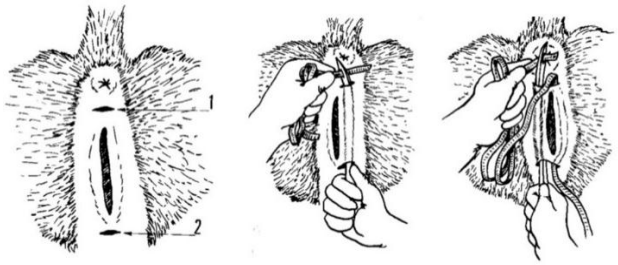

Figure 4 - The Buhner suture procedure sequentially from left to right. An important aspect is to place the suture deeply into the vulval tissue to mimic the vestibulovaginal sphincter. As a guide, it should be placed 5 to 6 cm cranial to the external vulval lips. Place two incisions side-by-side (at point "2" in the image above) if you wish to tie a bow for future release. This way, the knot will not be buried under the skin as it heals. A single incision at "2" is suitable if there is a requirement for the suture to stay in for a prolonged period to mimic the vestibulovaginal sphincter. In this instance, the skin will heal over the knot. (Image modified from Walker and Vaughan 1980).

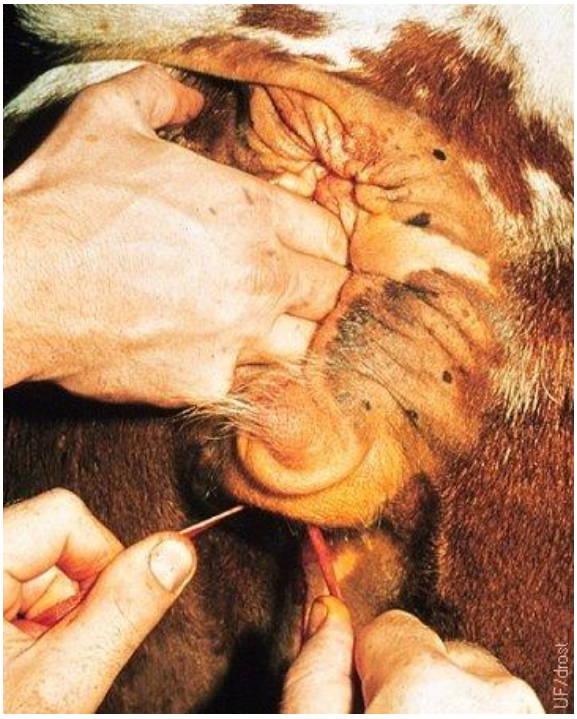

Figure 5 - Pull the Buhner tape up tight enough so that only 2 or 3 fingers can fit through the constriction.

The Buhner suture technique is a relatively simple and quick procedure. Provided the impending parturition is closely managed, this technique is very useful. One specific anecdote is that the greater the duration from prolapse repair to parturition, the more chance of uncomplicated vaginal delivery, as the vaginal tissue has time to rehydrate and soften. The huge disadvantage with this technique is in cows where breeding dates are unknown. In these situations, there is always the risk of severe perineal damage should the cow commence parturition prior to the suture being untied or removed.

A huge advantage of the vaginal prolapse buttons is that they can be left in place during parturition. The downside is the possibility of fistula formation where the trocar traverses the rump muscle.

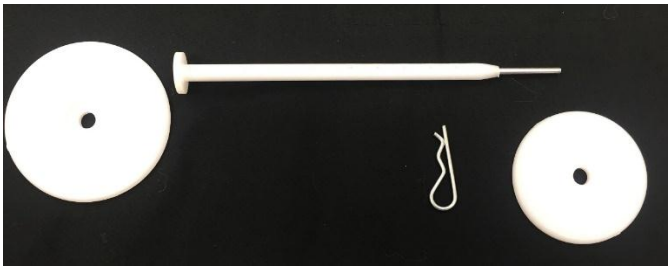

Figure 6 - Vaginal prolapse buttons that mimic the Mencheve technique of repair

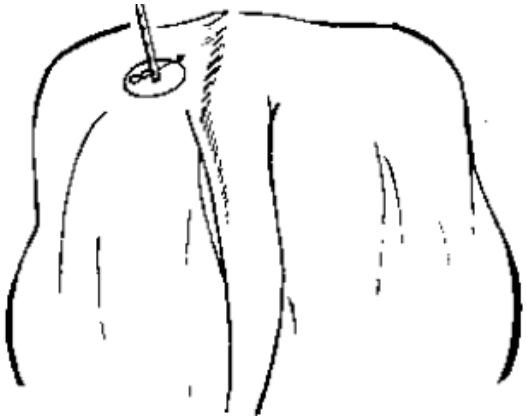

Figure 7 - Placement of vaginal prolapse buttons. It is important to insert the trocar of the button as close as possible to the cervix, but also aiming for it to exit through the rump at a point approximately 15 cm caudal to the tuber coxae and approximately 7 or 8 cm off the midline. By placing the internal button just 3 to 4 cm caudal to the cervix, there is reduced chance of anterior vaginal and cervical tissue re-prolapsing. Bilateral buttons can be placed if vaginal tissue is still apparent at the vulval lips after the first button has been inserted. It is useful to palpate, and become familiar with, the location of the internal pudendal artery and the pudendal nerve prior to utilising this technique.

Prevention

Due to the suspected hereditary component to this disease, it is recommended affected individuals be culled. Maintaining good body condition and avoidance of oestrogenic pastures are beneficial.

HYDROPS ALLANTIOS

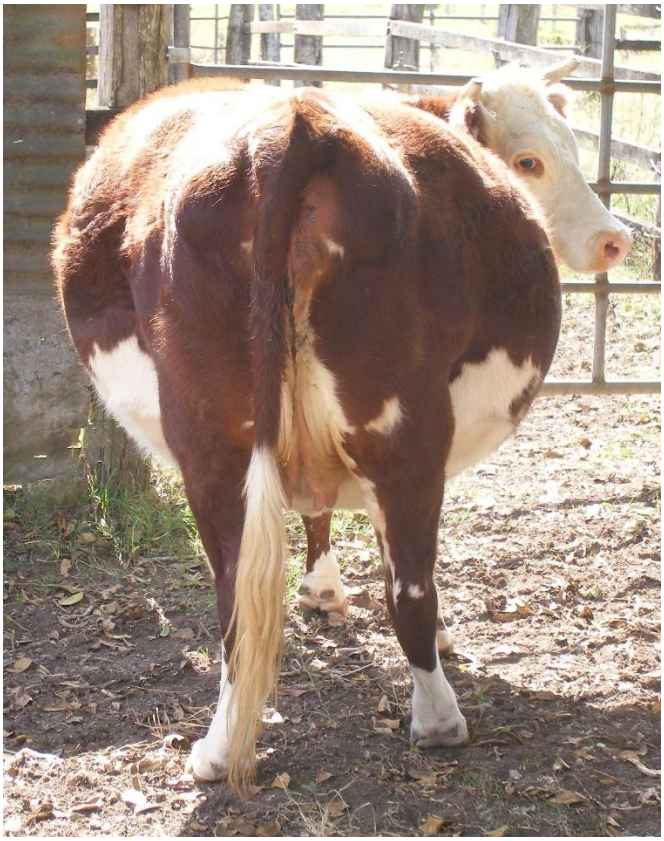

Figure 8 - Hydrops allantois. Note the barrel-shaped abdomen. due to rapid accumulation of around 100 litres of fluid. Placentomes and fetus were not palpable in this cow due to the extreme distension of the chorioallantois.

Aetiology/Pathogenesis

This accounts for greater than 90% of hydrops cases. Aetiology is not clearly defined, but conditions such as **adventitial placentation** (Figure 9), and possibly chronic increases in blood glucose have been implicated in hydros allantois. Adventitial placentation may interfere with the ability of the chorioallantois to process the fluids entering the cavity. Once abnormal fluid processing occurs, there is usually a rapid increase in fluid volume, with more than 150 litres accumulating. The adventitial placentation also makes the cow more susceptible to; placentitis, thickened chorioallantois, retained fetal membranes and metritis. Due to the extreme distension of the chorioallantois, placentomes and fetus are not detectable on per-rectal palpation. This is in contrast to the findings with hydrops amnion, and will assist with formulating a treatment and prognosis.

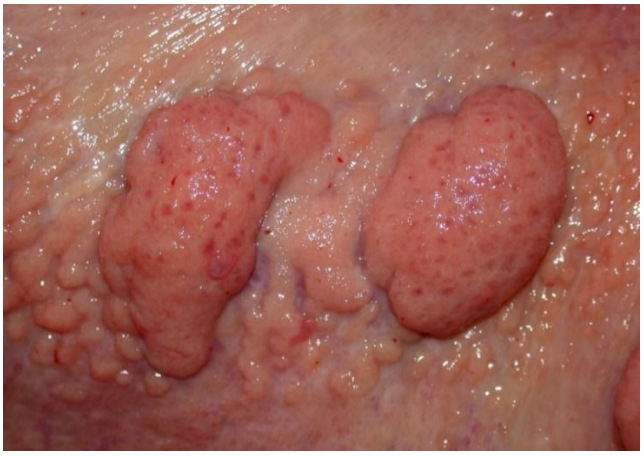

Figure 9 - Adventitial placentation surrounding two caruncles

Treatment

Salvage slaughter is a good option if detected prior to the cow becoming immobile.

The high pressure of the fluid-filled uterus restricts blood flow to the caudal abdomen and limbs. There is a risk of catastrophic hypotension with sudden release of pressure as would happen with a caesarean. Therefore, slow drainage and vaginal delivery in association with fluid replacement is a preferred option. Steps include: –

1. Commence systemic antibiotics to address the risk of placentitis and metritis.
2. Induce parturition utilising corticosteroids +/- prostaglandin $F_{2\alpha}$ depending on the estimated duration of gestation. I prefer corticosteroids alone if the cow is close to term and a viable fetus is expected, since this allows a little more time for fetal maturation (brown fat, thyroid function, adrenal maturation, etc), maximum softening of the reproductive tract, and colostrum production. It allows relaxin to have maximal effect on the cervix.
3. Depending on facilities and labour availability, consider slow release of the fluid by inserting a Foley catheter through a cannula via the lower flank of the cow (Figure 10). The cow will need to be restrained in a crush for 2 to 4 hours (depending on the flow rate) in order to allow the fluid to drain off slowly. A tap can be attached to the end of the catheter to control fluid flow-rate if needed. A second catheter can be inserted in the other side if needed. Fluid drainage can be commenced approximately 24 hours after parturition induction, but sooner if the cow is in distress. Allantoic fluid can rapidly re-accumulate and so repeated drainage will be needed to keep the cow comfortable until parturition.
4. Monitor cow circulatory parameters and administer fluid therapy as needed. Pay attention to plasma sodium.
5. Prepare for an assisted vaginal delivery 36 to 48 hours after induction if the duration of gestation is within two weeks of term. There may be a need to cut the chorioallantois open with a blade or scissors if there is thickening due to placentitis.

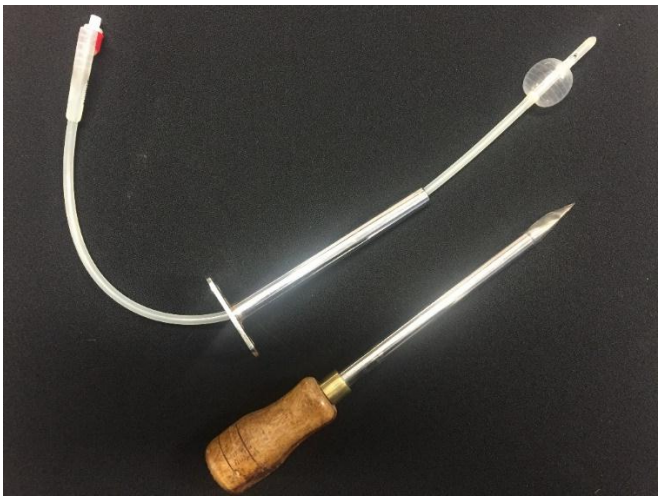

Figure 10 - A trochar and cannula for insertion into the lower flank of a cow with hydrops allantois or hydrops amnion. A Foley catheter is inserted through the cannula and the balloon cuff inflated to keep it stabilised within the uterus. The collar of the cannula can be sutured to the skin of the cow. The cow should be restrained in a crush for the duration of the fluid drainage. The process may need to be repeated daily until the calf is delivered.

HYDROPS AMNION

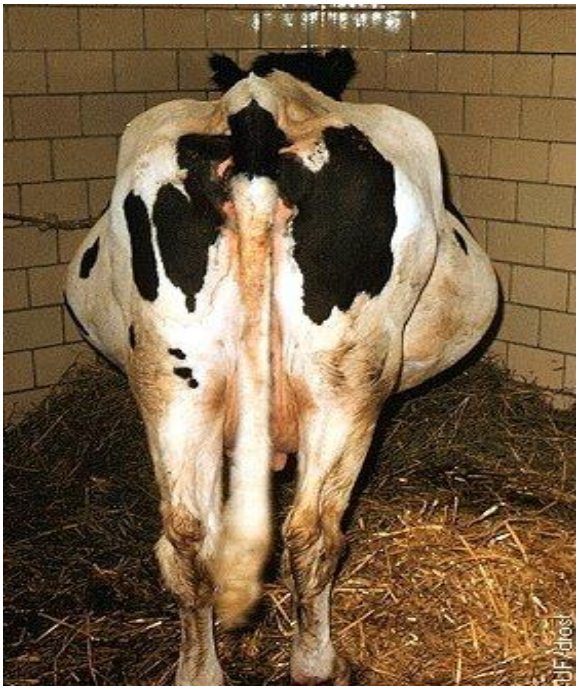

Figure 11 - The classic pear-shaped abdomen of a cow with hydrops amnion. The pear-shape occurs due to the slow accumulation of fluid compared to the barrel shape after rapid accumulation in hydropsallantois.

Aetiology/Pathogenesis

Hydrops amnion is most commonly associated with fetal deformity of the upper respiratory tract, or neuroendocrine dysfunction. Anterior pituitary dysfunction is also associated with prolonged gestation and may be more common after some arbovirus outbreaks. Calves from these cases are usually not viable. But the prognosis for future fertility of the cow is good – in contrast to hydrops allantois.

Treatment

The treatment is similar to the treatment of hydrops allantois, except there is not the urgency to commence systemic antibiotics. Induction of parturition followed by assisted vaginal delivery is suitable.

FETAL MUMMIFICATION

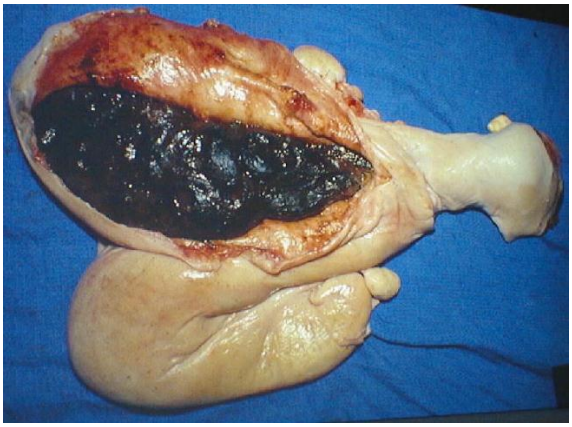

Figure 12 - A mummified fetus in-utero. The uterus can be tightly contracted around the mummified tissue.

Aetiology/Pathogenesis

Fetal mummification arises after fetal death, where luteolysis is incomplete (cervix stays shut) and no putrefactive bacteria enter the uterus. The presence of a functional corpus luteum is integral to the disease process and treatment. Documentation of the mummification process in sheep suggests that a fetus can reach the mummified stage, where most fluids are resorbed, as soon as 7 days after fetal death. The temporal limits of when mummification can occur during gestation are based upon when there is sufficient tissue that can be mummified. Therefore, mummification is possible from approximately 3 to 8 months of gestation. Mummification seems to be most common around 4 to 5 months of gestation, meaning there is a dried out fetus that was the size of a large cat or beagle dog. Examples of specific causative agents include bovine pestivirus, bovine herpes virus type 1, mid-gestational prostaglandin $F_{2\alpha}$ administration, and umbilical cord torsion. Genetic aberrations resulting in sterile death may also be involved.

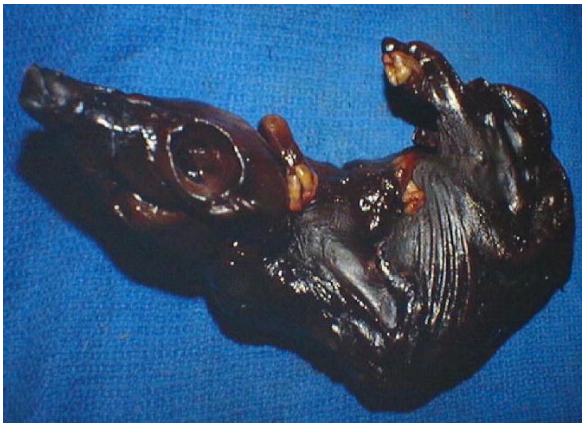

Figure 13 - A mummified fetus after extraction.

Treatment

Spontaneous abortion of fetal mummies before or near normal expected term sometimes occurs. However, most mummies will remain *in utero* until treatment is administered to expel them, or until they are removed by caesarean. While large-scale studies are unavailable, there is suggestion that the administration of prostaglandin $F_{2\alpha}$ will successfully aid the vaginal delivery of fetal mummies in more than 80% of cases. Yet, it is important for the vaginal delivery to be assisted in the period from 3 to 5 days after $PGF_{2\alpha}$ administration as the uterus is usually contracted around the dry fetal tissue. This assistance usually entails the administration of obstetrical lubricant via stomach tube, followed by gentle bougienage of the cervix, (with the hand in a conical shape acting as the bougie), until the fetus can be extracted. The bougienage process may take 30 to 45 minutes and should be done gently and with repeated lubrication if future fertility of the cow is important. In cases where the cervix doesn't dilate enough to allow the fetus to be extracted, there seems to be little benefit from the prior intramuscular administration of oestrogen conjugates (eg oestradiol benzoate), nor cervical application of prostaglandin E (200ug misoprostol). Although there is opportunity for further research into utilising PGE to soften the cervix.

In cases where the mummy can't be delivered vaginally, the options are salvage slaughter, or surgery. With regard to surgery, it can be quite difficult to gain good exteriorisation of the uterus via a left-flank approach with smaller mummies (approximately 5 months or less at the time of death). In these cases, a paramedian, or ventral midline approach provides improved access. An interesting approach is that described by Irons (1999) of exteriorisation of the affected horn and removal of the mummy via a colpotomy incision.

Prognosis for future fertility after successful treatment is fair to good.

FETAL MACERATION

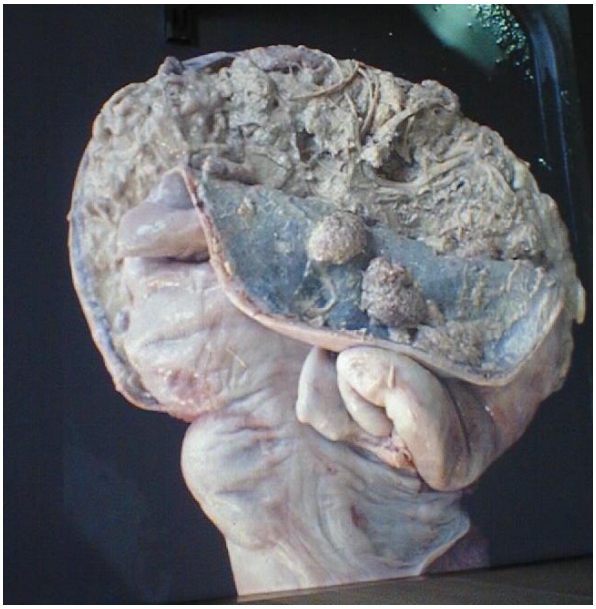

Figure 14 - The appearance of fetal maceration within the uterine lumen. Fetal ribs can be seen within the slurry. The endometrium is severely damaged in these cases, but has some potential for partial recovery if all of the debris and infection can be removed. However, the prognosis for return to fertility is poor.

Aetiology/Pathogenesis

In what could be considered the next step in a continuum from fetal mummification, fetal maceration arises after fetal death, where luteolysis is **complete** and the cervix opens (at least a small amount, and the mucus plug liquefies), allowing putrefactive bacteria enter the uterus. Anything that reduces plasma P4 for long enough can lead to this result, including; stressors, placentitis, iatrogenic $PGF_{2\alpha}$, BHV-1, grade 3 vaginal prolapse, etc.

Treatment

The treatment of macerated fetus can be difficult, particularly if future cow fertility is important. Often treatment is not warranted due to the poor prognosis for future fertility, but may be advisable to reduce the chance of uterine discharge should salvage slaughter be attempted. Despite the fact that the C.L is usually non-functional, it is prudent to administer a luteolytic dose of $PGF_{2\alpha}$ prior to treatment intervention if possible. This ensures there is no P4 in the system preventing cervical dilation. Most of the macerated debris can be removed

via repeated warm saline flushing through an appropriately-sized stomach tube. A tea-mug full of salt in a 10 litre bucket of warm water is a good field-guide to making up the saline. The water to be quite warm. When an arm is placed in the bucket of water, the temperature should be tolerable, but just a little uncomfortably warm. Be mindful of the capacity of the uterus, but provided the cervix isn't tightly sealed around the tube, saline can be infused in and out of the uterus in large volumes. Some of the smaller bones may come out with this flush, and as the return becomes clear, all that will be left will be the larger bone fragments. Common problems are the bony remnants of the pelvis and skull, and pieces of rib that may be embedded in the endometrium. These items are often too large to fit through the poorly dilated cervix. On occasion, a hand can be inserted through the cervix and the bony fragments crushed prior to removal. However, this is only possible in long-standing cases. In cases where the bony fragments cannot be removed vaginally and the cow is not to be progressed to salvage slaughter, surgery becomes the option. The best exposure in these situations is via the ventral midline, or paramedian approach. In exceptional cases, it may be possible to exteriorise the affected horn via a colpotomy incision. However, the potential for post-operative infection is high following this approach. Arcanobacter pyogenes is a common pathogen and so procaine penicillin is a suitable systemic option to commence pre-surgery. Three grams of oxytetracycline is still a good option for intrauterine infusion once debris is removed. Luckily, the oxytetracycline (bacteriostatic) and the penicillin (bacteriocidal) don't impinge on each other's territory when administered in this manner.

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SELENIUM SUPPLEMENTATION TO REDUCE THE INCIDENCE OF RETAINED FOETAL MEMBRANES

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Introduction

Prolonged retention of foetal membranes (RFM) increases the risk of uterine infection, which has significant consequences on the margin of profitability in dairy systems, as good post-partum uterine health is important for fertility and high milk yield. Loss of cell membrane function secondary to a deficiency of anti-oxidant activity may affect normal placental separation.[2,3] Deficiencies of Vitamin E and selenium have been linked to an increased incidence of RFM, however analysis of the independent effect of selenium is currently lacking. Dairy production systems are commonly located in regions of selenium-deficient soils and cows in pasture-based systems may be at a higher risk of deficiency (consuming less than 0.3mg selenium per kilogram of dry matter).[4] A systematic review was conducted to examine research papers investigating the sole effect of selenium supplementation during the dry period, on the incidence of retained foetal membranes in dairy cows.

Method

The specific question posed was: 'does selenium supplementation of dairy cows during the dry period reduce the incidence of retained foetal membranes?'

A selection criteria was applied to the articles found during the literature search to screen the results in relation to their relevance to the research question.

1. Only primary research articles will be included. Review articles will be excluded.
2. Dairy cows must be the population of interest. Cattle raised for beef production and calves will be excluded.
3. Selenium supplementation must be the intervention and used for comparison.
   a) Supplementation must occur pre-partum during the dry (non-lactating) period. Exclude articles with supplementation that occurs during lactation.
   b) Selenium is the sole supplement provided for comparison. Exclude articles with interventions that combine selenium with another supplement.
   c) Exclude studies if there is no control population (no selenium supplement) for comparison with the intervention.
4. Outcome: incidence of retained foetal membranes for more than 12 hours post-partum.
5. The environment and feeding system of the population should be specified. Exclude studies if the selenium concentration of the study diets is unspecified.

Literature search

The databases used to gather literature on the topic included PubMed, ScienceDirect, Web of Science, VetMed, Agricola, and CSIRO online journals. Access was restricted to the institution rights for the University of Queensland.
The combination of key words used included "dairy cattle", "selenium", "reproduction", "retained placenta" and "retained membranes". Only full-text English articles could be fully evaluated; all other articles were excluded. The time-frame by which authors defined 'retained' was not discriminated to increase the yield of articles and account for variation in interpretation.

Results

The results of the search and after screening for relevance are displayed in Figure 1.

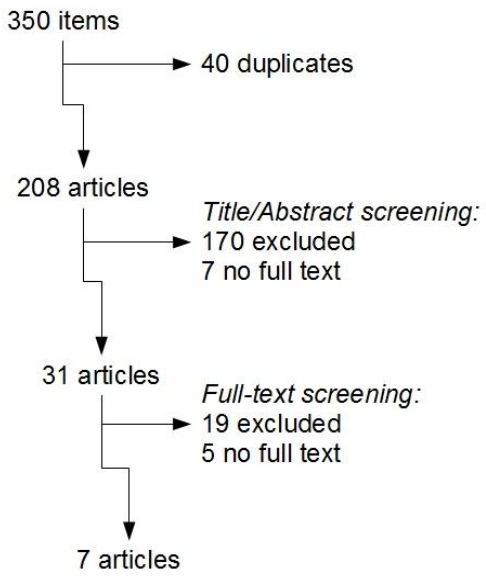

Figure 1: Relevance Screening

The bibliographical data for each of the seven articles is listed in the references.

All studies were conducted as prospective controlled trials. Each paper described multiple interventions and outcomes that were investigated at the same time as the intervention and outcome of interest to this review.
The research by each author took place in the Northern Hemisphere. Two of the studies (by Trinder, et al.) were conducted on the one farm using the same herd one year apart, for six months over winter (November to May 1967-1968 and 1968-1969). Two other studies (Hidiroglou, et al. and Eger, et al.) combined herd data obtained over three successive years.

The study populations were kept in yards or barns during the trial. Cows in the papers by Trinder, et al. were on pasture during summer. In six studies, cows received a form of forage and depending on the feed supply, received a supplementary ration. The ration components of the seventh article by Eger, et al. were not listed. The amount of selenium concentration varied between diets (0.02-0.2 mg/kg DM, Table 1).
Cows were assigned to different intervention groups in blocks, by sub-dividing groups and by sequential and alternating allocation. After allocation to the intervention group, one cow was excluded by Brzezinska-Slebodzinska, et al. because the cow calved more than five weeks early. Julien, et al. excluded the results from an unspecified number of cows that received six milligrams of selenium at 60 days pre-partum, after determining the plasma selenium concentrations of these cows did not increase after supplementation.
Selenium was given to the cows in the intervention group orally or by injection. The dose, frequency and time administered prior to parturition was different between trials (Table 1).

Selenium supplementation was concluded to contribute to reducing the incidence of RFM in five of the studies although two of these articles (Trinder, Woodhouse, et al. and Harrison, et al., Articles 3 and 4 respectively, Table 2) placed greater importance on adequate dietary Vitamin E, for selenium supplementation to be effective. Hidiroglou, et al. and Eger, et al. questioned the influence and efficacy of selenium supplementation when combined with other treatments and factors influencing the incidence of RFM. One paper (Brzezinska-Slebodzinska, et al.) was inconclusive on the role of selenium, due to the small sample size of the trial.

Table 1: Intervention and Comparison

Article	Basal diet concentration (mg Se/kg DM)	Intervention			
		Intervention (per cow)			Control
		Amount	Type (Route)	Frequency	
1	0.2	3mg Se	sodium selenite (gelatin capsule, PO)	q.24hrs[a] for 6wks pre-partum	No Se supplement
2	0.04-0.05	15mg Se	aqueous potassium selenate (IM)	Once 28d pre-partum	No Se supplement
3	0.02-0.03	15mg Se	aqueous potassium selenate (IM)	Once 28d pre-partum	No Se supplement
4	0.09	0.1mg/kg Se[b]	sodium selenite (IM)	Once 21d pre-partum	No Se supplement
5	0.02-0.07	12.5mg Se	sodium selenate (PO)	q.24hrs 60d pre-partum for 5d then once weekly	No Se supplement
6	0.2	6000 mg Se	elemental Se (rumen pellets, PO)	Once 60d pre-partum	No Se supplement
7	0.04	2.3mg Se	sodium selenite (IM)	Once 20-21d pre-partum	No Se supplement

[a] Every 24 hours (once daily).
[b] Mean bodyweight of 18 cows in the treatment group 21d pre-partum was 657 (±27, SE) kg; therefore the estimated dose is 65.7mg Se per cow.

Table 2: Outcome and Conclusions

Article	Outcome[c]		P-value	Effect of supplemental Se (Author's Conclusions)
	Intervention	Control		
1	4 of 15 (26.7%)	6 of 16 (37.5%)	Not specified	Inconclusive
2	5 of 17 (30%)	9 of 15 (60%)	~0.06*	Strongly supported
3	4 of 14 (28.5%)	7 of 19 (37%)	Not specified	Weakly supported
4	3 of 18 (17%)	3 of 19 (16%)	'No difference'	Weakly supported
5	0 of 11 (0%)	10 of 26 (38.5%)	<0.01*	Strongly supported
6	58 of 220 (26.2%)[d]	46 of 217 (21.1%)[d]	>0.05	Strongly opposed
7	7 of 65 (10.8%)	21 of 73 (29.0%)	≤0.02*	Weakly supported

[c] Outcome defined as the number (and percentage) of cows that had RFM. The outcome for articles 1, 5, 6, and 7 defined retained as >12h post-partum. Outcome for articles 2 & 3 defined retained as >6h post-partum. Outcome for article 4 defined retained as >24h post-partum.
[d] Derived numbers based on the percentage value given in the results.
* Statistically significant finding (as determined by authors).

Discussion

Each paper was assessed for its level of evidence and described broadly in accordance with the GRADE guidelines.[5] The evidence was justified by considering the risk of bias, imprecision, inconsistency, indirectness, and publication bias.

Feed constituents were consistent with most dairy systems, reducing selection bias. It is possible as a result of ongoing research and development in nutrition and agronomy over the past 25 years that there may be a temporal effect on delivery and bioavailability of nutrients and minerals in the supplementary feeds. All rations were below the recommended dry matter concentration of selenium. The results may be able to be extrapolated to herds grazing selenium-deficient pastures, in addition to herds fed total-mixed-rations. The value of fresh forage may be more important when considering the influence of adequate Vitamin E concentrations.[6]

The use of only multiparous cows by Brzezinska-Slebodzinska, et al. and Harrison, et al. reduces the applicability of the studies to a whole herd that includes heifers. However, heifers are often managed separately to cows pre-partum and confounding due to age, parity and the differences in management was reduced by restricting the study to multiparous cows. The other studies did not describe different housing for different parity cows. Although housed together, Eger, et al. showed that selenium reduced the incidence of RFM in both primi- and multi-parous cows compared to controls, however only multiparous cows showed a statistically significant reduction (P≤0.05). In contrast, Hidiroglou, et al. housed all dry cows together without a significant (P>0.05) interaction of parity with incidence. Keeping heifers together with cows during the trial may reduce the effect of other confounding factors (to enable data to be combined for total analysis of RFM incidence), but the applicability of this combined data may be reduced for farms that feed and manage heifers separately.

Although four authors (Brzezinska-Slebodzinska, Harrison, Julien, and Hidiroglou, et al.) quoted 'randomized' assortment of cows into the different groups, the method of randomisation was not specified and there was no confirmation that personnel were blinded, increasing the risk of bias.

Variation in selenium content of different diets fed to one herd during the same dry period potentially could have had an effect, however the selenium concentrations were both still below the required level and unlikely to significantly raise the blood selenium content.[7] Confounding associated with a different ration was recognised by the authors of the study where the herd was fed two different diets during the trial (Trinder, et al.). Although Eger, et al. described the lactating diet of cows fed prior to and during the study period as containing an adequate supply of selenium (more than 0.1ppm as per the 1978 NRC recommendation), this is below the current NRC requirements. Their conclusion that selenium supplementation is useful for the prevention of RFM when only the dry cow diet is selenium-deficient may be misleading.

There is a risk that using the same herd through the experiments conducted by Trinder, et al., Hidiroglou, et al. and Eger, et al. may have included data analysis of cows that had received antioxidant supplementation in multiple years. Whilst it is unlikely to have prevented RFM primarily (due to a reduction in plasma selenium over time), the incidence of RFM in these cows may have been reduced by preventing subsequent uterine damage, such as from infection or manual removal, that may predispose to RFM in the following years. The risk of an extended supplementary benefit into the next year from intraruminal selenium pellets was minimised by Hidiroglou, et al., who excluded cows from analysis in the subsequent year, if they had previously been allocated to Treatment 3 i.e. received two intraruminal pellets. The authors didn't exclude cows that had received selenium as Dystosel (selenium and Vitamin E) in previous years. Statistical analysis was performed by Hidiroglou, et al. accounting for selenium treatment alone or in combination with Vitamin E for multiple variables including veterinarian-assisted parturitions, RFM, time to first oestrus and first service, days open, and number of services per conception. No statistically significant ($P>0.05$) effect was found.

The lower effect of selenium supplementation as determined by Brzezinska-Slebodzinska, et al. and Hidiroglou, et al may have been due to the control diets (0.2 and 0.16mg/kg selenium, respectively) containing close to the recommended selenium concentration as defined by the NRC. Hidiroglou, et al. found only rumen-pellet-supplemented cows, not control cows, showed an increase in plasma concentrations great enough to achieve concentrations at parturition above what is hypothesised to be required for good heath (more than 0.08 micrograms per millilitre).[4] Plasma selenium from all cows with RFM (0.086µg/ml) was described as being insignificantly different compared to cows without RFM (0.0796µg/ml). As these figures are close to the above-mentioned minimum plasma concentration, it may suggest the basal dietary selenium concentration consumed by both treatment and control groups in these studies were sufficient to negate evidence of a difference in incidence as a result of the intervention. In the study by Harrison, et al. there was a statistical difference in plasma selenium between the intervention and control groups after supplementation however the levels in both groups at parturition were below 0.08µg/ml and may have been insufficient to have a clinical effect. Julien, at al. demonstrated an increase in plasma concentrations of the treated group, to above 0.08µg/ml at parturition, whilst control cows had an average of 0.03µg/ml. In conjunction with the significant ($P<0.01$) reduction in RFM of treated cows in this study (of which all cows consumed less than 0.1mg/kg of selenium in dry matter), this appears to support a beneficial effect of selenium supplementation of selenium-deficient diets.

Individual cows calving early or very late in the season may have an increased risk of RFM. In the study by Brzezinska-Slebodzinska, et al., the cows due to calve on a similar date were evenly spread across all groups, which minimised the influence of this on the results. A similar approach was taken by Eger, et al. that sequentially allocated cows as they were dried-off into either the intervention or control group. There was no discussion of the reproductive history of the cows, and previous health issues may increase the risk of retention. Without considering these effects, the difference in incidence may be inaccurate depending on the distribution of these cows to the intervention and control groups. Brzezinska-Slebodzinska, et al. recognised cows that have a complicated parturition may have an increased risk of retention. Three cows in the control group (one stillbirth and two that gave birth to twins) retained their placentas, and may have biased the results towards a greater difference between intervention and control group outcomes.

Any reduction in the incidence of placental retention is likely to be clinically significant for the application of the study results to the dairy industry, since selenium supplementation is relatively inexpensive but the cost of subsequent health and fertility problems in cows that retained their membranes is substantial. The increased selection for high productivity in the past 50 years has resulted in increased mortality and lowered fertility.[8] This suggests that any decrease in RFM incidence is likely to contribute to a reduced risk of uterine disease that would otherwise further detract from herd reproductive performance and increase culling numbers. However, small sample sizes can make the estimate of the true effect inaccurate and the influence of other treatments can disguise the effect attributable to selenium supplementation.

Classification of the outcome with respect to hours post-partum may have changed the incidence of retention within each group. Studies that use the same measure of outcome would be better for comparison of effect.

Each of the studies were published alongside the results of other interventions and outcomes under investigation. Other literature that solely investigated the effect of selenium on the incidence of RFM may have remained unpublished if there were statistically insignificant results.

In summary:

- All rations contained less than the recommended level (0.3mg/kg DM) of selenium, similar to selenium-deficient pastures.
- The results may be less applicable to herds where heifers are managed differently to cows, although parity was not shown to have a clear confounding effect.
- None of the studies adequately described a random method of allocation of animals to groups.
- Reproductive history and concurrent or previous antioxidant supplementation may be important factors to consider when determining the incidence of retained foetal membranes after selenium supplementation.
- Supplementation of cows consuming diet concentrations above 0.1mg/kg DM of selenium may not show a significant effect on RFM, compared to controls.

The quantity of evidence investigating the research question is low, presenting a small reflection of the potential effects of selenium supplementation on the incidence of RFM. The high level of inconsistency in trial design and results also reduces the value of combining these studies for direct comparison of effect. This could have been minimised by better defining the type of supplementation (dietary versus injectable) and dose amount, and the time from parturition that membranes are classified as being retained
The small sample sizes may cause the magnitude of the effect size to be disproportionate to the real value as the results can be heavily influenced by the presence of animals in each study group that are unrepresentative of the general population, reducing the power of the studies. There is a risk of over-interpretation of the results because of possible publication bias being present. The ability to understand non-English papers may have increased the quantity of evidence. Studies conducted in Australia and New Zealand may have increased the applicability of the results. Overall, the evidence provided is moderate in value, indicating that the true effect of selenium supplementation is likely to be close to the estimated effect found within the studies, but it is possibly substantially different.

Conclusion

Selenium supplementation may be warranted as a preventative measure of placental retention. Nevertheless, little support is present to recommend supplementation when cows are consuming close to or above the recommended selenium concentration (0.3mg/kg DM) from other supplements and fresh grass. A statistically significant effect was apparent when the diet consisted of less than 0.1mg selenium per kilogram of dry matter. Whilst selenium supplementation is inexpensive and the financial consequences of disease great, any effect is likely to be clinically important. There are multiple influences on the incidence of retained foetal membranes, and control of several factors may be required.

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HOW TO DART CATTLE

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Introduction

This paper describes a method for veterinarians to capture free-ranging Australian cattle with darting equipment and chemicals registered for use in food producing animals.

Australian cattle vets encounter many scenarios where treatment is required for cattle that are difficult to capture (muster or yard) or approach. Chemical immobilisation via darting is a very useful tool for facilitating the treatment of many large herbivores (e.g. free-ranging horses [1]). Cattle darting methods appropriate to other countries have been developed [2,3] but there has previously been no reliable published method for Australia. The aim of this study was to develop an efficacious, humane, cost-effective darting method for free-ranging Australian cattle.

Methods

Thirty free-ranging female cattle (*Bos taurus/indicus*) were darted and captured on a pastoral station in north-west Australia from a 4WD vehicle over six days in April 2015. Mean maximum air temperatures in the shade at the field site were 38°C. Animals were captured for the deployment of telemetry collars, permitting post-capture monitoring and the quantification of mortality rate at the time of capture [4] and at 14 days post-capture [5]. We used a CO_2-powered dart rifle fitted with telescopic sights and 6 mL charge-powered darts fitted with barbed or cuffed needles.

Capture agents used were veterinary chemicals that are registered for use in food producing animals in Australia [6], namely a combination of xylazine and ketamine, antagonised with yohimbine. These capture agents have been used for the darting of other large wild ungulates in Australia (e.g. red deer (*Cervus elaphus*) [7]). To accurately estimate dosage, body weight was calculated from morphometric measurements of recumbent cattle, using the 'weigh tape' approach [8].

We quantified the duration of procedures, specifically induction time (from darting to recumbency), and recumbent time (from recumbency until recovery). We measured induction distance (distance from darting to recumbency site) with a handheld GPS unit. We also quantified standard physiological parameters during recumbency, including heart rate, respiratory rate, body temperature and tissue oxygenation.

Results

Thirty cattle were successfully darted and captured. Mean induction time was 8 ± 1 minutes (mean ± SEM), while mean recumbent time was 27 ± 3 minutes. Two animals (7 %) did not become recumbent after initially being darted and required repeat darting (a second dart administered). One animal (3 %) escaped the capture team during the induction period through rapid escape behaviour into thick woodland.

Effective mean dosage for xylazine was 0.59 ± 0.02 mg kg^{-1}, for ketamine was 3.59 ± 0.012 mg kg^{-1}, and for yohimbine was 0.10 ± 0.009 mg kg^{-1}. Induction distance was 260 ± 203 metres. Physiological parameters during recumbency were unremarkable, with no animals displaying a mean body temperature > 40.0 °C. Mortality rate was zero on the day of capture but 7% at 14 days post-capture. The total cost of the consumable component of the darting method (excluding labour and vehicle use) was approximately $30 per animal.

Discussion

The majority of darted cattle were successfully immobilised with one dart and recovered within 30 minutes, with consumables costing around $30 per captured animal. Given legal inconsistencies between Australian jurisdictions, we emphasise the importance of using veterinary chemicals registered for use in food producing animals when darting Australian cattle. We urge against the use of xylazine alone for cattle darting, as the high doses required greatly increase the risk of adverse anaesthetic events [2].

Inducing anaesthesia via remote injection is a high risk practice and mortality rates in excess of 10% are often for encountered for Australian species such as kangaroos (*Macropus* spp.) [9]. However, mortality rates below 2% are

achievable for large mammals following refinement of practices [4].

Conclusion

The technique trialled represents a useful method for capturing cattle and could be used by Australian cattle veterinarians in many contexts, including the capture of escaped animals on roads [10], wharves or at saleyards. Further refinement of dosage and procedures will be required to lower the frequency of adverse events, especially post-capture mortality.

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BIOCHEMISTRY: THE TOOL CATTLE VETS NEED IN THEIR BACK POCKET

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Abstract
The use of biochemistry in the everyday workup of bovine medicine cases can be greater utilized by cattle veterinarians to give an accurate diagnosis, direct treatment choices and give a quantitative indicator of prognosis to producers.

Diagnosis of Ketosis

Traditionally, urine dipsticks like “Ketostix” or “Combur 9” have been employed to diagnose ketosis in cattle. Using nitroprusside test squares if acetoacetate (and acetate to a lesser degree) is present in the urine, the test square turns purple on a scale of low, moderate and high. The test is cheap (~25c/test), most likely explaining why the test has such a wide uptake and utilisation by bovine practitioners. However, the test does not detect beta hydroxybutyrate (BoHB), the most predominant ketone in a cow with ketosis. Compared with a blood BoHb serum level of 1.4mmol/L, the test has a sensitivity of 0.78 and a specificity of 0.96[1]. The poor sensitivity of the urine ketone detection strips suggests there is a high degree of false negatives. For example if ketonuria exisited in 100 cows, 22 cows would be positive but would be incorrectly diagnosed as negative.

Elevations in blood BoHb are considered the “gold standard” to diagnose subclinical ketosis. With a sensitivity of 0.91 and a specificity of 0.94[1], blood BoHb levels should also be used in preference to urine ketone testing for the diagnosis of ketosis in the unwell, poor-performing bovine. Practitioners should be reaching for a blood ketone monitor every time they complete a clinical exam of a sick cow, as ketones in the blood result in immunosuppression. In a cow that has metritis, pneumonia or other ailment, it is important that the diagnosis of an elevated blood BoHb isn’t missed, simply because the practitioner utilised a cheap test with a poor sensitivity.

Cows with sub-clinical ketosis (BoHb 1-1.4mmol/L) have eight times the risk of developing an LDA[2]. If a cow has ketosis in her first two weeks of lactation she has a reduced probability of pregnancy at her first insemination[2]. Cows with sub-clinical ketosis in the first two weeks post-calving have a three time greater risk of metritis[2]. Cows with a blood BoHb >1.8mmol/L in the first week post calving had a lower projected production for the whole lactation[2]. However early detection of ketosis allows quick corrective treatment to be administered thus reducing the risk of development of these problems. It is important to note that BoHb is a breakdown product from the mobilisation of fat and therefore BoHb levels may be normal in a starved (chronic) cows, and so as with all tests should not be read in isolation, but instead interpreted in conjunction with the associated clinical signs of the animal.

Anecdotally at Finley Veterinary Clinic (FVC), the handheld blood ketone monitors are also employed to measure blood glucose concurrently. If a ketotic cow is hypoglycaemic, then IV dextrose becomes a component of her treatment plan. When the blood glucose is very elevated (>10mmol/L, often >25mmol/L), then the cow is considered critically ill and anecdotally suggests grave prognosis. It is underdetermined if the elevated blood glucose is a stress hyperglycaemia or a loss in the cow’s ability to regulate glucose homeostasis.

Detecting Acidosis in Scouring Calves

At FVC, when the results of the in house calf scour diagnostic tests, (such as “Rainbow 6”) from the previous 5 years are analysed, the breakdown suggests the main pathogen seen in calves in the area is Cryptosporidium (Table1) (nb Salmonella not included in the results due to difficulty in getting a diagnosis on culture and also collating salmonella results from the data set at FVC)

The calves suffering from Cryptosporidium have profuse voluminous diarrhoea resulting from villous atrophy and fusion causing a malabsorption diarrhoea[3]. Secondary milk fermentation increases the osmotic pull, further contributing to the development of severe diarrhoea in young calves.

Mentation, eyeball position and skin tent is used to estimate the degree of dehydration in calves with diarrhoea[3]. However, for most cattle practitioners, the lack of on farm or in-house biochemistry means that no comment can be made on electrolyte changes, degree of acidosis and potential kidney damage. Instead, a number of assumptions are made based on gross signs, including that the calf is likely acidotic, hypoglycaemic and hyponatriemic. However, if the practitioner utilized basic biochemistry results, they would have the ability to quickly get a true quantitative picture of the acid-base and electrolyte status of the calf with ease.

A review of the blood results run on calves by FVC in the previous 2 years is summarised in Table 2.

Organism	Cryptosporidium	Rotavirus	E. coli	Coronavirus	Negative
Prevalence (%)	49%	22%	7%	<1%	21%

Table 1. Results of the previous 5years of in-house calf diagnostic kits at FVC

Biochemistry Change	Acidotic	Hyperglycaemic	Hypoglycaemic	Acidotic and elevated BUN and CREA	Acidotic and elevated BUN	Elevated BUN and CREA only	Elevated BUN only
Prevalence (%)	82%	65%	6%	59%	18%	0%	12%

Table 2. Results of the bloods run on scouring calves in the previous 2 years at FVC

Mild acidosis may be corrected with rehydration and many practitioners have traditionally rehydrated calves with Hartmanns solution (Lactated Ringers Solution) which has the dual action of correcting the fluid deficit while also serving as a moderate alkalinising agent. However for the lactate to be converted into bicarbonate it requires a functioning liver, and in a cardiovascular compromised calf there is often concurrent reduced hepatic blood flow. Therefore, relying on Hartmanns IV fluids to correct an acidosis is inefficient and ineffective in majority of cases. Instead isotonic fluids spiked with sodium bicarbonate should be utilised[4].

At FVC, as the TCO2 level is able to be measured in scouring calves, the exact bicarbonate deficit can be calculated (Table 3).

Mmol Bicarbonate required = body weight (kg) x (30-TCO2) x 0.6

13g in 1L water for injection provides 156mmol/L of Bicarb

For example, a 40kg 7 day old Holstein calf with a TCO2 of 7mmol/L in lateral recumbency with an absent suckle.

Mmol bicarb = 40 x (30-7) x 0.6

=552mmol of bicarbonate required

Amount of bicarbonate required/Amount of bicarbonate in isotonic 1L fluids =

552mmol/156mmol/L

=3.5L isotonic fluids containing bicarb required to correct acidosis.

Table 3. Calculations of Bicarbonate Deficit[4]

Using previously published tables (Table 4 & Table 5) that estimate base deficit based on clinical signs the above calf would qualify as an acidosis level 4, with an estimated base deficit of 10 and so therefore would theoretically require 1.3L of isotonic bicarbonate solution[3]. This is significantly less than calculated 3.5L actually required to completely correct the acidosis.

Acidosis level	Clinical signs	Urine pH	<8d base deficit	>8d base deficit
1	Bright, alert, strong suckle, warm mouth	>6.5	0	5
2	Standing or sitting, weak suckle, slightly cold mouth	6.0-6.4	5	10
3	Depressed, unable to stand, sternal, no suckle, cold mouth	<6.0	10	15
4	Collapsed, death imminent, sternal, no suckle, very cold mouth	<6.0	10	20

Table 4. Base deficit based on clinical signs and age of calf[3]

Calf weight	Base deficit (mmol/L)	Volume(L) of isotonic bicarb solution
30	10	1.0
	15	1.5
	20	1.9
40	10	1.3
	15	1.9
	20	2.6
50	10	1.6
	15	2.4
	20	3.2

Table 5. Bicarb requirements of calves depending on their estimated base deficit[3]

This strongly highlights that measuring basic biochemistry values in a scouring calf gives clinicians a quantitative value for the degree of acidosis, and therefore proved them with the confidence to know they have accurately corrected the acid base imbalance in a critically ill neonate.

At FVC, when calves had relapse episodes despite oral and IV therapy, a review of the biochemistry results can sometimes reveal surprising changes in the animal, including marked changes in kidney enzymes (Table 6).

	First Blood Test	4d after 1st visit	7d after 1st visit
Blood Urea Nitrogen (mmol/L)	37.1	39.7	55.1
Creatinine (umol/L)	637	900	1744

Table 6. Results of a calf treated with IV fluids that continually relapsed

The calf represented in Table 6 was treated with IV isotonic bicarbonate initially and responded really well, and was subsequently swapped onto oral fluids. However, 4 days after initial presentation, the calf presented again with acute collapse and deterioration. Biochemistry was repeated and kidney enzymes were noted to have elevated further. Again, IV fluid therapy was administered and the calf responded well. When the calf collapsed again 7days later, biochemistry revealed a marked serial elevation in BUN and Creatinine. A diagnosis of renal failure was made, the calf was given a poor prognosis, and euthanased on welfare grounds. This is another example of how basic biochemistry can be easily utilised in bovine medicine to get a quantitative result, direct treatment and evaluate prognosis. Anecdotally at FVC it is found that most calves that don't respond to IV fluid therapy are in acute renal failure.

Downer Cow Diagnostics

Traditionally, when a vet is called out to see a cow that has 'gone down' 24hours after calving, the presumptive diagnosis is often hypocalcaemia. However what if the practitioner had the ability to complete some basic biochemistry at the time? This allows a quantitative diagnosis to be made, and provides an accurate assessment of muscle damage, which in turn can be used to give a prognostic indicator for return to standing.

At FVC, all consults for recumbent cattle include blood BoHb and a basic biochemistry panel in the workup of their case. A review of the results of these tests from the previous 2 years are summarised in Table 7.

HypoCa	HypoMg	HypoP		HypoCa AND hypoMg	CK >300 U/La	Only CK elevation	CK WNL	ALT/GGT elevation. Macrominerals WNL	No Finding
		And HypoCa	Only P						
34%	13%	21%	11%	11%	67%	26%	7%	5%	1%

Table 7: summary of results for down cows seen by FVC in previous 2 years

Through determination of the current status of macrominerals in the animal, correct treatment of the condition can be initiated. No assumptive diagnoses are made, and therefore treatment of the recumbency is quick and less relapses are observed.

Furthermore, the level of CK can be used as a prognostic indicator using previously published data (Table 8). Where CK levels are at, or above, the data set, the cow has a <5% probability of survival[5]. This is a vital component to treating 'downer cows' as it saves the client money by providing an important indicator to how well a given cow will respond to treatment.

Length of Recumbency	CK level (U/L)
0.5 days	12,200
1 day	18,800
2 days	16,300
3 days	14,000
4 days	10,900
5 days	8,500
6 days	6,300
7 days	3,900

Table 8: Prognostic Indicator for recovery based on CK levels

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HOW TO MANAGE AND PREVENT DRENCH RESISTANCE IN CATTLE

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Introduction

Gastrointestinal nematode (GIN) parasitism is one of the most production limiting diseases of pasture-based dairy cattle worldwide leading to impaired live weight gain of young stock as well as reduced milk production and reproductive performance in lactating cattle [1].

Whilst drench resistance in the sheep industry has been an issue that has been well recognised in Australia since the early 1980's, for some reason we have been slow to look fully for this issue in the cattle industry. However recently there has been more attention to the issues involved with drench resistance in cattle and to assess the costs of this resistance in lost production to the industry.

The slower response to these issues are possibly in part due to

1. The lack of accuracy that worm egg counts(WEC) provide in cattle,
2. Mostly when resistance occurs to parasites the effects are subclinical rather than deaths and as such go unnoticed.
3. The belief that lower levels of drenching especially in adults would mean the likelihood of resistance was far lower.

Investigations over the last decade have shown that resistance within the cattle industry to the drenches available today is quite wide spread. [2,3,1,4.]

It is important that the lessons learnt from the sheep industry are well heeded by the cattle industry and that cattle farmers, be they beef or dairy farmers, understand the best mechanisms for reducing the development of resistance while maintaining profitability in their production system.

So what are some of the lessons we have learnt about resistance development?

Increased resistance is associated with

1. Increasing frequency of drenching
2. Poor use of rotations and or combinations of drench groups
3. Use of products which already have significant resistance or poor potency
4. Under dosing often associated with poor estimation of weight.
5. Lack of refugia while ensuring animals are not put back onto highly contaminated pastures.
6. Lack of quarantine drenching

What issues do we face in the cattle industry?

1. WEC are unreliable and relatively ineffective after 18 month of age[5]
2. The relatively low number of eggs produced by cattle make validation of FECRT very hard.[5]
3. Wide differences in egg laying capacity and pathogenicity of parasites[6]
4. The need for larval cultures due to the variable pathogenicity of the common parasites.
5. Pepsinogen is only an indication of damage done
6. Poor application of pour-ons and some concerns about their general efficacy
7. Lack of drench groups available for cattle drenches, especially those that are effective against type II *Ostertagiasis*
8. Macrocylic lactones(MLs) are almost solely being used with only rotations between drenches within this drench group being considered as rotating by many farmers.
9. Survival of parasites in dung pats for extended periods compared with sheep pellets.[7] Although the role of dung beetles in reducing this element of contamination has been found to be very valuable in the cattle industry.[8]
10. Drenches given on dairies by the calendar rather than using any measurements.[1]
11. Constraints placed on the use of many products during lactation due to withholding periods (WHP).

Based on modelling it has been found that "using actives concurrently was superior to using them individually either sequentially or in rotation, even in the presence of side-resistance between the two anthelmintic classes".[9]

The presence of resistance can be seen as a reduction in the period of efficacy of persistent products although other factors such as body condition may also influence this. Adding a primer dose is known in sheep to extend the persistence and to help reduce the onset of resistance[10]. This is believed to be due to the increase of initial potency which ensures resistant parasites are killed thus ensuring that resistant eggs are not contaminating the pasture for the period of the drugs persistence.

Background

To this effect in 2014 Virbac undertook a trial (Virbac AEC approval no. 575-14) to examine the use of primer doses of levamisole and benzimidazole with moxidectin long acting (LA) injection in dairy cattle heifers with known resistance in both the *Ostertagia* and *Cooperia* populations of the farm to both doramectin and moxidectin.

In 2013 a dairy property in South East Gippsland was identified as having both *Cooperia* and *Ostertagia* resistance to a range of MLs (eprinomectin pour on (PO), doramectin PO and to a lesser extent but still present to moxidectin LA injection) with the efficacy reduced over the expected period of persistency. The result of this was that within the expected period of protection both *Cooperia* and *Ostertagia* re-emerged in all groups with both the eprinomectin and doramectin requiring a second application due to high worm egg counts (WEC) and significant *Ostertagia* burdens half way through the trial period of 120 days.

Trial

A further study on 94 autumn (March, April) dropped heifers was then carried out on the 6th August 2014 for 90 days to look at the benefits of adding a priming dose of either a levamisole (Lev) oral drench or a benzimidazole (BZ) oral drench to a moxidectin LA injection and was compared with a non primed moxidectin LA injection and a doramectin injection.

The heifers were selected randomly into one of four groups and were within the weight range of 100 to 175kg (mean 132.5 ± 1.52). All animals were assessed as being in good health. The heifers were run as a single group on pasture with some supplementation for the entire period of the trial.

The results, below in table 1, showed an increase in growth rate over 90 days to be significantly improved by the addition of a BZ primer to the moxidectin LA injection.

Table 1. Weight gain over a 90 day period following application of different product combinations in dairy heifers. N=23 per group.

Treatment	Day 35	Day 65	Day 90
Doramectin injection	28.2 ± 1.46^{ab}	55.7 ± 2.37^{b}	76.2 ± 2.18^{b}
Moxidectin LA injection	26.0 ± 1.81^{b}	56.5 ± 2.37^{b}	79.9 ± 2.18^{b}
Moxidectin LA injection plus BZ primer	31.7 ± 1.98^{a}	65.7 ± 2.39^{a}	90.5 ± 2.29^{a}
Moxidectin LA injection plus Lev primer	24.8 ± 1.38^{b}	53.3 ± 2.32^{b}	79.4 ± 2.18^{b}

Results within columns with different superscript letters are significantly different (P<0.05).

Worm egg counts indicated that on day 35, the first monitor after treatment, there was significantly greater parasite control achieved by the moxidectin LA injection groups over the doramectin treatment (Table 2). These differences in WEC were not evident by day 60.

Looking at differentiated WEC's (Table 3), moxidectin LA treatment groups consistently provided greater control over *Ostertagia* at day 35 post treatment, but this effect did not continue and day 65 this difference was no longer significant. There was no apparent effect of priming moxidectin in extending efficacy

Table 2. Cube-root least square means (arithmetic means within parentheses) faecal worm egg counts of heifers treated at day

	WEC			
Treatment	Day 0	Day 30	Day 60	Day 90
Doramectin Injection	4.52 ± 0.83 (165)	6.09 ± 0.47[a] (359)	5.44 ± 0.58[a] (240)	4.80 ± 0.62[a] (187)
Moxidectin LA injection	5.09 ± 0.83 (345)	3.30 ± 0.49[b] (82)	5.52 ± 0.56[a] (244)	3.98 ± 0.59[a] (154)
Moxidectin LA inj with BZ primer	5.98 ± 0.87 (342)	2.28 ± 0.50[bc] (31)	6.54 ± 0.56[a] (416)	4.94 ± 0.59[a] (218)
Moxidectin LA inj with Lev primer	6.75 ± 0.87 (354)	1.80 ± 0.50[c] (38)	6.80 ± 0.53[a] (494)	4.47 ± 0.57[a] (192)

Results within columns with different superscript letters are significantly different (P<0.05)

Table 3. Mean differentiated faecal worm egg counts (WEC) of contributing species for heifers according to the day post treatment and treatment group.

	Day 0		Day 35		Day 65		Day 90	
	Ostertagia spp	*Cooperia* spp	*Ostertagia* spp	*Cooperia* spp	*Ostertagia* spp	*Cooperia* spp	*Ostertagia* spp	*Cooperia* spp
Doramectin	44	117	50	309	34	206	75	112
Mox LA	91	223	0	82	10	234	37	117
Mox LA/BZ	99	243	0	31	42	374	35	183
Mox LA/LV	103	251	0	38	119	376	46	148

Results within columns with different superscript letters are significantly different (P<0.05).

Conclusions

Persistent drugs have a valuable role in improving livestock production in parasite challenged environments. Used inappropriately, like all drugs they carry the risk of reduced efficacy over time due to the development of resistance. Observations from the field show that the period of persistency of drugs reduces as resistance develops, despite maintaining higher initial levels of efficacy after treatment than non-persistent formulations within the same chemical family. To reduce the risk of developing resistance, and to prolong the advantage of production gains that can be achieved by persistent drenches, leading sheep enterprises co-administer a second, non-related drench group. On several of these properties, along with an improved 'head-kill', persistency is apparently maintained longer than when the persistent drug is administered alone.

In theory, the addition of a priming dose of an alternative group should slow the development of resistance. Adopting this practice sooner rather than later would be beneficial to the industry to maintain effective drenches that increase livestock production. Improved weight gain was recorded in the BZ primed group although this was not reflected by a significant decrease in the worm egg counts. BZs are known to be the second most effective group

against *Ostertagia* after the macrocyclic lactones and as this is the most production limiting species in this region it would appear to be the best group to use in combination with ML's as a primer. This preliminary work would indicate that there may be benefits in further studies in this area with cattle drenches.

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HOW TO TREAT BRD IN FEEDLOTS (BY PREDICTING WHEN IT WILL OCCUR)

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Introduction

Bovine Respiratory Disease (BRD) is the main disease syndrome affecting feedlot cattle in Australia and around the world.[1] There are many challenges in managing and treating the condition due to its multifactorial nature and problems identifying individual cases.

A number of studies have examined patterns of BRD in feedlot cattle populations in Canada, the United States, and most recently in Australia.[2-7] Repeatable patterns of disease have been identified and veterinarians working with feedlots can use an understanding of these patterns to better prepare feedlot personnel for dealing with BRD and hopefully achieve better outcomes in the management of the disease complex. The simplest way to explore patterns of disease in feedlots is via epidemic curves.

What is an epidemic curve?

Epidemic curves are a graph showing changes in disease level versus time, in terms of either morbidity or mortality.[8] They are an effective tool for managing disease outbreaks both big and small. The 2001 FMD outbreak in the UK and the Equine Influenza outbreak are examples of where this tool has been used in large scale outbreaks to great effect. However, they have similar utility in managing feedlot disease and a range of other common conditions that cattle veterinarians manage in beef and dairy herds.

With access to a good set of records, veterinarians servicing feedlots can easily create epidemic curves specific to a particular feedlot and update them as necessary. Examination of epidemic curves provides an insight into when disease and mortality is occurring. While there are more sophisticated modelling tools that can be used to explore disease patterns, epidemic curves are relatively simple and straightforward to produce.

Rates of disease

Epidemic curves may be generated using the number of treatments over time as simple frequency distributions. However, determining the **rate** of disease occurrence in a population is more insightful. This implies that you consider both the number of cases that occur (the numerator) and the population at risk (the denominator). Veterinarians servicing feedlots often have access to treatment records (the numerator), but it can be more challenging to access details of feedlot inventory (the denominator) as many feedlot recording systems store this data separately and feedlot managers may require some convincing of the relevance of cattle numbers on feed to you as the feedlot veterinarian.

Fortunately, most feedlot computer systems in use in Australia now have the capacity to export data to Microsoft Excel, where data from different sources can be brought together and calculations performed relatively easily. A key function in Excel that will assist greatly with this process is the FREQUENCY function. There is also a Histogram feature that is an Excel Add-In, part of the Analysis ToolPak, which not only calculates frequency distributions, but guides you through the creation of a histogram – your epidemic curve. Where more complex calculations are required, data may also be exported to a statistical package.

Why is it important to predict when BRD will occur?

While it can appear to feedlot personnel and vets that BRD is a random event, there will be periods when the frequency of occurrence is greater. Knowing when most cases of BRD are likely to occur allows feedlot managers to be prepared and ensure that appropriate personnel are assigned to pen riding duties. If few cases are expected, personnel can be directed to other duties, allowing greater use of human resources. Pen riders can also be directed to focus more closely on pens when BRD is expected to occur improving the detection of sick animals and timeliness of treatments.

What patterns of BRD have been identified in feedlots?

There are two broad patterns of BRD that have been identified:

1. "Days on feed" pattern. Typically, a peak in BRD treatments and mortality will occur soon after cattle are transported to a feedlot and commingled into pen groups. This is why the syndrome is referred to as "shipping fever" in north America.[4] Understanding the shape of these curves and when the peak occurs can be very useful, especially when different classes of cattle are compared. An example would be saleyard derived cattle compared to cattle sourced direct from the property of origin.
2. Temporal or "calendar day" patterns.[3] This can involve examination of day of the week patterns, which, for example, may be useful for monitoring pen riding activities on weekends compared to weekdays. Or it may also be used to explore broader "seasonal" patterns of occurrence. For example, an autumn peak in BRD treatments in Australian and north American feedlots is well documented.[3, 7, 9]

Case definitions

It is important to create a clear definition of what constitutes a "case" of BRD.[2] An example of a case definition for BRD would be that cattle must be subjectively different from their pen mates (depressed and separated from the group), be exhibiting clinical signs only relating to the respiratory system, such as excessive nasal discharge, increased respiratory rate or a soft cough and have an elevated body temperature.

Case definitions need to be clear and easily understood by feedlot personnel. This ensures that they can be uniformly applied. In my experience, feedlot personnel are very good at applying case definitions. They help to "standardise the language" used in the feedlot. All feedlot personnel, including management need to use the same terms and have a clear understanding of what constitutes a case of BRD (or any other condition) on that feedlot. Developing short codes/abbreviations for each case definition also assists staff with keeping notes in pocket books while pen riding.

You may also want to break BRD down into a number of distinct syndromes to allow a more detailed investigation of known specific causes. In a large Canadian study, fatal fibrinous pneumonia, typically associated with the bacteria *Mannheimia haemolytica* was studied as a separate syndrome (based on post mortem findings) because of its significance as a cause of BRD.[3, 4] It is also possible to clinically differentiate infectious bovine rhinotracheitis (IBR) on the basis of additional clinical signs, such as bilateral keratoconjunctivitis and inflammation of the lining of the nasal passages. A further example would be animals with Diphtheria or necrotic laryngitis. These cattle have infections localised to the larynx and upper airways and often exhibit a harsh throaty cough when exercised. Similarly, animals showing acute respiratory distress syndrome which may be cases of Atypical Interstitial Pneumonia (AIP) or tracheal oedema ("Honkers") may also be differentiated from cases of BRD on the basis of the animals exhibiting severe inspiratory dyspnoea. It is especially important to separate out AIP's and Honkers from other cases of respiratory disease because they have different underlying causes, but their occurrence in Australian feedlots appears to be limited compared to north American feedlots.

What recording system should I use?

Records of treatment must be instance based i.e. they must be related to a date and an animal. Australian feedlots now do this well in order to comply with withholding periods and ESI's. However, the format of these records can vary between feedlots, especially small feedlots. Paper based records can still be extremely useful, you just need to transcribe them into a database in order to analyse them. Correctly formatted forms can assist greatly with data capture and transcription. "Smart" scaleheads (e.g. Gallagher and TruTest systems) could also be used to capture treatment data if set up correctly. A range of specialist feedlot data recording systems with animal health modules are also available for purchase and some larger feedlots and veterinary practices have developed their own systems. Whichever way it is done, the aim is to gather the data accurately and consistently.

When is "Day 0"?

For the "Days on feed" epidemic curve, Day 0 for all animals is generally taken as the day of induction or processing into the feedlot. This is generally when they are entered into the feedlot recording system. If a mix of "backgrounded" cattle and cattle that move straight into the feedlot are purchased, you could still use movement into pens as Day 0 but expect to see some cattle getting sick at "negative" days on feed if cattle are identified with BRD during backgrounding.

Care needs to be taken to ensure that feedlot recording systems are not taking the day a pen of animals is first started as “Day 0”. In this case, if a pen is filled over a 2-3 week period, cattle that arrive at say 21 days after the pen is started but get sick 2 days later will appear to be getting sick at 23 days on feed, when in fact they got sick after 2 days on feed. Epidemic curves need to be based on the time each individual in that pen has been “on feed”. If there was very little variation in the time that pens were filled over time, the day of induction of the first animal could be used as “Day 0” but this would be very rare.

Occasionally it may be useful to examine epidemic curves where another time point is assigned as “Day 0”. An example might be when cattle are re-handled at some time point further into the feeding period, such as to re-implant them[10] or when two pens were combined to make a larger pen group.

“First treatments vs All treatments”
It is important to focus only on the time that an animal is first treated for BRD. Including all times an animal is treated will blur the epidemic curve. Check that computer recording systems are only giving you new cases of BRD. If data is being exported into Excel, sorting treatments by date and if necessary filtering out other treatments will quickly identify the date of first treatment for each individual animal. Subtracting the date of first treatment from the date that animal was inducted will give the correct day on feed when the case was first identified.

If animals receive a second or even a third treatment, the proportion requiring re-treatment can be determined (first and second relapse risk) by dividing the number that are re-treated by the number initially treated for BRD.

Market “class”
Feedlots generally feed cattle to meet specific market requirements. Market requirements will dictate weight/age, breed, sex, duration of feeding and ration type/feeding program. As such, cattle in a certain market class generally share a similar risk of developing BRD. Different classes of cattle are typically fed for different time periods and this is often the best feature to group on, e.g. <85 days on feed, 85 to <120 days, etc. with different epidemic curves being created for the different market classes of cattle in the feedlot.

Patterns of disease in Australian feedlots
Meat and Livestock Australia (MLA) and Zoetis have recently funded an investigation into patterns of disease in Australian feedlots.[1] I am able to supply a copy of the report to those that are interested or this can be obtained from MLA. A total of ten feedlots that participated in this broader industry survey of feedlot diseases also documented in this report provided data that allowed analysis of treatment and mortality rates over a 12 month period from July 2009 to June 2010. For this study, the different market classes used by participating feedlots were collapsed down into 4 market classes based on the time cattle spent on feed: <85d, 85 to <120d, 120 to <250d and >250d. All the subsequent figures in this presentation are reproduced from that report.

Pattern of disease by days on feed
An epidemic curve was created for all animals treated by each 7 days (week) on feed, regardless of cause. Calculating by week on feed smoothed out day to day variability (Figure 1). A curve was generated for each of the 4 different market classes.

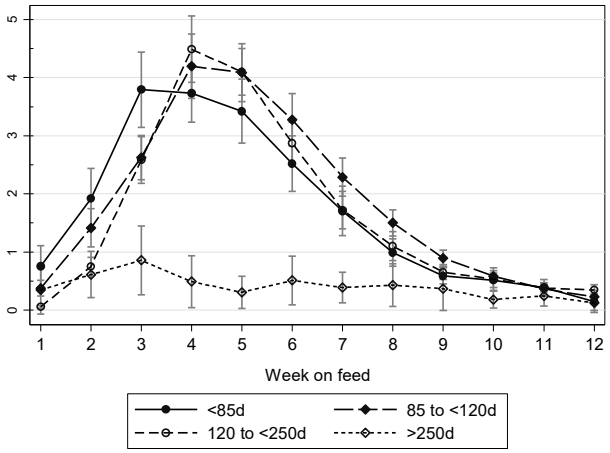

Figure 1: Treatment (morbidity) rate for all conditions combined, expressed as pulls per 100 animal-weeks, arranged by week on feed. Bars represent 95% confidence intervals. Limited to the first 12 weeks on feed.

Figure 2 shows just BRD cases by week on feed limited to the 85-120 day class of cattle. Other classes had very similar patterns for BRD cases. A clear peak in BRD cases at week 4 and 5 on feed is evident. Because BRD constituted 84% of all pulls in study feedlots, the pattern of BRD cases very much drove the pattern of total pulls for any cause.

The epidemic curves (in Figure 1&2) shows treatments or “Pulls” rising from the first week on

feed with a peak at 3-5 weeks on feed, followed by a steady decline. The curve is very similar for the 3 shortest fed market classes. The >250 days on feed class is quite different, appearing to peak at week 3 with a much lower overall morbidity rate. This same pattern is evident for the respiratory pulls in the 85-<120d market class.

Other syndromes classified by body system affected were investigated and further information on these is available in the report. It is important to note that some do not follow a "days on feed" pattern.

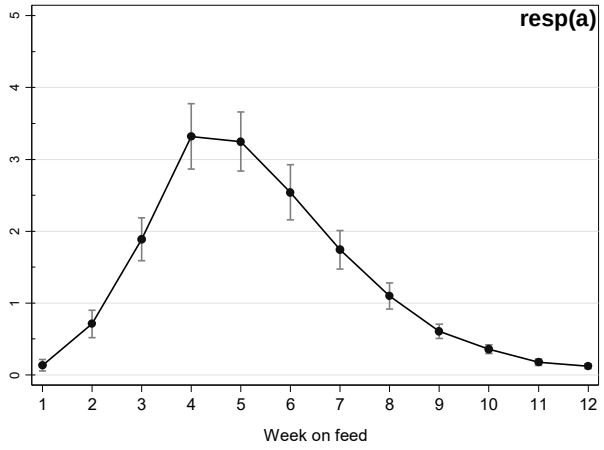

Figure 2: BRD treatment rate (pulls per 100 animal-weeks), arranged by week on feed: for respiratory conditions (resp) limited to those lots that were on feed for between 85 and 120 days and the first 12 weeks on feed. Bars represent 95% confidence intervals.

Mortality rate by days on feed

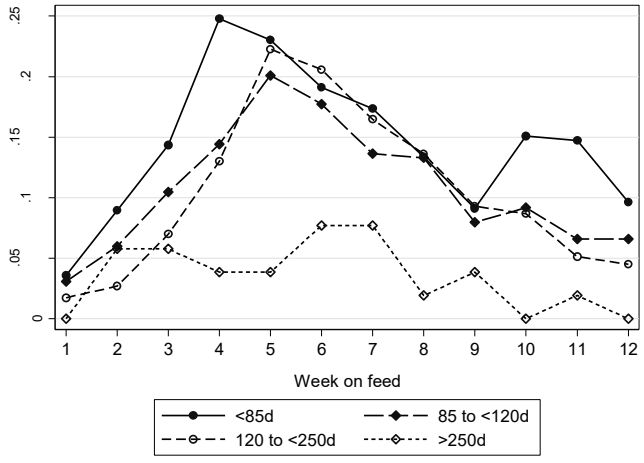

Figure 3: Plot of mortality rate (deaths per 100 animal-weeks on feed) by week on feed and market class. Limited to the first 12 weeks on feed.

The peak in mortality from all causes (Figure 3) occurs at week 4-6 on feed, a week later than the peak in treatments and the mortality curve is largely a lagged image of the treatment curve (Figure 1). For BRD specific mortality, the peak occurs very clearly at week 5 on feed in the 85-<120d class (Figure 4).

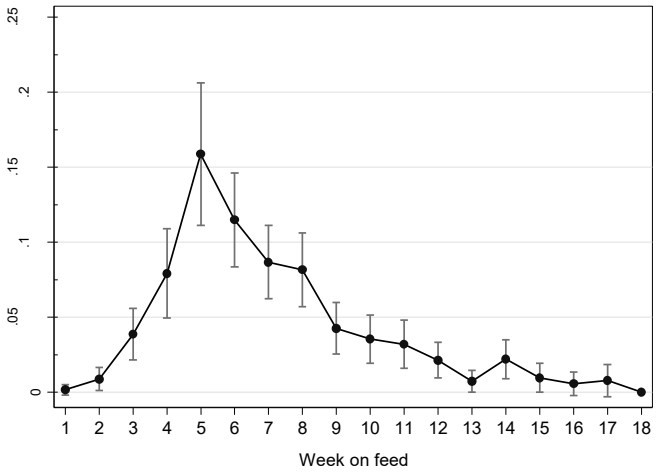

Figure 4: Mortality rate by week on feed for deaths due to respiratory disease only. Limited to those lots that were on feed for between 85 and 120 days. Bars represent 95% confidence intervals.

Relationship between BRD treatment and mortality

Epidemic curves can be used to plot two different factors that you want to compare. Figures 5 shows a very consistent relationship between treatment (pulls) and mortalities from BRD in the Australian feedlot study. Pull rates rise and fall about one week ahead of mortality occurring. This gives us confidence that pulls for BRD are not a random event and are at least temporally linked to subsequent mortality from the same attributed cause.

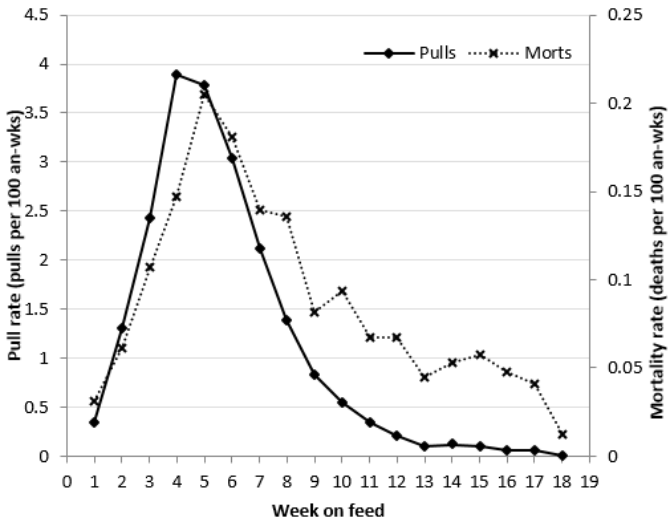

Figure 5: Pull rate (left vertical axis) and mortality rate (right vertical axis) by week on feed for animals on feed for between 85 and 120 days only.

Mortality rate for respiratory disease by calendar month

Because of the close association between BRD treatment and mortality, we will just look at the pattern of BRD mortality by calendar month. The overall mortality rate varied by month of the year in the study feedlots (Figure 6). It was lowest during the late winter and spring and highest in late summer and autumn, with a peak in cattle in their first month on feed in May. Note that because of the "days on feed" pattern to BRD and the fact that most treatments occur in the first month on feed, we have focused on the pattern in cattle in the first month on feed as this takes into account the seasonal pattern of cattle intake.

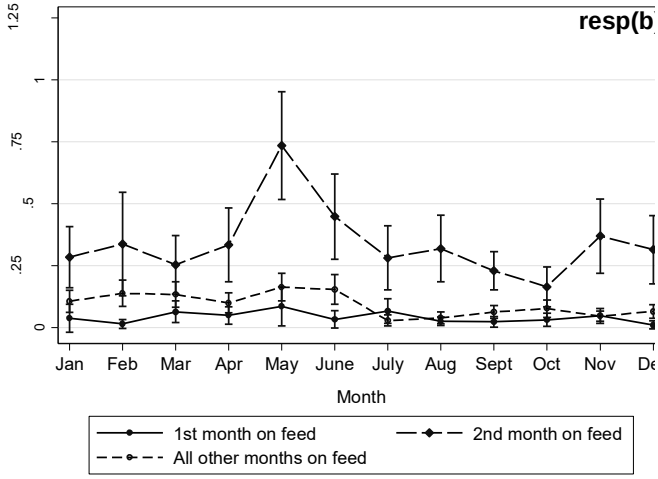

Figure 6: Mortality rate for respiratory disease (resp) (deaths per 100 animal-months) arranged by month of the year and three categories of time on feed. Bars represent the 95% confidence interval.

Pattern of cattle entry to feedlots

Is cattle movement into feedlots seasonal?

Figures 7 and 8 depict the broad pattern for cattle arrival into study feedlots and total cattle on feed. Through the course of a calendar year, total cattle on feed varied from 60-80,000 head in study feedlots. However, when this is broken down into cattle in their first month on feed only, there was an initial peak of arrivals in February-March, followed by a decline to an annual low in June and then a second peak in August. These patterns of arrival are most likely driven by the seasonal availability of cattle. Most calves are born in late winter or spring in Australia and sold in late summer and autumn to make way for the next crop of calves. The surge of young cattle onto the market in summer/autumn drives average selling price of cattle (measured through the Eastern Young Cattle Indicator) and feedlots typically fill up with cattle at this time if market factors are in their favour.

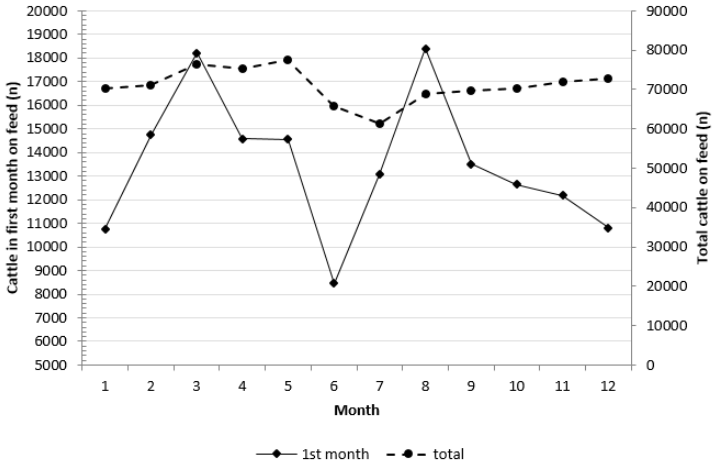

Figure 7: Count of cattle in their first month of days on fed and total cattle on feed, arranged by calendar month of the year.

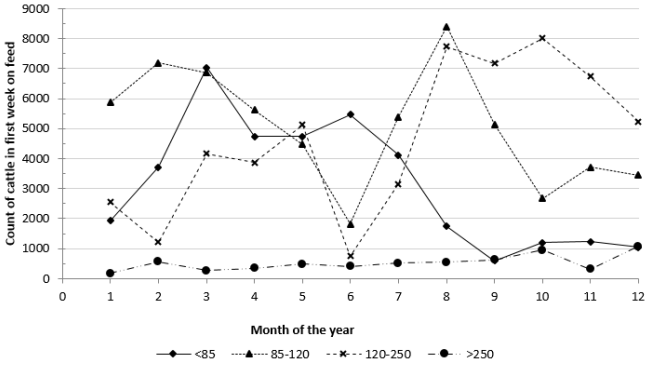

Figure 8: Number of cattle arriving at feedlots arranged by market class and month of the year. Limited tothose cattle in their first week on feed.

Figure 8 provides additional detail on the pattern of incoming cattle by breaking it down by market class. The two shorter fed categories (<85 and 85-120 days) of cattle were the major contributors to the peak numbers of cattle starting on feed in the earlier part of the year. In contrast, the second peak of incoming animals in August was due to a large spike in the 85-120 day class and a large contribution from the 120-250 day class. The very long fed animals had a reasonably constant arrival pattern, possibly with a rise in the second half of the year. This pattern is obscured slightly by the scale of the other arrivals.

The seasonal movement of young cattle into feedlots in Australia explains a large proportion of the apparent seasonal variability in BRD in Australian feedlots and this needs to be further explored.[1]

How to use Epidemic curves for managing BRD

Epidemic curves indicate a clear pattern of BRD occurrence soon after cattle arrive in feedlots in Australia, with the peak in treatments occurring 3-5 weeks after arrival, closely followed by a subsequent peak in mortality. The shape of this curve indicates that a classic propagative epidemic is occurring, with transmission of BRD pathogens occurring in the feedlot environment between cattle carrying these pathogens and susceptible individuals.[9]

In the Australian study, there was also a seasonal peak in BRD occurrence in March/April with a peak in mortality in May. While it is logical that variation in ambient temperature at this time of the year may play a part,[7, 9] it is also important to point out that the most at-risk animals that a feedlot receives may also have arrived in this time period. Perkins was not able to find any significant increased risk of BRD in cattle which arrived in any one season of the year when other risk factors such as arrival weight were controlled for using statistical models.[1] Failure to consider the underlying pattern in cattle movement into feedlots (the denominator) could lead to false interpretation of an analysis based solely on treatment records (the numerator).

The most logical use of this information is to prepare feedlots for BRD. If a feedlot purchases a large number of cattle at any time of the year, it is likely to be followed soon after by a corresponding peak in BRD. This peak in buying activity generally occurs in autumn, but occasionally happens at other times of the year if market forces dictate.

Feedlot managers need to be ready for this by ensuring they have sufficient staff allocated to the management of sick cattle during this peak time. Often they are caught flat footed as they are also struggling with the induction and feeding of large numbers of cattle.

In Canadian feedlots, a peak in BRD treatment and mortality occurs in November, late in their autumn when their feedlots are filled to capacity.[3] They have documented that the case fatality risk for BRD, i.e. the proportion of cattle treated for BRD that went on to die, increased at this time of the year relative to other time points. One of the theories proposed by the study authors was that resources available to manage BRD cases were at or past their capacity at this time, resulting in a lower level of treatment success.

Australian feedlots may be able to improve the success of BRD management during the autumn months by adopting programs that reduce labour requirement, such as the use of single dose antimicrobial therapies, with treated cattle immediately returned back to their home pen to recover. Likewise, preventative vaccination programs may be strategically employed on arrival to reduce the number of cases of BRD using vaccines that work after a single dose. The delayed peak in BRD in Australian feedlot populations, compared to the earlier peak typically seen in cattle in the northern hemisphere, allows more time for vaccines to become effective when given on arrival.

Pre-feedlot vaccination programs are also an option, but are often not applied to the <85d class of cattle which, for a range of reasons (age, often sourced direct from saleyards), are at the highest risk of BRD morbidity (treatment) and mortality. Vaccination on arrival is often the only available option to manage BRD in this class of stock.

Conclusions

Epidemic curves can be a valuable tool in the day to day management of BRD in feedlots. If veterinarians are familiar with their correct development and interpretation, the information they provide may improve the success of management of BRD in their clients' feedlots.

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HOW I TREAT LOW FERTILITY IN YEAR ROUND CALVING DAIRY HERDS: A PROBLEM BASED APPROACH

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Reproductive losses are of considerable concern and frustration to year round calving herd dairy managers. A high level of reproductive performance usually means:

- Freshly calved cows enter the herd every month or in even batches through the year. Most milk processors in year round calving areas require an even supply of milk throughout the year and hence offer payment systems that discourage uneven calving patterns.
- The overall proportion of stale cows in the herd is kept to a minimum. Stale cows have lower production and lower feed conversion efficiency. Improved reproductive performance for an individual cow means that more of her lifetime will be spent in early lactation.
- Minimal cows empty 200 – 300 days after calving as these will frequently be culled.
- High conception rates to reduce semen and insemination costs.
- Adequate high genetic merit replacement heifers (25% of total herd size) being born and reared each year.
- Minimal frustration and effort involved with getting cows in calf.

Reproductive performance in many Australian year round calving herds has declined over the past 20 years. Figure 1 compares the 100 day in-calf rates for a number of herds on the Atherton Tableland during 2015. Few of the herds are achieving the target set by Dairy Australia's InCalf project of 58% which in 1997-1998 was the median of the top quartile of farms involved in that study[1, 2]

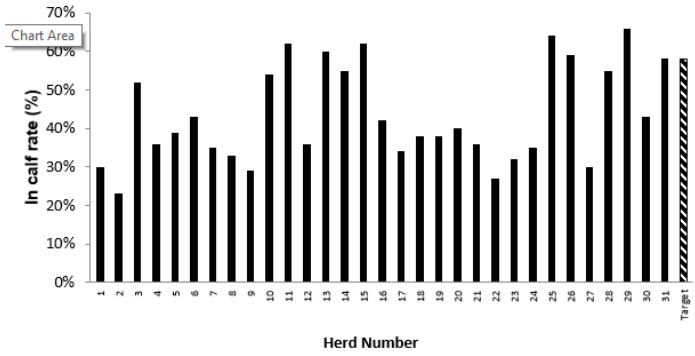

Figure 1. Comparison of 100 day in-calf rates for Atherton Tableland herds Median 40% (23 – 66%)

Achieving acceptable in calf rates is dependent on achieving high submission rates and moderate conception rates. Efforts aimed at "treating low fertility" must focus on identifying the most likely risk factors for both, or either, low submission rates and low conception rates and then developing strategies for managing them. This requires detailed and routine record analysis, regular cow examinations including early pregnancy diagnosis and familiarity with attributes of the herd's management. Knowledge of the herd's feeding program, the herd manager's style of management and the calibre of labour employed are all helpful in designing strategies for improving herd reproductive performance.

This paper describes our experiences working with year round calving dairy herds on the Atherton Tableland in North Queensland.

Regular monitoring of herd performance.

Figure 2 is an example of the farm monthly monitor sheet that we use. It contains as many of the most important pieces of information about each herd's performance as can be fitted on one page. The cells are filled in each month as results of record analysis become available.

The middle third of the page focuses on recent reproductive performance of the herd. A quick review of this monitor sheet before each of our herd visits provides background information about the herd, highlights areas of shortcomings and helps identify areas where there are opportunities for improvement.

MONTHLY PERFORMANCE INDICATORS

2015

Uluru

	HERD STATUS					PRODUCTION			HEIFERS	
	Number Milking	Av DIM	% Milking	%Cows Pregnant	%Calved >150 Empty	Production @ 40-70 DIM	Total Litres/day	Litres/cow per day	Age at Calving	% Mature Prodn
Jan-15	306	178	74	48	12	27.2	7313	23.9	26	84
Feb-15	296	178	75	45	15	29.8	6785	22.9	24	85
Mar-15	293	159	71	42	22	33.2	6805	23.2	27	80
Apr-15	282	163	76	39	24	-	6882	24.4	27	-
May-15	281	162	75	40	31	31.4	7279	25.9	27	77
Jun-15	297	168	80	49	21	-	7879	26.5	26	-
Jul-15	326	171	80	48	16	32.2	8474	26.0	23	79
Aug-15	337	169	83	44	17	31.0	8748	26.0	22	82
Sep-15	323	177	85	51	14	35.3	9089	28.1	27	83
Oct-15	319	187	80	52	11	34.4	8666	27.2	27	85
Nov-15	306	182	81	55	10	-	7845	25.6	27	-
Dec-15	298	178	80	53	11	-	7018	23.6	-	85
Average	305	173	78	47	17	31.8	7732	25.3	26	82
Target		160 days	83%	> 65%	< 10%				26	85%

	REPRODUCTIVE PERFORMANCE						Fresh Cow Components					
	80 Day SR	Average Return Interval (days)	Conception rate		100 Day ICR	200 Day NICR	0-30 DIM		50-90 DIM		BCS @ calving	
			1st AI	All Services			Acidosis %F:P<1	Ketosis %F>4.75	Acidosis %F:P<1	ME or MP %P<2.9	% < 4.5	% loss > 0.9
Jan-15	74	24	27	21	44	16	11.1	0.0	7.4	55.6	5.4	8.5
Feb-15	81	25	30	23	31	29	7.6	17.6	19.0	25.0	6.7	3.6
Mar-15	69	29	29	22	59	18	21.9	21.1	0.0	13.3	5.4	6.1
Apr-15	78	26	35	32	56	15	-	-	-	-	3.2	3.0
May-15	71	25	40	34	32	29	7.9	29.3	7.5	0.0	6.1	18.4
Jun-15	79	25	33	26	39	39	-	-	-	-	3.2	15.5
Jul-15	89	27	41	35	78		13.8	17.2	5.8	11.5	5.3	12.9
Aug-15	63	26	40	48	44		11.8	11.1	16.4	13.7	1.5	13.6
Sep-15	76	26	45	33	53		9.1	20.0	15.4	28.2	0.0	14.3
Oct-15	69	26	43	37	38		42.5	7.7	26.3	13.6	5.3	15.1
Nov-15	53	25	23	28			-	-	-	-	8.7	12.5
Dec-15	60	30					-	-	-	-	12.5	19.5
Average	72	26	35	31	47	24	15.71	15.5	12.23	20.1	5.3	11.9
Target	>73%	<30 days	>51%	>51%	>58%	<13%	<20%	<20%	<20%	< 20%	<15%	<10%

	MILK QUALITY								
	Mastitis							Milk Composition	
	% Cows Infected		Mast Cases per 100 Cows	Fresh Cows Clinical	Bulk Cell Count			Fat%	Protein %
	All Cows	Lact. One							
Jan-15	21	25	11.6	19	166	156	163	4.05	3.33
Feb-15	19	17	14.7	24	211	197	193	4.08	3.35
Mar-15	21	21	15.4	21	214	195	163	4.05	3.35
Apr-15	-	-	7.4	17	162	175	192	3.95	3.31
May-15	23	21	7.5	7	185	177	184	4.02	3.32
Jun-15	-	-	5.8	13	202	201	186	4.17	3.32
Jul-15	21	11	7.4	10	175	162	181	4.10	3.31
Aug-15	21	10	6.6	19	187	184	158	4.06	3.38
Sep-15	16	10	5.7	10	124	153	134	3.85	3.34
Oct-15	20	16	4.7	8	136	164	154	3.91	3.43
Nov-15	-	-	8.8	14	163	170	186	3.85	3.32
Dec-15	-	-	9.3	10	192	199	212	3.71	3.30
Target	< 20%	< 10%	<2.0	< 5.0	< 200,000 cells / ml			>3.95%	>3.15%

Figure 2: An example of a monthly monitor sheet

1. Treating low submission rates:

Low submission rates are identified by:

- 80 day submission rates < 60%
- Average inter-service intervals > 30 days

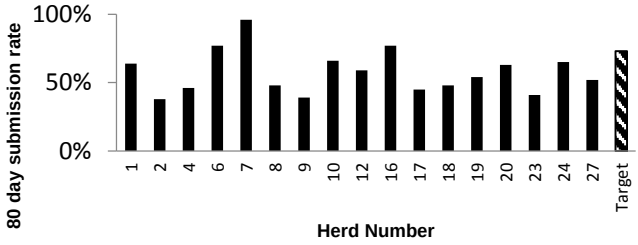

Figure 3. Comparison of 80 day submission rates of Atherton Tableland herds. Median 54% (38 – 96%)

When submission rates are low we direct our attention to the following most likely risk factors:

A. Shortcomings in heat detection

Confirmed by:

- Percentage of cows pregnant at pregnancy diagnosis <75%
- Percentage of return intervals of length 18-24 days < 45%
- Less than ideal use of heat detection aids
- Cows in acceptable body condition examined for no visible oestrus with active corpora lutea (dioestrous)

Treatment:

- Trial various heat mount detectors or tail paint until the herd manager finds one that he/she likes to use.
- Facilitate staff training on the topic of heat detection: when and what to look for, how to interpret partially rubbed tail paint or partially activated heat mount detectors, when to inseminate and when to leave, what system will be used to communicate observations from one staff member to another especially when milking shifts change from milking to milking.
- Encourage the use of a tail tape tagging system (as described in the "InCalf book for dairy farmers"[1]) to identify cows that require twice daily inspection of heat detection aids.
- Become familiar with the benefits and limitations of automated heat detection systems and activity meters so as to assist herd managers in making informed decisions about these investments.
- Adopt routine oestrus synchrony with fixed time insemination to avoid the need for heat detection at all for the first oestrus.
- Use early (30-32 day) pregnancy diagnosis to identify empty cows early and immediately resynchronise these cows for fixed time insemination.

B. Low body condition at calving

Confirmed by:

- > 15% cows with BCS <4.5 at calving

Whenever possible at herd visits cows are body condition scored:

- When cows are rechecked to confirm pregnancy prior to drying off
- In the close-up springer paddock to record the body condition pre-calving
- Pre- mating. This is a more difficult time to score cows on a routine basis but can be done easily in those herds using routine oestrus synchrony programs when our vets are involved in this process.

Cows are identified by the month or, in smaller herds, by the season of their calving. For example, cows calving in April 2016 will have:

- An average score at drying off (recorded at pregnancy recheck in February): % >5.5 and % < 4.5
- An average score precalving (recorded as springers on transition feed in April): % >5.5 and % < 4.5
- An average score premating (recorded at the time of oestrus synchrony in June) to calculate average BCS loss between calving and mating

Treatment:

- If the problem relates to low body condition scores at drying off:
  - Alter feed inputs in late lactation
- If the problem relates to loss of body condition during the dry period:

- Optimise the transition cow diet with an emphasis on dry matter intake (DMI) and energy density of the ration prior to calving (see the "Transition cow management" handbook[3]).
- Encourage removal of passengers (e.g. cull cows) that may be competing with dry pregnant cows for limited pasture

- Treat anoestrous cows to induce cycling using a normal Ovsynch plus progesterone ten day program:

  Day -10 : inject GnRH, insert P4 device

  Day -3 : inject PG, remove device

  Day -1 : PM Inject GnRH

  Day 0 : MSD – Fixed-time AI

C. Excessive BCS loss and energy deficiency post calving

Confirmed by:

a. Average BCS loss between calving and premating > 0.6
b. >20% cows calved 0 – 30 days with milk fat% > 4.75%[4]
c. >20% cows calved 50 – 90 days with milk true protein % < 2.9%[4]
d. >20% cows 5 - 50 DIM with a Beta-hydroxybutyrate (BHB) > 1.4mmol/l[5]
e. Increase in peri-parturient disease incidence (see section 1E). These can be a cause or effect of excessive body condition loss.

Treatment:

- Optimise transition cow management prior to calving with particular emphasis on DMI, energy density and fat content of the diet and the use of ionophores. If peri-parturient disease incidence is high, additional emphasis on macro mineral nutrition will be necessary.
- Minimise the number of cows that are over conditioned at calving (>5.5 / 8).
- Consider all feeding options including pasture/forage allocations, supplementation with concentrates and ionophores, herd splitting etc. to maximise energy intakes during the early lactation period.

D. Heifers failing to meet live weight targets at calving

Confirmed by:

- >15% heifers calving with BCS < 4.5
- First lactation milk production yields < 83% of mature cows

Treatment:

- Focus on all aspects of calf and heifer nutrition and management to ensure the best possible growth rates are achieved with the resources that are available using the "Heifers on target" manual[6].
- Delay first mating until maiden heifers have reached their target weights for mating. If there is the possibility of below target weight gains post conception then delay first joining until weights are well above industry target mating weights.
- If scales are available check pregnant heifers are on target to reaching desired calving weights. If weights are below target separate lighter heifers and provide supplementation.
- Ensure maiden heifers are inseminated to bulls with calving ease ABVs > 103 or paddocked with bulls with low breed related risks of dystocia.

E. High incidence of post calving disease events

Confirmed by:

a. Incidence of retained foetal membranes > 6%
b. Incidence of metritis > 10%
c. Incidence of lameness in first 100 days lactation > 4%
d. Incidence of hypocalcaemia > 3%

Treatment:

- Increases in peri-parturient diseases are commonly associated with disorders of lipid mobilisation (e.g. Ketosis) and/or macro mineral homeostasis (e.g. Milk fever). It is therefore important to evaluate and optimise all aspects of transition cow management. Ensure the diet is formulated to meet the

cow's requirement for metabolisable energy (ME) and protein (MP), dietary cation and anion difference (DCAD) and selected macro minerals (calcium, phosphorus and magnesium).
- Ensure lameness prevention is being targeted by laneway maintenance and staff training in patient cattle handling.
- Ensure treatment protocols are in place to ensure cows that are sick, off feed or lame are treated by farm staff promptly.
- Routinely examine cows suffering from any calving related disorder by metricheck between 14 and 40 days post calving and treat cases of endometritis appropriately.

2. Treating low conception rates

Low conception rates are identified by:

- First service (following last calving) conception rates < 35%
- Total Service conception rates < 35%
- AI Conception rates in maiden heifers < 50%

Conception rates have declined over the past 25 years[7]. Figure 4 below shows the total service conception rate for artificial inseminations in a number of herds on the Atherton Tableland for 2015. None of the herds achieved the InCalf target of 51% which was based on the conception rates from the top 25% of herds in the 1997-98 study[1, 2]. Figure 5 shows the first service conception rate over time for one herd in which most inseminations have been performed by the same technician. During the same 24 year period the conception rate in maiden heifers with inseminations by the same technician have remained relatively constant.

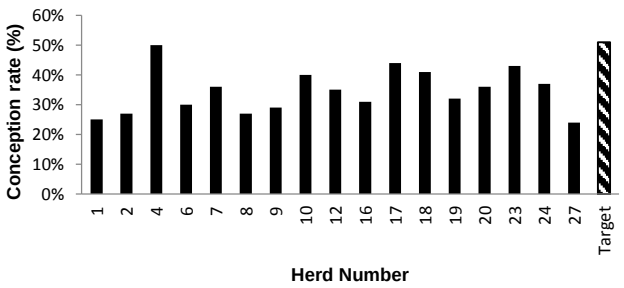

Figure 4. Total service conception rates to artificial inseminations in Atherton Tableland herds for 2015. Median 35% (24 – 50%)

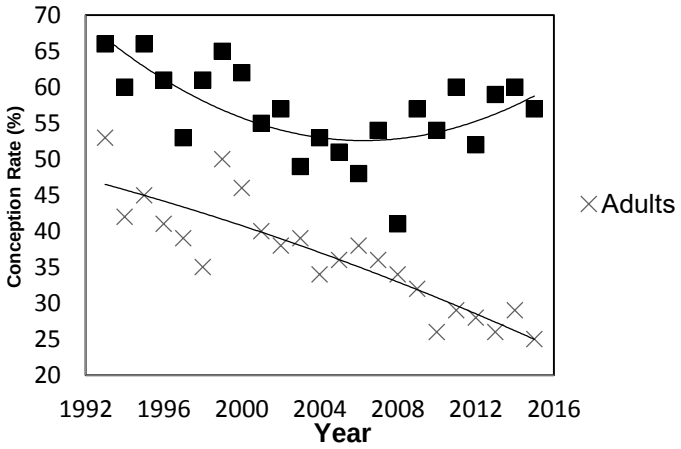

Figure 5. Comparison of conception rates between maiden heifers and adult cows over a 24 year period for one Atherton Tableland dairy herd

Our experience is that it is much more difficult to identify strategies to improve conception rates than it is to improve submission rates. When rates are low we direct our attention to the following most likely risk factors:

A. **Errors in heat detection** – cows inseminated when not in oestrus – or poor timing of insemination.

Confirmed by:
- % of return intervals shorter than 18 days > 15%
- An increase in the number of cows pregnant to the second or third last service at pregnancy diagnosis

Treatment:
- Facilitate staff training on the topic of heat detection: how to interpret partially rubbed tail paint or partially activated heat mount detectors, when to inseminate and when to leave, what system will be used to communicate observations from one staff member to another especially when milking shifts change from milking to milking.
- Trial a fixed time insemination program using oestrous synchrony

B. Excessive body condition loss and energy deficiency post calving

Confirmed by:

a. Average BCS loss between calving and premating > 0.6

b. >15% cows calving with a BCS < 4.5
c. >20% cows calved 0 – 30 days with milk fat% > 4.75%[4]
d. >20% cows calved 50 – 90 days with milk true protein % < 2.9%[4]

Treatment:

- See section 1C.

C. Suboptimal insemination practices

Confirmed by:

- Comparing the conception rates achieved by technicians who have performed more than 50 inseminations in the herd. Differences in conception rate > 15% are likely to be significant
- If maiden heifers are inseminated and conception rates are <50% for any technician who has performed > 50 inseminations

Treatment:

- Facilitate a review of AI practices used by technicians on the farm. In our experience the most common faults include:
    - Continuously raising semen straws above the frost line in the AI canister. Our advice is: “Install a good light over the tank and buy a pair of glasses”
    - Incorrect thawing temperature. Periodically be sure to check that the temperature reading from the thermometer being used agrees with that of a second thermometer
    - Poor hygiene. Dirty equipment. Failure to adequately wipe the vulva clean and open the lips of the vulva before inseminating
    - Failure to deposit all the semen gently into the body of the uterus. Do it yourself (DIY) technicians often forget how short the body of the uterus is and insert the insemination pistolette too deep up one horn. Reviewing the anatomy of a reproductive tract with technicians, using specimens collected from an abattoir can be a valuable refresher.

D. Failure to select for genetic improvement in fertility

Confirmed by:

- Failure to select service sires that have ABVs for daughter fertility > 105.
- Check the Genetic Progress Report produced by ADHIS for the herd to discern the herd progress in the herd fertility ABV.

An extract from a farm’s Genetic Progress Report is shown in figures 6 and 7. This Holstein herd has achieved gains in the Balanced Performance Index (BPI) over the past ten years similar to that of the top 10% of Holstein herds in Australia (figure 6). The BPI reflects the economic drivers of net profitability for a range of dairy farming systems and includes the traits of production, type, milking speed, temperament, cell count, fertility and feed efficiency. The herd depicted has made progress well above the national average for traits such as type, longevity and mastitis resistance but has flat-lined for fertility and remains well below the national average (figure

Treatment:

- Select AI sires that have ABVs for daughter fertility > 105

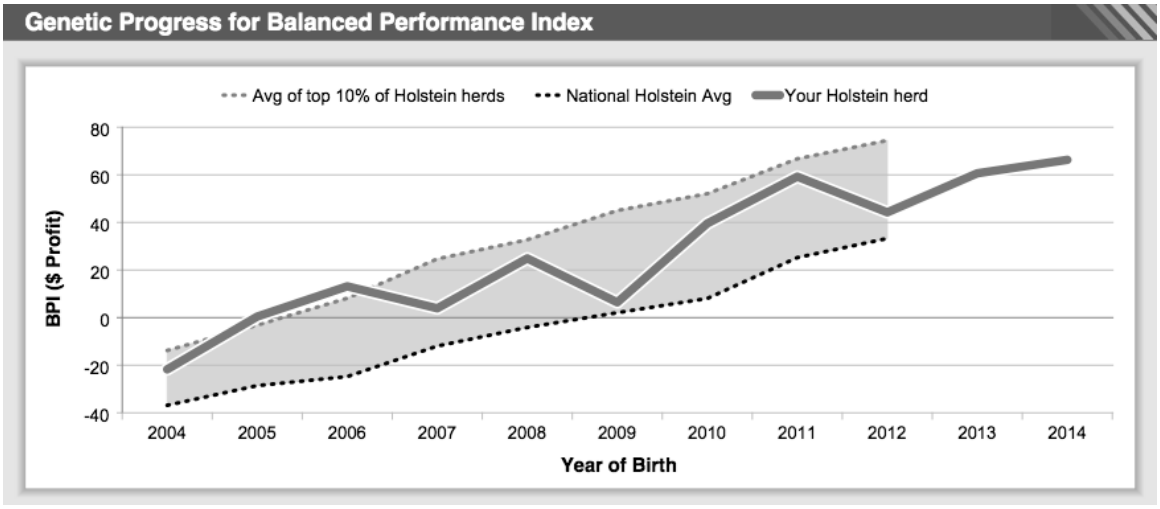

Figure 6. Genetic progress report for Balanced Performance Index (BPI) comparing one herd's performance with the national average

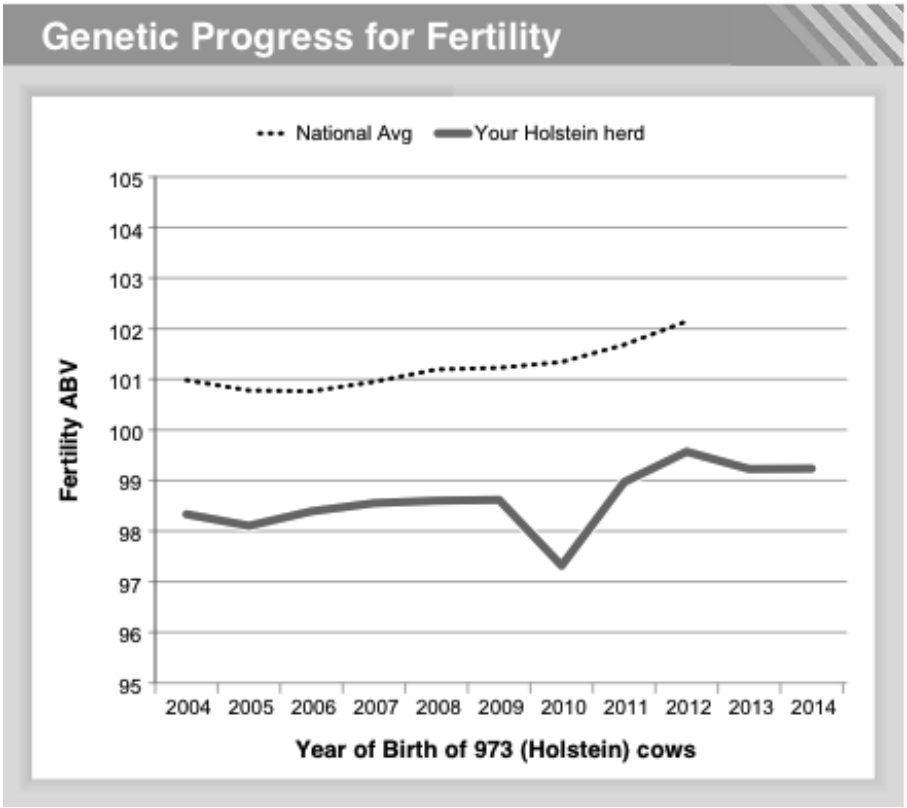

Figure 7. Genetic progress report for fertility comparing one herd's performance with the national average

E. Heat stress

Confirmed by:

- Lower conception rates in summer vs winter
- Clinical signs of heat stress in the herd: cows respiratory rates >60, open mouth breathing, seeking shade, reduced DMI and production.

Treatment:

- Refer to Dairy Australia's manuscript, "Cool Cows"[8]
- Ensure appropriate sprinklers are used in the holding yard at milking time.
- Avoid milking cows in the hot times of the day.
- Consider infrastructure options for providing shade.
- Evaluate the milker ration: supplement with high quality fibre, slower fermenting

starches, increase sodium and bicarbonate in the ration.

F. Failing to cull repeat breeders

Confirmed by:

- All services conception rate significantly lower than first service conception rate.
- Many cows in the herd with >5 services

Treatment:

- Establish a culling protocol with critical limits (e.g. cull after 5 services)
- Assist the farmer with identifying potential culls during the reproductive visit
- Make sure farm staff are reviewing how many services a cow has had before inseminating again.

3. Managing high foetal loss rates between pregnancy test and calving

Abortions and embryonic mortality remain a major cause of reproductive failure, frustration and financial loss for our dairy herds, despite continuous effort to identify causes and develop strategies to prevent them.

High abortion rates are identified by:

- Pregnancy loss rates between early pregnancy diagnosis and calving of > 8%. Some consideration needs to be given to the stage of gestation when the pregnancies are confirmed. More cases of early embryonic loss will be detected when the average stage of gestation at pregnancy test is 33 days (weekly pregnancy tests between 30 and 35 days) than when it is say 50 days (monthly pregnancy tests between 35 and 63 days).

Figure 8 shows the abortion rates for some Tableland herds through 2015.

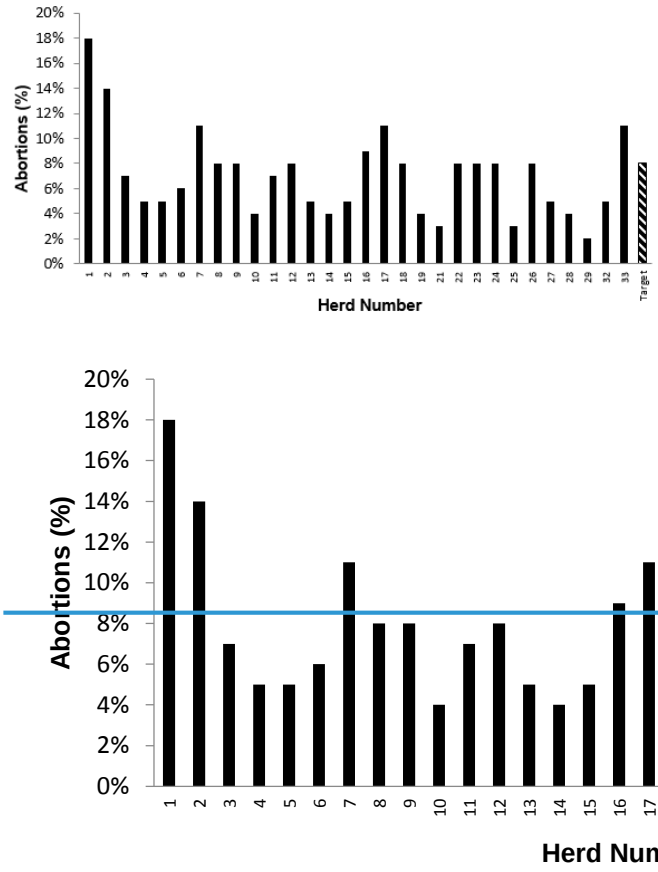

Figure 8. Crude abortion rates for a number of Atherton Tableland herds in 2015. Median 7% (2 – 18%)

When pregnancy loss rates are high we direct our attention to the following possible causes, controlling them when there are cost effective strategies to do so. Having done that we then encourage our clients to learn to live with the frustration of abortions and manage their herds with the adage:

"Control the controllable. Only worry about things you can do something about".

Treatment:

- Explain to herd mangers that all herds expect some pregnancy loss – the earlier the diagnosis of pregnancy the greater the loss to be expected. It is important to assert that early diagnosis of pregnancy does not cause early embryonic loss.
- Perform foetal necropsies whenever possible.
- Ensure there is no active Bovine viral diarrhoea (BVD) infection in the herd. We encourage all of our herds to do a bulk milk BVDV antibody ELISA every year. Significant rises in sample to positive (S/P) ratios are investigated and if necessary an eradication program is initiated. Some herds

routinely ear notch all heifer calves to be reared to ensure no persistently infected (PI) animals are reared. Serological surveys of unmated heifers are sometimes performed as another check to confirm the absence of any PI animals.

- Understand the Neospora status for herds with high abortion rates. We explain that Neospora infected cows are three to four times more likely to abort than non-infected cows[9]. Attempts at eradicating Neospora from herds, or even dramatically reducing the prevalence by selectively breeding replacement heifers from only Neospora negative dams, have been frustratingly unsuccessful because of new infections via horizontal transmission. Further the cost of routine serology is uneconomical except in certain circumstances such as embryo transfer recipients. Therefore our efforts tend to be aimed at reducing dog-associated infections.
- When natural mating is used, all bulls are routinely vaccinated annually for Vibriosis (Bovine venereal campylobacteriosis). If in-calf rates are below target in naturally mated herds vaginal swabs from repeat breeders are tested for Campylobacter using PCR or IgA ELISA. If any cows are tested positive then a whole herd vaccination program is implemented for the following two years and bulls are treated with an intraprepucial infusion of cephaparin (Metricure).
- Serological testing in our herds have shown that infections with *Leptospira pomona* are rare but infections with *Leptospira hardjo* are common. There is usually little evidence that *hardjo* infections contribute much to pregnancy loss. Nonetheless herds are encouraged to vaccinate for Leptospirosis primarily to prevent zoonotic infections in farm staff.
- Other possible causes of abortion are rarely identified. Once the known infectious agents have been ruled out herd managers are encouraged to stop focusing on the problem. Cull cows that abort twice. Rear some extra replacements to offset the losses.

CONCLUSION

Without routine farm visits and the maintenance of accurate herd data, many of the problems discussed above cannot be easily and quickly detected. The main drivers of reproductive performance are submission rate and conception rate. Of these two, farm managers usually have most influence over the submission rate through heat detection strategies, synchrony programs, policy on voluntary waiting period, transition nutrition management and peri-parturient disease prevention. Common factors affecting conception rates include heat stress during summer months, faults with semen handling and struggles with DIY AI technique, accuracy in the timing of insemination and nutritional inputs. These influences are often less easily manipulated.

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HOW I FIX FRACTURES IN THE FIELD

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Introduction

Fractures in cattle often result from trauma sustained during dystocia, handling, or trampling by other cattle.[1-3] Optimal treatment may entail radiography and / or referral to a specialty practice, but economic or logistical issues may limit these options. The veterinarian must then determine if the external fixation methods employable in field practice carry a reasonable prognosis for success, and if treatment is justified on humane and economic grounds. The signalment, intended use, and history are vital considerations in this decision. If flawed animal handling, obstetrical techniques, or facilities management contributed to the event, a teaching opportunity arises – farm personnel can be educated on how to prevent future injuries.

A thorough physical examination must follow, including evaluation of the location of the fracture, the status of the bone (open vs. closed, comminution), and the status of the nerve and blood supply of the limb. One should look for other injuries and concurrent diseases.[2] Newborn calves must be carefully examined for failure of passive transfer and for congenital defects, which often preclude the justification for fracture treatment.

Prognosis

Cattle are generally considered to be good orthopedic patients, owing to their tendency to rest in recumbency for prolonged periods.[1,2,4] However, fractious cattle carry significant potential for self-inflicted injury and treatment failure.

The metacarpal and metatarsal bones III/IV are the most commonly fractured bones of cattle.[1,2] Disruption of the nerve and blood supply to the distal limb can accompany fractures caused by obstetrical chains.[4] A good prognosis is warranted for closed fractures with minimal comminution and an intact neurovascular supply in calves. Highly unstable fractures and fractures in heavier cattle warrant a guarded prognosis and warrant consideration of cast reinforcement methods and/or application of a Thomas splint-cast combination (TSCC).

If the fracture is located in the distal physis of the metacarpus or metatarsus, a cast that extends to the proximal aspect of that bone (a “half limb” cast) can provide sufficient immobilization; however, for fractures of the diaphysis, the cast should extend to the proximal radius or tibia, respectively (a “full-limb” cast).[1]

Metacarpal and metatarsal fractures can easily become open fractures, given the limited soft tissue cover. Open fractures with a fresh wound bed should be debrided and lavaged. A sterile bandage should be applied, and the limb should be immobilized appropriately. Administration of long-term antimicrobial therapy is necessary. Cast removal in 1-3 week intervals may be required to allow for further management of the wound. Serial evaluation of animal comfort, gait, rectal temperature, and appetite facilitate judgment of progress. Longstanding sepsis of an open fracture warrants a poor prognosis.

Fractures the radius / ulna or tibia cannot be successfully treated with casts because the proximal end of the cast creates a destabilizing fulcrum effect on the fractured bone. A TSCC allows weight on the fractured limb to be transferred from the ground to the inguinal or axillary areas. It is considered to be the sole option for field-based external coaptation of these fractures.[2,5,6] Lateral deviation of the healed bone and axillary / inguinal pressure sores are potential complications.[5,6] The duration of time for healing in a TSCC was found to range from 5-13 weeks.5 The prognosis for healing is good for closed fractures in lighter cattle, but guarded for larger adults and open fractures.[5,6]

Owing to the close proximity of the radial nerve to the spiral groove of the humeral diaphysis, humeral fractures carry considerable risk of potentially permanent radial nerve damage.[4] Femoral fractures commonly occur in calves delivered by forced extraction during dystocia; concurrent femoral nerve damage may result from severe overextension of the hindlimbs. For humeral and femoral fractures, contracture of the heavy surrounding musculature typically produces severe overriding of the fracture ends.

There are no external fixation methods that are appropriate for management of humeral or femoral fractures in the field. Although occasional return to function has been reported with several (12+) weeks of stall confinement, success with this form of management appears inconsistent; further, there is no easy way to identify beforehand which affected animals might respond well and which will develop nonunion or complications resulting from prolonged recumbency.[2] Therefore, if internal fixation is not an option, euthanasia is warranted for most cases of femoral or humeral fracture.[2]

Principles of cast application

For cast application, the animal is sedated, anesthetized, and restrained. Preparation of the limb for casting requires that mud and manure be washed or brushed off. The interdigital space should be cleaned and dried. Two-layer stockinette or foam resin cast padding should be applied to the skin. Application of excessive padding may paradoxically promote the development of cast sores and nonunion, as compression of padding may result in movement of the limb within the cast.1 For "half-limb" casts, felt padding should be applied only to the dewclaws and to the top of the cast; these are taped in place over the stockinette. Holes can be cut in the stockinette and felt to allow the dewclaws to protrude. For full limb casts of a fore limb, additional padding is needed over the accessory carpal bone and styloid process of the ulna. For "full limb" casts of a hind limb, additional padding is required over the calcaneus and medial and lateral tibial malleoli.[1]

In calves, a wire saw can be placed within the cast to facilitate subsequent cast removal. Two lengths of obstetrical wire, each measuring approximately 1.5 – 2 X the length of the cast, are placed within plastic intravenous fluid tubing. One wire is then taped along the longl axis of the limb on the medial side of the surface of the stockinette or foam cast padding, such that extra wire will protrude from the top and bottom of the cast. The second wire is taped in a similar fashion along the lateral aspect of the stockinette or padding. The cast is applied over these tube-encased wires, and the excess wires that protrude from each end of the cast are then wound into tight coils and taped securely to the external surface of the cast. For cast removal, vise-grip pliers are secured to each end of the medial wire and broad, back-and-forth strokes are used to saw through the cast from interior to exterior. The process is repeated for the lateral wire, and the two halves of the cast are removed. For very thick casts, the wires may heat and break before complete cutting is achieved.

To maintain alignment and tension on the limb, an assistant can place traction on wires placed through holes drilled through the toes of the hooves. Alternatively, in calves, adhesive tape can be applied lengthwise to the dorsal and palmar or plantar surface of the limb and then apposed together at the toes to create a traction device. This can be held in traction or secured to a fixed object. The foot should always be included in the cast.[1] During application, slight tension should be maintained on the fiberglass cast material, creating slight stretch in the material. Each wrap of the material should overlap its predecessor by ~50%.

The thickness of the cast is determined by clinical judgment. Calves less than 150 kg may need a cast that is 6-8 layers thick; however, 12-16 layers may be necessary for adults.[1] Full limb casts on the hind limb must be made even thicker in order to compensate for force concentration created by the angle of the hock.2 A U-shaped metal bar (a "walking bar") can be placed under the solar surface of the casted hoof and integrated into the cast to help dissipate force away from the distal limb. In heavier cattle, incorporation of metal rods lengthwise into the cast may increase its strength. Acrylic cement can be applied to the sole of the cast to add durability.

Confinement to a stall or small corral is recommended to minimize rub sores. The date of the next veterinary examination or anticipated cast change should be printed in indelible ink or paint on the surface of the cast. Cattle should be monitored closely for changes in ambulation, increased time spent in recumbency, reduction in appetite, malodor, and fever, as these may indicate pressure sores, compounding of the casted fracture, or septic osteomyelitis. Casts can remain on calves for 3-6 weeks, depending on the rate of growth and patient comfort.[1] In calves, clinical union of the fracture (determined by palpation and near-normal weight bearing with the cast removed) may occur in 4-6 weeks. Adult cattle may require 3-4 times as long to fully heal.[1]

Thomas splint-cast combination
For cattle, TSCCs are constructed from steel rods, with the diameter of rod needed dependent on the animal's body weight. For stabilization of radial /ulnar fractures, bodyweight is related to steel rod diameter as follows: Less than 225 kg, 3/8 inch (0.95 cm); 225-450 kg, ½ inch (1.27 cm); and greater than 450 kg, 5/8 inch (1.59 cm).[5] For TSCCs used to stabilize fractures of the tibia, bodyweight is related to steel rod diameter as follows: Less than 180 kg, 3/8 inch (0.95 cm); 180-360 kg , ½ inch (1.27 cm); and 360-540 kg, 5/8 inch (1.59 cm). For cattle weighing over 540 kg, 1-inch (2.54 cm) diameter steel conduit pipe is needed for stabilization of tibial fractures.[5] The length of the apparatus should be pre-measured on the normal limb with the animal in a standing position. The distance from the base of the splint ring to the bottom of the splint is measured as the distance between the axilla or inguinal space to the bottom of the hoof. The ring should be well padded and of sufficient diameter to fit into the axilla or inguinal area without impinging on the shoulder or pelvis, respectively. The ventral third of the ring should be bent slightly inward (medially on the animal), such that this section of the ring rests securely within the axilla or inguinal area.

The animal should be sedated or anesthetized and placed in lateral recumbency with the affected limb on the up side. A stockinette, padding, and cast are applied to the affected limb, beginning at the level of the mid-metacarpus or metatarsus and extending proximally to the level of the proximal radius or tibia. The splint is then applied to the limb. The foot is attached to the bottom of the splint by wires passed through holes drilled in the hoof wall; acrylic cement further secures the foot to the splint. Casting material is then used to attach the cast firmly to the cranial bar of the splint frame. Casting material or tape can be used to cover the cranial and caudal bars of the splint in order to prevent the opposite limb from becoming entrapped between the splint bars.

Frequent observation of treated animals is necessary for several days after application, as the immobilized limb may become trapped beneath the recumbent animal, resulting in secondary free-gas bloat.[1,5,6] Cattle with Thomas splint and cast combinations may need to be assisted to stand several times per day in the first week after application until the animal learns how to rise on their own.[1,5,6] The splint-cast combination may need to be changed because loosening of the cast can occur as swelling subsides.

Summary
Effective fracture management in the field setting requires careful consideration of multiple patient, environmental, management, and economic factors. The veterinarian must determine if the cast or TSCC are valid treatment options, with due consideration of animal welfare, orthopedic, logistical, and economic factors. Prompt euthanasia is indicated for fractures judged to be untreatable.

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HOW I GET CALVES OUT

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Introduction

Successful delivery of calves requires patience and adherence to fundamental principles of obstetrics. Cleanliness, patience, application of obstetrical lubricant, appropriate placement of chains or straps, and an understanding of the physical interactions between the fetal body and the maternal pelvic inlet are key factors for successful delivery. Successful mutation and traction can be predicted by the successive appearance of particular anatomic features of the calf at the dam's vulva. That stated, failed attempts at vaginal delivery are inevitable, and much can be learned from the time and effort put forth in such endeavors. In this seminar, obstetrical fundamentals will be combined with a few novel approaches for delivery of calves taught to the author by the humbling hand of fate, by fellow practitioners, and by farmers and farm hands.

Dystocia

The author begins intervention with a brief physical examination, aimed primarily at detecting any signs of sepsis or hypocalcemia. If signs of sepsis are apparent, the viability of the fetus and integrity of the uterus are assessed. Hypocalcemia is addressed by slow intravenous infusion of calcium salts. The animal's tail is secured by a tail tie.

The author prefers to begin the obstetrical examination with a rectal examination of the dam. This is intended to evacuate feces from the rectum, assess the dorsal uterine wall for crepitus (which is often indicative of an emphysematous fetus), and ascertain the position of the broad ligaments. The position of the broad ligaments can be assessed with the examiner's hand at wrist-to-forearm depth relative to the dam's anus. The ligaments are expected to be symmetrical, soft, and located lateral to the caudal uterus and cervix. Ligaments that are taught and asymmetrical are indicative of uterine torsion. The broad ligament on the side opposite of the direction of torsion is pulled across the top of the uterus; the palpable sensation when this ligament is patted with the hand is that the ligament feels like a trampoline. The broad ligament on the same side as the direction of torsion is pulled taught in a dorsoventral direction.

The perineum is cleaned, and vaginal examination is conducted next. Full cervical dilation is expected. If cervical dilation is incomplete, the possibility of early intervention is considered; however, if the fetus is dead and the uterus relatively dry, the cervix is likely in contracture. Fetal presentation, position, posture, and signs of viability are assessed next. If forced extraction is deemed likely, the author prefers to consider administration of non-steroidal anti-inflammatory drugs (e.g. flunixin meglumine, 1.1-2.2 mg/kg IV) prior to application of traction. Mitigation of neural injury to the dam is the rationale for this treatment.

The author recommends placement of abundant amounts (3-4 l) of clean obstetrical lubricant into the uterus and around the fetus. Use of a marine bilge pump and stomach tube can facilitate this process. Lubrication is critical for minimizing friction as the fetus engages the maternal pelvic inlet. Carboxymethylcellulose is preferred over polyethylene powder as the latter is expected to induce severe peritonitis if leaked into the abdomen during cesarean section.[1]

Dorso-ileal or dorso-pubic malposition should be reassessed as potential uterine torsions of less than 180 degrees. Rotation into normal position can usually be achieved manually or with the use of a detorsion rod. Failing that, the cow can be cast into recumbency and placed in the appropriate lateral recumbency needed for gravity to facilitate proper fetal positioning.

Retained fetal limbs or retention of the fetal head typically warrant administration of sacrococcygeal epidural anesthesia if the dam is straining excessively. These expulsive efforts often impair the necessary repulsion of the fetal body and securing of the retained structure.

Repulsion is greatly facilitated by copious lubrication. Retained limbs are manipulated such that the fetal hoof is brought ventral to the fetal body before the limb is extended into the correct posture. This translates to initial mutation of the upper part of the retained limb dorsally and laterally. Retention of the fetal head or limb can be a challenging malposture for the short-armed obstetrician. In such cases, it can be helpful to cast the cow into lateral recumbency such that the retained fetal part is on the upside relative to the rest of the fetus. For example, if the head were retained toward the cow's left side, the cow would be cast into right lateral recumbency (right side down), such that the retained fetal head is upwards relative to its body. Gravity may move the fetal mass downward, giving precious more room for the examiner to secure the limb or head for mutation into proper posture.

Momont (2005) estimated that 95% of bovine fetal presentations are anterior, with the dorso-sacral position expected in normal deliveries.[2] The Utrecht guidelines for forced extraction form the ground rules for assisted delivery by traction. When possible, the author prefers to attempt most mutations with the cow standing and to provide traction (pulling) with the cow in sternal recumbency. If mutations have been performed, the author pauses to reapply copious lubricant into the uterus prior to pulling.

Once the fetus is determined to be in proper posture, chains or straps can be placed on the fetal forelimbs. If one fetal limb is positioned more cranially in the birth canal than the other, it is often helpful to place traction on the cranial-most limb first. This prevents the weight of the calf from pinning that limb's shoulder or elbow against the dam's pelvic inlet.

The author prefers to apply traction to the fetal forelimbs in an alternating fashion. If the fetus and maternal pelvis were to be viewed from above (from dorsal to ventral), alternating traction of the fetal forelimbs can be considered more likely to reduce the cross-sectional diameter of the fetal shoulders. Passage of the fetal shoulders through the maternal pelvic brim is evidenced by the fetlocks extracted ~ 10 cm from the vulvar opening; this rule can be condensed to state that appearance of the fetal carpi at the vulva typically signal that this has occurred. Once the head and shoulders appear at the vulva, traction is stopped, lubricant is reapplied, and the calf's head and shoulders are rotated 90-180 degrees to facilitate passage of the fetal hips into the maternal pelvis, which is widest in the diagonal dimension.

For posterior presentations, lubrication is paramount as the lay of the fetal haircoat is opposite of the direction of extraction. The calf is repelled and rotated such that the calf's greater trochanters are aligned along the diagonal dimension of the cow's pelvic inlet. The fetal tail should be located and pulled ventrally so it lies between the fetal hind limbs. Although it is not generally accepted that alternate pulling on the hind limbs is any better than application of simultaneous traction of both hind limbs, the author still prefers alternate pulling. Passage of the fetal greater trochanters into the maternal pelvic canal is evidenced by appearance of the hocks at the vulva.

Postpartum care includes uterine lavage with warm, clean water, administration of oxytocin, administration of analgesics, and milking out of the dam so that the calf can be fed colostrum via orogastric tube. The last step helps to ensure adequate passive transfer, which may be jeopardized if the exhausted dam remains recumbent postpartum. Broad-spectrum antimicrobial therapy is indicated for severe dystocia (2 people pulling for > 15 minutes) or those wherein obstetrical hygiene is compromised by poor environmental conditions. Cows that strain excessively postpartum or show other signs of prolonged discomfort (e.g. tail extension) often benefit from epidural administration of morphine (0.1 mg/kg) or xylazine (0.05 mg/kg) at the sacrococcygeal space.

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HOW I SELECT AN ANTIMICROBIAL DRUG

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Introduction

Selection of antimicrobials for food animals requires consideration of more factors than for most companion animals. Legal restrictions on use and avoidance of residues in meat and milk are foremost. The oral route of administration is often not an option, owing to chemical degradation of the antimicrobial in the fermentative forestomach, as well as potentially detrimental alteration of forestomach microflora by the drug in question. Patient size and temperament often necessitate the use of restraint devices, which carries a concurrent labor costs, as well as risks of stress and injury to the animal. In many cases, economic and time constraints often limit the application of conventional ancillary diagnostic tests, such as culture and sensitivity, unless problems with poor treatment response are documented beforehand. In this seminar, the primary pharmacologic characteristics of some of the commonly used antimicrobials will be discussed, and case examples will be used to open up discussion on which antimicrobials might be applied to each case. Obviously, such decisions are shaped by previous experience, economic factors, and other variables that cannot be tallied into each example. Where appropriate, additional discussion points about the disease in question will be raised. While many of the antimicrobials discussed are not legal or advised for use in food animals, they are included in the presentation to increase awareness of their limitations. Further, non-food producing animals (e.g. camelids) often comprise some component of the rural or food animal practitioner, and examples involving these species will be included.

When helping veterinary students with decisions on antimicrobial selection in clinical settings, the author has encouraged students to apply certain criteria to their decision. These criteria are collected into a decision-review process called "The Six Ss of Antimicrobial Therapy:" Site, solubility, spectrum, status of the immune system, sensitivity (or susceptibility), and side effects. A seventh "s" could be added: Statutes (legal restrictions). Each of these criteria is discussed in greater detail below. This decision-review process is similar to that recommended by Langston.[1]

Site

Consideration of tissue conditions at the site of infection is essential for selection of the appropriate antimicrobial drug. Although ancillary diagnostic tests such as radiograph and ultrasound are often helpful in determining the status of infected structures, when these are not available, the veterinarian must use the history and examination findings to directly view or envision the conditions at the site of infection. Consideration of the microenvironmental conditions at the infection site is critical. What is the status of blood supply at the site, and is local hypoxia likely? Trauma and accumulation of purulent or necrotic debris may reduce the blood supply to infected structures. A lack of oxygen, a low tissue pH, and accumulation of necrotic or purulent debris all render the aminoglycosides ineffective at the site of infection.[1]

Aminoglycoside penetration into bacteria requires the presence of oxygen and non-acidic conditions, and these antimicrobials are readily bound to nucleic acid; hence, their activity in purulent or necrotic tissue is poor. Such conditions also reduce the efficacy of sulfonamides and beta lactam antimicrobials, albeit by different mechanisms.[1] When voluminous, fibrin and purulent debris may act as a protective barrier to embedded organisms that have survived the host's defenses, and when safely applied, drainage or debridement of such sites is always a preferred action to antimicrobial treatment alone. Infection sites that have extensive tissue necrosis have limited oxygen concentration, facilitating colonization and replication by anaerobes and/or facultative anaerobes (such as many coliforms). In mixed

infections, release of beta lactamase by certain organisms (e.g. *Staphylococcus*) may impart protection to more susceptible species of bacteria.

Is the infection likely in the acute, subacute, or chronic stage? In subacute to chronic infections, the presence of fibrin, debris, or fibrous tissue may impair the ability of certain water-soluble antimicrobials to diffuse into the colonized tissue. Are the suspect bacteria in the extracellular or intracellular space? Certain bacteria remain in the extracellular fluid (e.g. *Pasteurella, Hemophilus Histophilus,,Leptospira*), others are obligate intracellular pathogens (e.g. *Anaplasma, Mycoplasma, Chlamydophila*) while others are facultative intracellular organisms (e.g. *Listeria, Salmonella*). When the organism is located in the host's cells for most or all of its lifespan, it is usually necessary to select an antimicrobial with sufficient lipid solubility to penetrate inside the host's cell membranes. The presence of foreign bodies, suture, metallic implants, and bone sequestra often enable infection to persist in spite of appropriate antimicrobial treatment.

Spectrum

The spectrum of an antimicrobial drug refers to the range of microorganisms considered likely to be susceptible to conventional therapeutic regimens of that drug. The spectrum of an antimicrobial drug is relatively easily acquired through multiple sources, ranging from label information to peer-reviewed literature to textbooks.[1-6] The veterinarian must understand that a given drug's spectrum of activity for one clinical application (e.g. pneumonia) may not correlate well with that activity in a different application (e.g. enterocolitis).[3] Further, lay personnel frequently have a very limited perception of antimicrobial spectrum (as well as other relevant facets of antimicrobial use), and veterinarians are uniquely well-positioned to provide this critical audience with valuable instruction and guidance.

The development of resistance in a strain, species, or genus of bacteria can break the relationship between published data on the spectrum of a drug and the actual clinical effect. Drug spectrum for novel antimicrobials is often well-publicized at the onset of market release, and changes in susceptibility of target organisms should be carefully monitored in order to maintain a relevant and valid understanding of a drug's spectrum. Susceptibility is discussed below.

Status of the immune system (or -static versus -cidal drug)

The classification of a particular antimicrobial agent as bacteriostatic versus bactericidal is not definite, as the effect of a drug on a given organism is affected by multiple factors, including susceptibility of a given strain of organism and the treatment regimen utilized. Strictly speaking, this criterion is a measurement of the relationship between the minimum inhibitory concentration (MIC) and the minimum bactericidal concentration (MBC) for a given drug – microorganism combination; obviously, the variation in this measurement can be substantial across different patients, different bacteria, and different regimens. In conventional terms, bacteriostatic antimicrobials inhibit growth and particular facets of metabolism of a given microbe, and growth and those metabolic functions resume upon removal of the drug. Immune mechanisms of the host cause the ultimate demise and clearance of the microorganism. Bactericidal antimicrobials more reliably induce the death of the bacteria. Bactericidal drugs are generally warranted for animals with acute, life-threatening infections (e.g. colisepticemia) and those with a substantially crippled immune system (e.g. failure of passive transfer, immunosuppressive drugs or viral infections). Most bacteriostatic drugs used in food animal practice require a regimen that enables the drug to remain above the MIC for the majority (estimated at 60-80%) of the dosing interval.[6]

Solubility

Water-soluble antimicrobials dissolve well in the plasma and ECF, although limited binding to blood and tissue proteins may occur (e.g. ceftiofur, certain sulfonamides).[1] As a rule, most of these drugs cross the glomerulus and concentrate well in the urinary tract. Water-soluble antimicrobials also distribute well into the extracellular fluid, achieving high concentrations in plasma, joint fluid, pleural and peritoneal fluid, and bone. However, these drugs do not cross lipid bilayers easily.

Because of this property, water-soluble antibiotics are usually not the drugs of choice for treatment of intracellular pathogens, particularly those that spend the majority of the life cycle inside the cells, with little exposure to the extracellular fluid. For the water-soluble antibiotics, plasma drug concentrations are a good indicator of the levels of drug available to fight the bacteria. The question then becomes, can the water-soluble antibiotic diffuse into the infected tissue in adequate concentrations to fight the infection? In the subacute to chronic stage of infection, cellular infiltrates, fibrin, and necrotic debris become “diffusion barriers" (e.g., consolidated lung tissue in pneumonia) which can limit the extent that a water-soluble drug can penetrate into the infected area.[1]

Lipid-soluble antimicrobials can achieve high concentrations in tissue, owing to their ability to cross lipid bilayers. These drugs are protein-bound when circulating in plasma and as a result, most do not extensively cross the healthy glomerulus and do not achieve high urine concentrations. These drugs tend to distribute out of the plasma and into tissue quite readily, so don't be misled by data that shows these compounds to have a lower plasma concentration than, say, a particular water-soluble antimicrobial.

In order to obtain the highest probability of a treatment response to antimicrobial therapy, the solubility of the drug must match the site of infection. For example, most sulfonamides, being water-soluble and often protein-bound, would not be an optimal choice for treatment of an infection suspected to be caused by an intracellular pathogen such as Mycoplasma. Exceptions to this rule, however, are plentiful. For example, penicillin has high efficacy for treatment of listeriosis, provided that treatment is initiated early, yet the infection is located in the brain and the organism is a facultative intracellular pathogen.[1] This exemplifies the role of susceptibility in the clinical response.[1] Other characteristics of an antimicrobial will influence its localization to a certain site. For example, through a process termed ion(ic) trapping, weakly basic antimicrobials, such as macrolides, become charged in an environment below their pka (e.g. the low pH of most infected tissue) by gaining a hydrogen ion; the resultant change in the molecule’s charge may impair its diffusion across membranes, resulting in accumulation of the drug at the site.[7]

Sensitivity

Sensitivity (susceptibility) is a measure of the likely effect of an antimicrobial in combating infection caused by a specific pathogen in a specific host. For a successful clinical response to treatment, the sensitivity of the microorganism(s) must match the spectrum of the drug. Of the six criteria listed in the decision review process, sensitivity is almost always the most difficult to define. Antimicrobial sensitivity can vary extensively among bacterial genera, among species of a genus, and within species (strains or subtypes). Sensitivity is most accurately defined by laboratory testing; however, the application of culture and sensitivity in clinical practice is often limited by expense, logistics (including the time delay for results), and initiation of antimicrobial therapy in advance of consultation with a veterinarian. The correlation of antimicrobial sensitivity of an isolate to the patient’s response to treatment is not absolute. Further, susceptibility and resistance criteria established for a given drug – bacteria combination – in, say, the pneumonic lung - may not apply to infections with the same organism another tissue site.[3] Interpretation of sensitivity (susceptibility) testing is influenced greatly by the testing methodology used. Finally, susceptibility data may have little relation to clinical response if treatment is initiated too late in the clinical course. Proper training and continuing education of animal caretakers is critical to optimizing treatment success.

When initiating treatment, prediction of antimicrobial sensitivity in the patient with a suspected bacterial infection requires, to some extent, a prediction of the organism(s) likely contributing to disease; this prediction is often based on literature and education, rather than isolation of bacteria from similarly diseased patients through ante- or post-mortem susceptibility tests. Although inherently flawed, response to treatment is the surrogate measurement of sensitivity often employed in clinical practice, even when culture and sensitivity testing is initiated – there is a time lag between submission and results that makes

initiation of treatment on relatively empirical grounds of logic necessary. Empirical decisions are appropriate for life-threatening infections, infections with consistent pathogen and sensitivity profiles, and when financial constraints prevent susceptibility testing.

Whenever possible, specimens from infection areas (aspirates, smears, etc) should be examined by conventional stains (e.g. Diff-Quik®) and Grams' stain to better define the characteristics of the agent involved. Further, on-farm initiation of protocols for antimicrobial treatment and recording of treatment responses are integral to detection of a change in pathogen, susceptibility, or both. How can you monitor what you don't record? A strong effort should be made to convince producers of the role of regular sensitivity profiling in population medicine programs; a budget for such testing can be developed and interpretation / consulting can integrated into the regular fees for service. Finally, some diagnostic laboratories publish annual summaries of susceptibility tests for certain bacteria, and web-based decision support systems, web-based databanks, and published reviews will greatly aid veterinarians in making valid decisions about this often elusive criterion.

Side effects and statutes

In food animals, violative residues in meat and milk represent one of the most widespread and socially-relevant side effect of antimicrobial therapy, although some may argue that induction of antimicrobial resistance is of greater importance. Regardless of their perceived order of importance, induction of antimicrobial resistance and residues in meat and milk should always be considered as potential consequences to antimicrobial therapy in food animals.[8] The issues of veterinary antimicrobial use and antimicrobial resistance, as well as expert positions on prevention of resistance, have been reviewed.[8] Injection site reactions also carry tremendous significance in the livestock industries, and these reactions should be considered a side effect of treatment rather than a nuisance for slaughter facilities. In the author's experience, however, veterinary students in clinical settings rarely factor this side effect into their decisions. Educators, including practitioners, need to work to remedy this problem.

Idiosyncratic reactions aside, the pathologic side effects of most antimicrobials are well established in the veterinary profession but often poorly understood by lay personnel. Antimicrobial treatment protocols for livestock operations are an often overlooked means of limiting side effects, as these enable the veterinarian to guide application of particular drugs (particularly OTC medications) on a farm. For example, while the nephrotoxicity of aminoglycosides is well-established, the potential nephrotoxicity of oxytetracycline, particularly in dehydrated, endotoxemic animals administered large doses, is worthy of consideration in decision making.[1] Many veterinarians understand that calcium ion may chelate tetracyclines, as this is a postulated mechanism for induction of collapse in animals following rapid IV administration of oxytetracycline.[1] The fact that topical tetracycline preparations reconstituted in hard water may lose efficacy, as may oxytetracycline diluted in intravenous fluids containing calcium (e.g. Lactated Ringer's solution), are potentially lesser known, but related issues. Most pathologic side effects for a given drug can be easily located in the package- or label insert.

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HOW I TREAT ABOMASITIS AND ABOMASAL BLOAT IN CALVES

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Introduction

Abomasitis is a sporadic disorder of young ruminants that can occasionally occur in outbreaks (typically, clusters of cases over short periods of time on a given farm). The disorder is suspected to have a multifactorial cause, with nutritional and management factors appearing to play a preeminent role. The case fatality risk appears to be highly variable, and the pathophysiology of this disease is not completely understood. Treatment and prevention strategies that are consistently successful have remained elusive, in the author's experience. In this seminar, a brief literature review will be provided to shed light on the etiology, treatment, and prevention of abomasitis and abomasal bloat.

Clinical signs and necropsy findings

Abomasitis is defined as diffuse inflammation of the abomasal wall, which can be accompanied by concurrent abomasal gas accumulation (bloat) and variable ulceration, edema, necrosis, and emphysema of the abomasal wall.[1] This disorder typically strikes beef and dairy calves, lambs, and kids under 1 month of age, although occasional cases in older animals occur. This disease is characterized by the rapid onset of abdominal distension, depressed attitude, and variable signs of colic. Affected animals may be seen to grind their teeth and salivate. Diarrhea may or may not accompany these signs. Once abdominal distension is severe, if the flank of the affected animal is shaken by hand, a tinkling and splashing sound may be heard. Distension occurs bilaterally; differentiation from abomasal volvulus by physical examination alone is difficult. Death may occur within hours to 2 days after the onset of signs. According to a limited number of reports and reviews, the case fatality rate for this disease can be high (60-100%).[2-4]

At necropsy, a gas-filled and inflamed abomasum is commonly seen. Lesion distribution is typically diffuse, but focal or segmental disease may also be evident. The abomasum often contains foul-smelling, sour clots of milk. Bile reflux from the small intestine may impart a greenish color to the abomasal contents. Hemorrhage from the abomasal lining, however, may cause the contents to become rust-colored or even black. Ulcers may be visible in the abomasal wall, and occasionally, these perforate to release abomasal contents into the abdominal cavity, resulting in peritonitis. Near death, calves show signs of shock, compromised respiration from the pressure of the distended stomach, and dehydration. Histologic examination of the abomasum may reveal edema, emphysema, vascular congestion, and necrosis.[4]

Etiology

There are many proposed etiologies for this disease. Primary bacterial or fungal infection, immunosuppression, pica, trauma from coarse feed or trichobezoars, and vitamin / mineral deficiencies (specifically, copper and Vitamin E) have all been implied as causes or contributory factors. In 1987, investigators at Kansas State University detected *C. perfringens* types A and E in stomach contents of affected calves.[3] Subsequently, these investigators reproduced the disease in calves by intraruminal inoculation of *C. perfringens* type A.[5] Belgian investigators have also detected *C. perfringens* in the abomasum of affected calves;[6] in a survey of US veterinary practitioners, this organism was also reported to be a common isolate of affected calves.[2] The ability of this organism to produce exotoxins and gas may contribute to the mucosal lesions, gastric dilation, and intramural emphysema that are commonly seen in affected animals.

Clostridium perfringens is a large, Gram-positive, anaerobic bacillus that exists ubiquitously in the environment and in the gastrointestinal tract of most mammals. This organism can be found in the neonatal gastrointestinal tract within 1-2 days after birth, with the source of the inoculum thought to be

the maternal urogenital tract or skin. Diets high in concentrates have been shown to increase the rate of isolation of *C. perfringens* from the rumen and cecum of healthy ruminants. Proliferation of *C. perfringens* in the ruminant gastrointestinal tract, resulting from concentrate feeding or overeating, is considered the pivotal event in the onset of intestinal disease (enterotoxemia); a similar pathogenesis is speculated to cause forestomach disease.[1-6]

Interpretation of positive culture results for *C. perfringens* from the intestinal lumen of a ruminant is a complicated matter. *Clostridium perfringens* type A inhabits the intestine of normal animals and can overgrow in the gut lumen *post mortem*.[1] Thus, its isolation should be considered significant only from a fresh cadaver with compatible history, clinical signs, and lesions.

Salmonella typhimurium DT104 has been isolated from the abomasal wall of veal calves with abomasitis in the Midwestern USA.[7,8] *Sarcinia* are a diverse species of bacteria found in the gastrointestinal tract. While these organisms have been identified in the abomasum in some cases of abomasitis and abomasal tympany,[2,4] the association between this organism and abomasal disease remains poorly defined. Further, bacterial cultures of abomasal contents or ulcers may not consistently yield pathogenic enteric bacteria.[2]

In earlier case reports, authors associated copper deficiency with abomasitis and abomasal ulcers in beef calves.[9] However, Roeder and colleagues demonstrated that abomasitis could occur spontaneously and be induced experimentally in the absence of copper deficiency.[5] Thus, although copper deficiency could act as a contributory factor for abomasitis of calves, its role as a necessary cause remains uncertain. Cases of this disease in neonatal beef calves have been associated temporally with management practices that cause delays in regular nursing patterns (e.g. calf separation at branding) or changes in environment that interrupt normal nursing patterns (e.g. winter storms). In dairy calves, poor milk or colostrum hygiene, intermittent feeding of large volumes of milk, high-osmolality milk replacers, irregular feeding schedules, and altered milk or milk replacer temperature have been empirically incriminated as potential contributory factors for abomasal tympany, ulceration, and abomasitis.[1-4]

Treatment

If detected early, abomasitis and abomasal bloat may respond to oral or systemic antimicrobials. Assuming that milk nutrients provide a growth substrate for luminal bacterial growth and gas production, the author prefers to remove milk from the diet of affected calves for 24 hours and to provide oral electrolytes and/or parenteral fluids to maintain hydration. Mineral oil administered via orogastric tube may aid in suppressing luminal bacterial overgrowth and facilitate egress of substrate (milk or solid feed) from the forestomach. Re-introduction of milk to the diet of affected calves is then resumed, albeit with frequent (q 3-4 h) feedings of small volumes of warm cow's milk or high-quality milk replacer.

Severe cases are defined here as those with marked abdominal distension, dehydration, and weakness. For severe cases, intravenous fluid therapy, parenteral antibiotic therapy, and oral antibiotic therapy are warranted in the initial medical management. Parenteral treatment with high doses of penicillin is commonly used. It is the author's impression that, despite its documented efficacy against *Clostridium* spp., penicillin must be administered relatively early in the course of disease to positively affect outcome; if necrosis and ulceration are advanced at the time of treatment, a positive response does not occur. The development of necrosis within the abomasal wall is speculated to result in impaired delivery of penicillin to all areas of the infection, as an intact microvasculature is needed for a water-soluble drug such as penicillin to be delivered to the affected tissue.

Orogastric tube passage and decompression can be accomplished in rare cases. Elevation of the calf's forequarters and massage and gentle compression of the caudal abdomen while the tube is placed may be helpful in releasing gas from the abomasum. However, in most cases, minimal gas is released by orogastric intubation, which is suggestive that the gas is effectively sequestered in the abomasum. Oral antibiotics such as penicillin or tetracycline may be helpful in reducing the rate of intraluminal gas production. If forestomach atony is present, however, orally-delivered antimicrobials may not reach the abomasum in time to influence bacterial gas and/or toxin production.

Decompression of the abomasum via percutaneous ventral abomasocentesis, with the calf restrained in dorsal recumbency, has been described,[10] and intraluminal injection of antibiotics could be performed after decompression. With the calf held in dorsal recumbency, a 14-gauge needle is inserted in the highest point of the abdominal wall between the sternum and the umbilicus. Decompression of a distended abomasum with the calf in the standing position has been associated with the development of peritonitis and is not recommended.[10] The efficacy of passive immunity imparted from *Clostridium perfringens* type C and D antitoxin[2] is unclear, given that this medication is typically administered in conjunction with laxatives, antacids, and antimicrobial drugs.

Differential diagnoses include abomasal or cecal volvulus, left displacement of the abomasum (LDA), and right displacement of the abomasum. In light of the apparent high rate of ulceration of the abomasum associated with LDA in calves, surgical exploration is warranted in cases that do not respond to medical management. Abomasotomy may be indicated for refractory cases of abomasal tympany. Surgical intervention results in removal of luminal foreign bodies such as trichophytobezoars and removal of putrefying milk, both of which may prevent a satisfactory response to medical management.[13]

Prevention

Because the cause(s) of abomasal bloat and abomasitis are not well proven, preventive measures are currently based on the assumption that the pathogenesis centers upon stasis of large volumes of readily available carbohydrate and protein in the abomasum. On dairies, careful critique of milk or milk replacer preparation and delivery to calves must be done. Inconsistencies in temperature, volume, timing of feeding, ingredients fed, and cleanliness of buckets and bottles should be identified. Contamination of milk or milk replacer while in storage or during delivery to the calf may also contribute. Sampling milk at delivery to the calf (i.e. from the bucket or bottle) may be used to determine if contamination of milk or milk replacer is occurring, particularly with such agents as *Salmonella* spp. Provision of clean water is essential, and feeding high-quality hay or calf starter in small amounts, kept fresh, is important to maintain consistent intake of dry feeds.

Because the fermentation of milk by bacteria seems to be the source of gas, one must consider environmental or management factors that may trigger changes in the volume of milk ingested by a hungry calf. Management practices that cause prolonged interruption of suckling must be made time-efficient in order to limit engorgement of the udder and subsequent ingestion by the neonate of a larger-than-normal milk meal. Sudden and severe changes in weather may cause dams and their offspring to seek shelter or remain recumbent for prolonged periods of time. In such instances, when the weather clears or daybreak occurs, the dams then stand up – and the hungry calf is then presented with an engorged udder. This can be remedied by providing multiple locales for shelter and bedding, or simply encouraging dams to stand up and eat by providing hay (weather permitting). This practice may encourage the dams to stand, thereby enabling more frequent, lower-volume nursing than if the cattle were left to “sit the storm out.” Group penning, inadequate fiber diets, and ectoparasites may trigger hair chewing by calves, and hairballs that form in the abomasum may irritate the lining and trigger the disease.

Currently, in spite of abundant anecdotal reports, there is no clear data on whether or not conventional vaccines that include the inactivated toxins from *C. perfringens* types C and D induce antibodies in the colostrum that protect the calf from this disease. Administration of an inactivated *C. perfringens* type A bacterin-toxoid to dry cows and pregnant heifers resulted in significantly higher serum neutralizing antibody titers to alpha toxin in calves fed colostrum from immunized dams than in calves fed colostrum from control animals.[14] However, the efficacy of vaccination in the prevention of abomasal bloat / abomasitis remains unproven. The lack of published evidence for a single intervention to result in resolution of this disorder may reflect a multifactorial causation (Songer JG, personal communication, 2012).

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HOW I TREAT DEEP SEPSIS OF THE DIGIT

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Introduction

Lameness remains a major cause of disease and economic loss in dairy, cow-calf, and feedlot operations. The majority of bovine lameness involves the structures of the digit. Sole ulcer (pododermatitis circumscripta), white line disease, and interdigital necrobacillosis (footrot) are common digital disorders. If not treated promptly, these conditions can progress to create infection of bone, synovial structures, tendons, and ligaments of the digit. These deeper structures may also become infected from solar puncture wounds, lacerations, avulsion injuries of the hoof, and proximal progression of subsolar abscesses. Infection of these structures has been termed generalized digital sepsis, or, for the purposes of this discussion, deep sepsis of the digit.

Deep sepsis of the digit is commonly treated by digital amputation. Amputation of the digit may not be the best treatment option if the animal is heavy, maintained on range, pasture, or a large dry-lot dairy, intended for natural breeding, or intended for long-term (>18-24 mo.) productivity. Medical and surgical treatments aimed at salvage of the affected digit may be more appropriate in such cases, although these cattle represent a therapeutic challenge to the veterinarian. Obviously, it is preferable to intervene in foot diseases prior to the establishment of deep sepsis of the digit, because therapeutic options are limited for this condition. The goals of this seminar are: 1) to review the criteria that owners can use when making diagnostic and treatment decisions regarding lame cattle, so as to minimize the number of deep sepsis cases; and 2) to describe therapeutic options if deep sepsis does occur.

Protocols for on-farm diagnosis of lameness

One of the most critical evaluations that an owner can make regarding a lame animal is to address the question: Is the affected foot swollen? Cases of lameness that do not usually result in swelling of the digit include papillomatous digital dermatitis (hairy warts), interdigital dermatitis, sole ulcers (pododermatitis circumscripta), laminitis, subsolar abscesses, or injuries or disease of the proximal limb.

Swelling of the digit proximal to the coronary band is commonly seen in cases of footrot (interdigital necrobacillosis). Deep sepsis of the digit generally results in visible digital swelling as well. Digital swelling can be readily visualized from behind the animal by comparing the distance between the dewclaws of the affected foot to that of unaffected feet. Owners should be encouraged to thoroughly clean the feet of lame cattle in order to more reliably detect swelling. Substantial (> grade 3/5), persistent lameness is typical in cattle suffering from footrot and deep sepsis of the digit.

Many livestock owners choose to make the initial diagnosis of digital disorders, with varying degrees of accuracy, and to initiate treatment on premises. In a retrospective study of facilitated ankylosis of the bovine distal interphalangeal joint (DIPJ), Desrochers and colleagues found that all 12 cases in their study had received antimicrobial treatment prior to presentation.[1] The mean duration of lameness prior to surgery for cattle in that study was 5.3 weeks (range, 1-24 weeks).[1] Pejsa and colleagues found that over 70% of cattle undergoing digit amputation had been treated prior to presentation, with 47% of these animals having a lameness duration of greater than 3 weeks.[2] These practices may reflect, in part, greater access to livestock antimicrobials sold over the counter (directly to laypeople) in the USA versus in other countries.

Because footrot begins in the interdigital skin, the swelling of soft tissues proximal to the coronary band is usually symmetrical relative to the longitudinal axis of the distal limb. In contrast to footrot, deep sepsis of the digit usually results in asymmetrical swelling of the distal limb and foot, with the majority of swelling located on the side of the affected digit. If

livestock owners can make this distinction when the affected animal is first examined, cases of deep sepsis of the digit can be identified early and veterinary intervention sought promptly.

Protocols for on-farm treatment of lameness

In our practice area, owner-initiated treatment of presumed footrot in beef cattle usually consists of parenteral long-acting oxytetracycline (22 mg/kg SC q. 48 h). Dairy personnel usually administer ceftiofur (1.1-2.2 mg/kg IM q 24 h) for initial therapy. While these regimens prove efficacious for many cases of footrot, livestock owners should understand the expected course of a positive treatment response. Assuming that drug administration technique and dosing are accurate, cattle with footrot that are treated with an appropriate antimicrobial regimen typically show resolution of lameness within a 7 to 10-day treatment period. Failing this, affected animals should be brought to the attention of the veterinarian, as many of these treatment failures represent misdiagnoses or are footrot cases which have progressed to deep sepsis of the digit. A footrot treatment protocol allows the herd veterinarian to: 1) intervene early in cases of deep sepsis of the digit, 2) appraise treatment response rates, and 3) monitor the antimicrobial dosing and administration procedures on the ranch or dairy. In the absence of such a protocol, owners may make the error of rotating from one antimicrobial to another, mistaking deep sepsis of the digit for a difficult case of footrot. As Greenough wrote in 1962, "This practice of hit-or-miss therapy has its successes, but all too frequently [such cases] end up with a practitioner being presented with an advanced case of generalized foot sepsis."[3]

Infection of the DIPJ is a common sequel to footrot. The interdigital skin lies very close to the axial and dorsal aspect of the DIPJ, and extension of infection from the interdigital skin into the synovial space of this joint occurs commonly. Septic arthritis of the DIPJ is characterized by asymmetric swelling of the foot with a visible bulge present in the soft tissues immediately proximal to the coronary band. A synovial fistula (sinus) may develop at or proximal to the coronary band.

Digital wounds involving puncture of the sole or hoof wall may inoculate deep structures of the digit. Such wounds usually generate discernable swelling of the soft tissues proximal to the coronary band. Lacerations of the heel and pastern also usually create sufficient cellulitis to cause visible swelling of the foot. Again, an accurate diagnosis by ranch or dairy personnel requires that the foot be washed and examined closely. Owners should also understand that injuries to the distal limb that create visible lameness and/or swelling of the soft tissues are rarely trivial in severity. Owners may merely apply topical disinfectants onto such wounds, a form of treatment that is almost uniformly unsuccessful. A protocol for digital wounds could be stated as follows: If a digital wound is severe enough to cause the animal to be lame, contact the veterinarian.

Treatment options for deep sepsis of the digit

Once the bones, synovial structures, or ligaments / tendons of the foot become infected, antimicrobial therapy alone usually does not elicit a cure. It is possible that necrosis of infected tissue causes loss of adequate blood supply, and effective concentrations of an antimicrobial cannot reach all areas of bacterial colonization. Thus, debridement, drainage, and lavage of infected deep structures is usually required for resolution. However, antimicrobial therapy is a necessary adjunct to surgery in order to prevent further extension of infection in viable tissues.

Amputation. Amputation of the affected digit is a common method of treatment for deep sepsis of the digit in cattle. The advantages of this procedure include its low cost, rapidity and ease of accomplishment, and a relatively rapid rate of recovery in the patient. These advantages make this procedure the preferred method of treatment in the majority of cases of deep sepsis of the digit. The main disadvantage of digital amputation is the limited productive lifespan of the patient following the procedure. While extraordinary exceptions occur, many dairy cattle remain productive for 1-2 years beyond the amputation before the remaining digit suffers breakdown injuries from excessive weight bearing. This is often considered a satisfactory outcome for dairy cattle. Bulls, beef cows, and valuable breeding animals, however, may be more optimally treated by a procedure aimed at

salvaging the affected digit. In pasture- or range-based operations, the need for animals to travel long distances for feed, shelter, and water may preclude the use of digital amputation for cows intended to remain in the herd for years to come. Similar demands are placed on beef bulls, who also require digital salvage because their excessive weight would cause rapid breakdown of the remaining digit, particularly during mounting and dismounting of cows in estrus.

Salvage of the digit with septic arthritis. In many cases, a relatively accurate impression of the location of deep sepsis can be obtained by thorough examination and palpation of the digit. Placement of a sterile probe into the external aspect of the entry point of infection (sole ulcer, puncture wound, etc.) is frequently helpful in defining the depth and extent of deep sepsis. If acute septic arthritis of the coffin or pastern joint is present, large volume lavage of the joint with lactated Ringer's solution or 0.9% sodium chloride is indicated. Aggressive parenteral antimicrobial therapy is also necessary. High concentrations of antimicrobials in the synovial fluid can be obtained through intraarticular administration or regional perfusion. However, presentation of such cases in the acute stage is quite rare. In the more common subacute to chronic stages, fibrin deposition, necrosis of the synovial lining and articular cartilage, and extension of infection into the subchondral bone make are usually present, and management via joint lavage and antimicrobial therapy is not a viable option. If the digit is to be salvaged in such cases, facilitated arthrodesis of the joint is indicated.

Facilitated arthrodesis of the DIPJ can be performed with a bone curette, hand-held trephine, variable speed burr, or drill. The author prefers to use a variable-speed carpentry drill with chemically-disinfected drill bits. Facilitated ankylosis of the DIPJ of an average-sized (~ 600 kg) beef - breed cow is performed as follows: A stab incision is made on the dorsal aspect of the digital skin, immediately abaxial to the digital extensor tendon, and approximately 1 cm proximal to the coronet. A 1/4 to 3/8" drill bit is used to debride the distal interphalangeal joint via this dorsal approach. The drill bit is directed toward the sole of the digit at the junction of the soft heel horn with the hard solar horn; the drill is activated and the bit passed from the dorsal stab incision to exit at this point in the sole. This drill pass allows for debridement of the dorsal and proximal aspect of the joint beneath the extensor process of the third phalanx.

A second drill pass is directed from the abaxial to axial aspect of the DIPJ. A 1/4 to 3/8" drill bit is placed at the midpoint between the dorsal hoof wall and heel, approximately 1 cm distal to the coronet. The drill bit is directed toward the interdigital space and is passed through the hoof wall and into the DIPJ. The drill bit is kept oriented parallel to the solar surface and passed completely through the DIPJ. The bit then exits the axial aspect of the hoof wall into the interdigital space. Care must be taken to avoid trauma to the coronary band and the hoof wall of the opposite digit.

A larger (1/2-5/8") drill bit is introduced into each arthrostomy site, and the bit is used to completely debride the necrotic cartilage and subchondral bone. While healthy bone resists passage of the powered drill, necrotic bone is easily passed through with the drill. The arthrostomy sites are repeatedly lavaged to remove tissue and bone fragments. A curette can be introduced into the arthrostomy sites and the integrity of the remaining bone can be assessed. Once debridement is complete, the arthrostomy sites are lavaged a final time. Penrose drains (or nylon ropes) are then looped through the arthrostomy sites and tied to each other to secure them in place. A wooden or plastic block is secured to the sole of the sound digit. The toe of the affected digit is wired to the block or sound claw such that the ankylosis occurs with the solar surface of the affected digit remaining parallel to that of the sound digit. The foot is then bandaged with a strong pad of bandage tape placed in the interdigital space to maintain normal dimension of the interdigital space. This pad prevents axial deviation of the affected digit.

Antimicrobial and anti-inflammatory therapy is administered for two to three weeks. The animal should be housed alone in a stall or small corral. Repeated daily lavage and application of a clean bandages is considered

optimal treatment; however, in many cases, economic considerations dictate that follow-up care occur on a less frequent basis. Weekly recheck examinations are recommended, with removal of the Penrose drains in 1-2 weeks. Reduction in lameness is usually not appreciated until 10-14 days after surgery. In the author's experience, reduction in the extent of soft tissue swelling one week after surgery appears to be indicative of successful debridement of all necrotic tissue. The bandage, block, and wire should remain in place for at least 8 weeks to allow for support and proper alignment of the affected digit as healing progresses. In a recent study of DIPJ arthrodesis, lameness persisted for 1 to 7 months after surgery.[1]

Septic Tenosynovitis. Involvement of the plantar or palmar aspect of the third phalanx and/or the deep digital flexor (DDF) tendon may cause detachment or rupture of the DDF tendon. In such cases, the toe of the affected digit often tips proximally. The synovial sheath of the DDF tendon extends distal to the level of the coronary band to terminate at the middle of the diaphysis of the second phalanx. The sheath lies immediately proximal and palmar / plantar to the navicular bone and bursa. Thus, concurrent septic tenosynovitis is common if extensive infection of the navicular bone and bursa exists. Aggressive lavage with non-irritating polyionic solutions, drain placement, support bandages, and parenteral antimicrobial therapy are indicated for cases of septic tenosynovitis. If the infection results in rupture of the DDF tendon or detachment of the tendon from the third phalanx, placement of a cast should follow successful management of the infection. If cast placement is not an option, the toe of the affected digit should be wired to the sound digit in order to allow tendon healing to occur with the solar surfaces of the digits parallel to each other.

Septic osteitis of the distal phalangeal bone and / or the navicular bone: Deep sole ulcers or puncture wounds in the plantar or palmar third of the solar surface may progress to septic osteitis of the distal sesamoid bone (navicular bone) and its bursa. Necrotic areas of digital bone can be identified by their relatively soft consistency; a clean hoof knife, bone curette, or drill can then be used for thorough debridement. Excision of the navicular bone and debridement and lavage of the involved synovial structures is indicated.

Summary

The structures involved in the septic process, the animal's value and intended use, and the facilities available for surgery are frequently the main considerations that determine the method of surgical management. The methods of treatment described above is particularly suited for cases in which owner investment is limited and for cases to be managed under field conditions. If treatment is not successful, euthanasia should be conducted on-farm, rather than having the affected animal endure the stress of transport. To encourage early recognition of deep sepsis of the digit, the author advocates application of the following principles:

1. Causes of lameness can be categorized according to the likely presence or absence of visible swelling of the soft tissues of the foot.
2. Because interdigital necrobacillosis is centered in the interdigital skin, early cases are characterized by swelling that is symmetrical relative to the longitudinal (axial) midline of the foot.
3. Deep sepsis of the digit is characterized by swelling that is asymmetrical relative to the longitudinal (axial) midline of the foot.
4. On-farm lameness treatment protocols should include an expected deadline for resolution – once the deadline is reached, if the animal has not recovered, the veterinarian should be consulted.
5. Cattle that become lame from digital wounds should be scheduled for prompt veterinary examination.

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HOW I TREAT PENILE AND PREPUTIAL INJURIES IN BULLS

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Introduction

Injury to the external genitalia can result in loss of seasonal serving capacity and/or premature culling of breeding bulls. Bulls with preputial injuries must be thoroughly evaluated, potentially on a repeated basis, so that an accurate prognosis for recovery of intended breeding use can be made. If the injury is severe or is improperly treated, permanent loss of the affected animal's breeding ability may result. If preputial injuries are treated, treatment must be diligent and persistent, as several weeks of therapy may be required in cases of severe injury. In some cases, surgical therapy is eventually required to restore breeding ability. However, almost all cases of preputial injury in bulls require a relatively common set of medical treatments for the first several days to weeks following injury. In this article, evaluation and medical management of preputial injuries in bulls will be addressed.

Pathogenesis

The prepuce is the hairless, stratified squamous epithelium that is connected at the preputial orifice to the haired skin of the sheath. The prepuce extends to the shaft of the penis. During erection, the prepuce must evert as the penis is extended from the sheath. Elastic tissue located deep to the prepuce allows the prepuce to evert and stretch during penile extension. An injury that damages the prepuce and / or its underlying elastic tissue may result in phimosis, which is an inability to extend the penis from the sheath.

Contusion or laceration of the prepuce usually occurs during copulation, when the prepuce becomes entrapped between the bull's abdomen and the pelvis of the cow. These injuries may also occur immediately after copulation, when the bull dismounts with the penis still extended, or during habitual prolapse of the prepuce. The prepuce may then be kicked or stepped upon, or may come into contact with sharp objects in the environment. Bulls of the *Bos indicus* breeds are predisposed to preputial injuries because the sheath of these animals is pendulous, the prepuce is relatively long, and the preputial orifice is large. Polled bulls may be predisposed to preputial injuries because the retractor prepuce muscle may be rudimentary or absent.[1] On the other hand, owing to the dependent location of the prepuce, edema of the contused and lacerated prepuce may result in preputial prolapse. Tension created by the prolapsed segment may pull additional prepuce out of the orifice, resulting in the prolapse of normal, uninjured prepuce. Exposure and trauma to additional areas of prepuce can then occur.

Prolapse of the prepuce may also occur secondary to penile hematoma. In these cases, rupture of the tunica albuginea during erection forces a large volume of blood into the elastic tissues surrounding the distal aspect of the sigmoid flexure. The blood forms a clot in this location, and the pressure exerted by the clot and the resultant venous and lymphatic obstruction to the prepuce may cause secondary preputial prolapse.

Preputial prolapse may also occur secondary to sedation with such agents as acepromazine or xylazine. These agents may induce enough relaxation of the retractor penis muscle and retractor prepuce muscle to cause prolapse of the prepuce and less commonly, of the penis. Although the prolapse is usually mild and temporary and resolves as the sedative effects dissipate, the prepuce and penis are subject to injury while prolapsed. Thus, when these agents are used in bulls, precautions should be taken to prevent injury until recovery from sedation or anesthesia is complete.

Preputial prolapse is a potential sequel of balanoposthitis caused by bovine herpesvirus-1 infection. The inflammation associated with this viral infection may cause edema of the preputial mucosa, resulting in eversion and eventual prolapse.

Initial treatment and evaluation

It is essential that the affected bull be isolated from other cattle as soon as possible in order to prevent further injury from continued attempts to breed. To limit sunburn, drying, and frostbite, the affected bull should be moved to a sheltered area. Producers should be strongly encouraged to seek veterinary intervention as soon as possible. A thorough physical examination should be performed because detection of other disorders in the affected bull may influence the decision to treat or the course of treatment.

The injured prepuce should be gently cleaned with warm, soapy water. Soaking the prepuce may loosen adherent dirt and necrotic debris. The bottom half of a 1-gallon plastic bottle can be rinsed, filled with soapy water, and suspended around the bull's abdomen with gauze or string to create a soaking container.

When inspecting and palpating a preputial injury, the veterinarian should determine as well as possible the extent of prepuce involved, the degree of necrosis and fibrosis, the orientation, depth, and size of any abrasions or lacerations, and the presence of concomitant urogenital disorders.

The use of an electroejaculator to extend the penis for inspection of the prepuce is dangerous, because swelling of the prepuce may prevent retraction of the penis back into the preputial cavity, resulting in paraphimosis. In cases of penile hematoma, extension of the penis by electroejaculation will cause the hematoma to enlarge and is not recommended. In many cases, the entire prepuce cannot be visually evaluated at the time of initial examination, and repeat inspection of the prepuce after initiation of medical therapy may be necessary. In such cases, resolution of edema within the injured tissues after several days of medical therapy often facilitates more thorough visualization and palpation of the prepuce. Endoscopy of the preputial cavity can be used to visualize the extent of injuries to the retracted portions of the prepuce, although this practice usually does not change the course of medical management. Ultrasonography can be used to evaluate the condition of the penis and the elastic tissue deep to the prepuce; this imaging modality is most useful when applied to differentiate penile hematoma from retropreputial abscesses.

Because a dense bacterial population normally inhabits the prepuce, secondary infection of abrasions and lacerations occurs commonly. Bruised areas beneath the preputial epithelium are also prone to infection, as blood is an excellent medium for bacterial growth. In cases of laceration or necrosis of the prepuce, extension of infection into the deep elastic tissue may result in acute septic cellulitis of the retropreputial tissue. Bulls affected by acute retropreputial cellulitis show diffuse swelling of the sheath, with affected areas being warm and painful to digital pressure. With time, the infection localizes into a retropreputial abscess. Retropreputial abscesses are characterized by a localized swelling in the sheath that is often mildly painful or non - painful to pressure. The swelling may be firm if the abscess capsule is thick or fluctuant in cases with a thin capsule. Purulent exudate may be expressed into the preputial cavity when pressure is placed on the swelling.

Differentiation of retropreputial abscesses from penile hematomas is critical. A penile hematoma typically manifests as a swelling in the sheath that begins immediately cranial to the scrotum, at the distal sigmoid flexure of the penis. Penile hematomas are symmetric along the longitudinal axis of the penis. Retropreputial abscesses, on the other hand, are usually located more distally (cranially) in the sheath and are rarely symmetric along the longitudinal axis of the penis. Percutaneous aspiration of such swellings is not recommended because 1) if the swelling is a penile hematoma, infection may be introduced despite efforts at aseptic technique; and 2) if the swelling is a retropreputial abscess, infection will inevitably spread into additional areas of the elastic tissue, thereby increasing the likelihood of peripenile adhesion formation.[1]

Ultrasonographic examination is helpful to differentiate between these two conditions if results of examination are inconclusive. Retropreputial abscesses are characterized by *homogenous*, echogenic contents within a distinct, echogenic capsule of variable thickness. In contrast, ultrasonographic examination of a penile hematoma reveals a

pattern of *heterogeneous* echogenicity surrounding the distal sigmoid flexure of the penis. The center of the hematoma may show lacy or honeycomb-like echogenic structures (fibrin and clotted blood) within a background of echolucent fluid (serum). A thick capsule is not visualized on the periphery of hematomas.

Prognosis

The prognosis in cases of preputial injury is influenced greatly by the nature, extent, and chronicity of the injury. Provision of an accurate prognosis may be limited by the veterinarian's inability to evaluate the entire prepuce and penis if retraction of the injured area into the sheath occurs. In addition, in the days immediately following an injury, the veterinarian cannot always accurately predict the development of such sequelae as retropreputial abscesses, preputial stenosis, and peripenile adhesions. It is prudent for the veterinarian to explain these limitations to the owner so that he or she is aware of the potential for unforeseen complications to develop.

Lacerations or necrotic segments of prepuce that are identified as having circumferential or hemi-circumferential orientation may create stenosis of the preputial cavity and phimosis once scarring is complete. Retropreputial abscesses warrant a grave prognosis for natural service because the elastic tissue is replaced with the fibrous tissue of the abscess capsule. Adhesions may develop between elastic layers or between elastic tissue and the skin of the sheath. Such adhesions limit preputial extension during erection, resulting in pain during erection and / or phimosis. Surgical drainage into the preputial cavity, along the limits of the original preputial laceration, may be attempted but is rarely successful. Drainage of retropreputial abscesses through the skin of the sheath is not recommended, as this procedure virtually guarantees development of peripenile adhesions.[2]

The length of prepuce involved in the injury is another important determinant of prognosis. For successful natural service, bulls require a normal, mobile prepuce that is approximately 1.5 times the length of the penis from the site of preputial attachment to the glans (the free portion of the penis).[2] Necrosis and/or fibrosis of large areas of the prepuce may result in limitation of the length of normal prepuce, with resultant limiting effects on penile extension.

Concurrent penile laceration is uncommon, but may create additional complications that jeopardize breeding ability. In such cases, impotence may occur if the laceration involves the dorsal nerves of the penis and produces sensory loss to the glans. Affected bulls can achieve erection but fail to achieve intromission and ejaculation during natural breeding. Deep lacerations that penetrate the tunica albuginea of the penis may heal with fistulas that allow blood to leak from the corpus cavernosum during erection, resulting in erection failure or blood contamination of the semen.
While severely affected bulls or those of limited economic value may be salvaged for slaughter, valuable bulls or those with reparable or minor preputial injuries may be treated with medical or medical and surgical therapy. In cases of uncertain prognosis, re-evaluation after 1-2 weeks of medical therapy may more accurately indicate the extent of injury, as this time period often allows for sloughing of non-viable tissue and resolution of edema and bruising. A more accurate prognosis may be possible at this stage.

The future use of the bull in natural or artificial service settings should also be considered in the prognosis. Natural service requires that the preputial injury heal to the extent that extension, intromission and ejaculation can occur on a frequent basis, necessitating nearly complete restoration of penile and preputial mobility, elasticity, and tissue strength. Artificial collection of semen demands less frequent services by the bull, and collection techniques can often be altered to fit the limitations of the injury.

Treatment

The decision to treat a bull with a preputial injury should be made after careful consideration of economic factors and the owner's wishes. Treatment decisions should also be made with the understanding that, at the time of initial examination, the veterinarian cannot always accurately predict the type and severity of complications associated with these injuries. In the Western USA, many commercial cow-calf

operators maintain a 45 to 60-day breeding season, and bulls with significant preputial injuries are frequently disabled for at least this length of time. Thus, injured bulls often must be removed from the breeding herd for that breeding season, a circumstance that may greatly influence the decision to treat.

Kasari and colleagues[3] have developed an economic model that compares the cost-effectiveness of treatment of an affected bull with culling of the affected bull and replacement with a healthy bull. Treatment may be economically justified for bulls with very high genetic merit, provided that the replacement price for such bulls is sufficiently high. Treatment may also be requested for affected bulls of sentimental value to the owner.

Treatment

Edema, bruising, and wound contamination are often advanced at the time of initial examination, usually precluding the option for immediate surgical repair of penile or preputial lacerations. Therefore, the goals of treatment are typically 1) to protect the prepuce from further trauma; 2. promote resolution of preputial prolapse; 3. limit infection of affected tissues; and 4) allow for second intention healing of any preputial or penile lacerations. During treatment and recuperation, strict isolation of affected bulls from all other cattle is necessary to limit recurrent injury during breeding attempts.

If lacerations, extensive bruising or necrosis, cellulitis, or severe edema is present, prompt initiation of parenteral antimicrobial therapy is recommended. Antimicrobial therapy limits the development of retropreputial abscesses and facilitates the healing of contaminated wounds. Long-acting oxytetracycline (22 mg/kg SC q 72 h) or procaine penicillin G (22,000 IU/kg IM BID) have been used in our clinic with success. If facilities or the bull's temperament do not allow for repeated injections to be administered, sulfadimethoxine may be used (55 mg/kg PO loading dose, 27 mg/kg PO q 24 h maintenance dose). Treatment should be continued until bruising has resolved and any lacerations are filled with a healthy bed of granulation tissue, a process that usually requires 1-3 weeks to complete.

Administration of non-steroidal anti-inflammatory drugs may be beneficial in resolving swelling of inflamed tissues, thereby facilitating resolution of prolapse, reducing pain, and facilitating urine egress. Treatment with flunixin meglumine (1.1 mg/kg IV q 24 h) should be initiated promptly after diagnosis and continued for approximately 5-7 days. If the option for prompt slaughter is to be retained, aspirin (100 mg/kg PO q 12 h) can be administered.

If the prepuce is prolapsed, additional therapy aimed at reducing edema in the affected prepuce must be initiated. The goal of this treatment is to reduce the edema such that the prepuce can be retracted back into the sheath, where it is relatively protected against desiccation and further trauma. Resolution of edema of the prolapsed prepuce can be promoted with 30-60 minutes of hydrotherapy per day. Warm water jetted onto the prolapse from a hose is used to massage the prolapsed tissue. Hydrotherapy also aids in removing necrotic debris and exudate from the wound surface. A support sling should be used to limit the formation of dependent edema in the prolapsed prepuce. Heavy burlap, canvas, or netting can be suspended beneath the bull's abdomen with surgical tubing or sections of inner tube to create a flexible cradle that presses the prepuce against the ventral abdomen. Ointments containing antimicrobial compounds can also be applied one or two times each day. The author has used a mixture of 40 ml of procaine penicillin G (300,000 IU/ml) in 16 oz. of anhydrous lanolin ointment.

Application of a preputial support bandage is an effective means of protecting the prolapsed prepuce and promoting resolution of edema. The exposed prepuce is coated with an emollient ointment. A length of orthopedic stockinette can be used to cover the exposed prepuce and limit its contact with the bandage adhesive. A 15-25 cm length of latex tubing of 0.75-1.5 cm diameter is inserted into the preputial lumen; a mid-sized large animal stomach tube works well for this purpose. This tubing directs urine to the exterior and acts as a template against which the bandage exerts pressure. The tubing should be inserted gently into the preputial lumen until slight resistance is met, and then maintained at that level for

incorporation into the bandage. Approximately 5-10 cm of tubing should protrude from the end of the prolapsed prepuce.

The tubing and stockinette are held in place with elastic tape wrapped around the circumference of the prolapsed prepuce. The protruding section of tubing should be thoroughly dried, and the elastic adhesive bandage should be wrapped around it. The elastic adhesive bandage wrap is then continued proximally around the stockinette and prepuce until it is anchored in the haired skin of the sheath at the level of the ventral abdomen. To limit the tendency for the bandage to roll distally on the prepuce, the upper edge of the bandage can sutured to the skin of the sheath. Two to four simple interrupted sutures are placed in the skin of the sheath near the level of the abdominal wall. One end of this suture is passed through the upper edge of the bandage. The ends of the knotted suture are left long and are tied in a bow knot, thereby allowing the sutures to be untied for bandage changes. The amount of exudate and degree of bandage soiling will determine the necessary frequency of bandage changes. The bull should be observed several times per day to ensure that urination occurs freely through the tube.

In the initial stages of treatment, daily bandage removal for cleaning and hydrotherapy of the prepuce, followed by repeat bandaging, may be necessary. When the prolapsed prepuce can be retracted or manually replaced into the preputial cavity, the bandage can be applied without a stockinette, with the tubing and elastic tape being used to keep mild pressure on the prepuce and keep it retained within the preputial cavity.

Amputation of the prolapsed prepuce can be considered for bulls with extensive injury, necrosis, or fibrosis of the affected tissue.

Re-evaluation

Even in cases of minor preputial injury, careful reevaluation is necessary before recovered bulls are returned to the breeding herd. Returning the bull to natural service prematurely may result in aggravation of injury because the healing tissue may lack the strength to withstand the forces of motion and friction encountered during natural service. Up to 60 days, possibly more, of sexual rest may be necessary to allow complete tissue healing in cases of preputial laceration. Thus, if the convalescence period for an affected bull occurs during the planned breeding season, a replacement bull will be required.

In cases of deep lacerations, extensive preputial necrosis, or extensive preputial prolapse, preputial bandaging and topical therapy may need to be continued for up to 6 weeks. These treatments should be continued until edema and prolapse have resolved and all necrotic tissue has sloughed. Frequent reevaluation is recommended in order to detect abscesses deep to the prepuce. Abscesses should be drained into the preputial cavity rather than through a skin incision, as the latter technique frequently results in severe adhesion formation.

After medical treatment and convalescence are completed, the bull's ability to perform the intended means of service may be estimated by manual extension of the penis, which is aided by sedation and / or trans-rectal massage of the urethra, seminal vesicles, and prostate. Manual extension allows the veterinarian to evaluate the degree of scarring of preputial lacerations and the ease of penile extension. Penile extension can also be evaluated during careful, gradual stimulation with an electroejaculator. A trial breeding can also be used for observation of breeding ability; this should be avoided, however, if exacerbation of the previous injury is deemed probable.

Surgical intervention is needed if there remains a potentially correctable lesion that prevents the bull from either performing natural service or having semen collected for artificial insemination. Fibrosis (scarring) of lacerations is a complication of preputial injuries that may require surgical repair to return the bull to the desired level of breeding soundness. Fibrosis may cause stricture of the preputial cavity or orifice, limit normal extension of the prepuce, or influence the direction of penile extension during erection. Posthioplasty and amputation are commonly used surgical procedures used to resect fibrotic areas of the prepuce. A 60-day postoperative convalescence period has been recommended following these procedures.[2]

Similar convalescent periods are required following drainage of a retropreputial abscess.

Summary

Prompt identification of preputial injuries is critical for successful medical management. If the value of the bull warrants treatment, aggressive medical management and strict sexual rest are necessary to expedite healing. Diligence, persistence, and patience are necessary during the period of medical management because returning the bull to breeding service prematurely can result in reoccurrence or worsening of the injury. Surgical treatment of preputial injuries may be necessary to return the bull to natural service, but such procedures should be delayed until second intention healing appears to be complete.

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HOW I TREAT THE ACUTE ABDOMEN

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Introduction

An acute abdomen will be defined as a potentially serious disease involving intra-abdominal organs that presents with a sudden and possibly progressive onset of signs. A thorough history, inspection of the feed, and physical examination (including examination per rectum, when possible) are essential for an accurate diagnosis and treatment plan. Without surgical exploration, an accurate prognosis may be difficult to render, depending on the suspected disease. Ancillary diagnostic tests may help to refine the diagnosis prior to or in lieu of surgical exploration; these include compass testing for presence of a reticular magnet, rumen fluid analysis, trans-abdominal ultrasonography, abdominocentesis, and, where available, cow-side tests of blood acid-base and electrolyte disturbances. In this seminar, the author's approach to the bovine patient with the acute abdomen will be described.

Signalment and history

Careful consideration of the signalment is a necessary first step, simply because certain disorders occur at a greater frequency in animals of a certain age and production status. Examples include rumen acidosis in feedlot animals, atresia coli in newborn calves, inguinal hernia in bulls, uterine torsion in periparturient animals, and urolithiasis in intact and castrated males. Cows and heifers in the puerperal period should be considered at greater risk of uterine perforation, ileus from hypocalcemia, and traumatic reticulitis. The onset, duration, and progression of the chief complaint(s) should be clarified in the history. It is vital that the veterinarian establish that the animal in question is experiencing a truly acute disorder versus acute complications of a pre-existing, subacute to chronic problem; the latter circumstance brings a vastly different set of differential diagnoses to mind than the former.

The veterinarian should inquire if similar diseases or signs of disease have been identified on the premises, and, more specifically, in the same age or production group of the animal in question. An affirmative response may be indicative of a problem rooted in nutrition, animal management, toxin exposure, or infectious causes, while a negative response lends weight to consideration of more sporadic, spontaneous etiologies. Because of the central role that changes in diet have in many acute gastrointestinal disorders, a specific query about this topic is warranted. Diet composition, quantity, frequency and mode of presentation, and changes in social stratification of the group may be clues to a change in diet for the affected animal.

A brief inspection of feed is often informative. Does the feed in question carry inherent risk for choke, rumen acidosis, or frothy bloat? The author makes a point to note the quantity of fiber in the ration. If the ration is composed primarily of roughage, it is expected that palpation of the rumen through the left paralumbar fossa (and, when possible, per rectum) should reveal the presence of a fiber mat. If such a fiber mat is not palpable, these possible explanations are considered:

1) The animal has been off of feed for longer than expected for an acute abdominal disorder, and the fiber mat has degraded with time;
2) A large volume of fluid has been ingested or administered by orogastric tube;
3) Egress of ingesta from the reticulorumen is impaired by obstruction or motor dysfunction of the reticuloomasal groove;
4) Generation of stable foam (froth) in the rumen precludes palpation of a fiber mat;
5) Water has been drawn by an osmotic gradient into the rumen (as expected with acute ruminal acidosis or urea intoxication); or

6) The voluminous abomasal secretions (HCl and water) have been retained in the forestomachs from a pyloric outflow obstruction, forestomach motility disorder, or disease of the proximal small intestine.

If the signalment, history, or examination data raise the suspicion of 4-6 above, the author recommends that a sample of rumen fluid be obtained for analysis of physical features, pH, and, if possible, chloride concentration. Rumen fluid chloride concentration aids in determining that abomasal outflow is impaired; a value of less than 30 mEq/l is considered normal. However, the workup is not reliant upon obtaining this parameter.

Physical examination

It is essential that the affected animal's demeanor, posture, and abdominal contour be evaluated from a distance prior to hands-on examination. Profuse salivation, bloat, and extension of the head and neck are suggestive of esophageal obstruction. Lordosis can be indicative of renal pain; if accompanied by a splayed stance (forefeet forward and hind feet to the rear), urinary outflow obstruction may be present. Rectal prolapse can be a primary disorder or a complication of excessive straining from proctitis, urethritis, vaginitis, or neurologic disorders such as rabies. Early in the course of small intestinal obstruction, distension of the lower right abdominal quadrant may be seen; as intestinal distension progresses, however, the abdomen typically shows a uniformly rounded appearance. Distension of the left upper- and left and right lower quadrants can accompany ruminal free-gas or frothy bloat; however, the potential for disease aborad to the rumenoreticulum should be considered, as retained ingesta may be flooding the ruminal cardia, causing secondary bloat. Severe ruminal tympany may warrant immediate intervention by rumen trocarization.

During physical examination, the author recommends that sources of pain outside of the peritoneal cavity be considered and ruled out; these include the kidneys, bladder, ureters, urethra, vagina, and uterus. Compromised esophageal patency can produce acute ruminal tympany; esophageal patency can be compromised by intraluminal or extraluminal (mediastinal) diseases. The umbilicus should be palpated carefully in neonates presenting with signs of abdominal disease, as these signs could reflect adhesions between abdominal viscera and inflamed internal components of the umbilical vasculature and/or urachus.

Physical examination should be conducted in the veterinarian's preferred sequence. At some point in the examination, a compass should be held near the animal's sternum to determine if a reticular magnet is present. If so, veterinarian should inquire as to whether or not the magnet had been administered recently, possibly in response to early signs of gastrointestinal dysfunction. The presence of a magnet reduces, but does not eliminate, the likelihood of traumatic reticuloperitonitis and associated sequelae. Sharp objects composed of aluminum, copper, or plastic can cause reticuloperitonitis in the presence of a rumen magnet.

Simultaneous auscultation and ballottement (pinging) is a very useful technique for identifying gas accumulation in various parts of the gastrointestinal tract. Succussion (auscultation during deep ballottement of the abdomen) may reveal sounds of fluid accumulation within a viscus. Whenever possible, the author prefers to use rectal palpation and/or trans-abdominal ultrasonography to confirm suspicions of the origin of gastrointestinal disease that pinging and succession might reveal. Rectal palpation is considered a reliable means of confirming cecal dilatation, cecal volvulus, intussusception, and small intestinal volvulus; abnormal rectal examination findings are less consistent for hemorrhagic bowel syndrome (jejunal hemorrhage syndrome).[1,2] The contour of the caudal and right walls of the rumen should be evaluated during rectal palpation; retention of ingesta in the forestomachs is characterized by a subjectively enlarged rumen with a ventral

sac that extends to the right, creating a rough “L” shape to its medial wall.

Trans-abdominal ultrasonography can be utilized as an adjunct test to facilitate the decision for surgery. It is typically conducted in the right paralumbar fossa with a 3.5 – 5.0 MHz sector- or linear-array probe. During ultrasonographic examination, the author’s primary intent is to determine if loops of distended small intestine are present and if normal peristaltic activity exists. Normal intestine, as described by Braun[3], has a wall thickness of 2-3 mm and a luminal diameter of 2-4 cm. The diameter of normal small intestine changes constantly with peristalsis.

The author begins scanning in the ventral right paralumbar fossa and slowly moves the probe dorsally. Diffuse distension of the small intestine with ileus could reflect intestinal obstruction, paralytic ileus from acid-base or electrolyte disorders, enteritis, peritonitis, or indigestion (a.k.a. pseudo-obstruction). Pseudo-obstruction typically produces mild to moderate distension of small intestine (3-6 cm); importantly, luminal size is varied, and weak peristaltic contractions are typically visible. In cases of pseudo-obstruction, fecal passage is typically restored with rehydration, analgesics, and treatment with calcium salts.[1] Obstructions such as hemorrhagic bowel syndrome and intussusception are characterized by more severe and uniform dilation of bowel, with little to no peristaltic activity evident over time. Extraluminal fluid suggests fluid transudation from compromised bowel or peritonitis; ultrasonography greatly aids in identifying sites of fluid accumulation for abdominocentesis. A solitary tube of distended bowel that spans the paralumbar fossa from cranial to caudal is likely distended duodenum. This finding may accompany the relatively rare disorders of the proximal small intestine. The absence of distended loops of small intestine greatly reduces the likelihood of strangulating- or non-strangulating small intestinal diseases. If abdominal distension is present but no distended small intestine is seen on ultrasonography, obstruction of the duodenum, pylorus, or reticulo-omasal orifice should be considered. These conditions would be expected to result in an enlarged rumen with minimal to no fiber mat present. Obstructions of the pylorus and duodenum (e.g. abomasal impaction or lymphosacroma) would be expected to induce hypochloremic metabolic alkalosis.

Decision for Surgery

The decision to perform a laparotomy in cattle with abdominal disease involves integration of the signalment, history, physical examination findings, and, if available, ancillary diagnostic tests such as ultrasonography. This decision may be influenced by such factors as available facilities, the veterinarian’s expertise, and the animal’s physical status. If the clinical signs do not clearly indicate the need for prompt surgical intervention, or if medical treatment is to be attempted first, the veterinarian should clearly outline to the owners or caretakers those changes in patient status that will indicate a positive response to medical treatment. Medical management may consist of oral and/or parenteral fluid therapy, antimicrobials, a rumen magnet, analgesics, calcium salts, antacids or gastrointestinal protectants, laxatives, anti-foaming agents, and/or judicious amounts of palatable feeds.

Repeat evaluation must occur at a frequency sufficient to detect any deterioration in status that might support a decision for surgical intervention. Parameters to evaluate include attitude, appetite (if feed and water are to be offered), fecal production, abdominal contour, presence of tympany or fluid on ballottement, heart and respiratory rates, rectal temperature, hydration status, membrane color, and abdominal distension. The degree of abdominal distension can be monitored over time by serial measurement of the abdominal girth. A tape measure or a strip of bandage tape with marks made to denote serial measurements can be used for this purpose. The abdominal girth should be measured at the same location each time. The space between the second and third lumbar vertebrae is a potential site for girth measurement, as is the caudal edge of the

13th ribs on each side. Progressive abdominal distension indicates that gas, fluid, and/or ingesta are accumulating in the gastrointestinal tract or abdominal cavity; surgical intervention is usually prompted by such findings unless there is a clear cause for the distension that can be addressed with medical management. If economic limitations do not allow surgical intervention, euthanasia should be performed at the point in time when the animal's status reaches the point where surgical correction is necessary to limit suffering. In cases for which no specific diagnosis can be made, if the gastrointestinal disease process is characterized by accumulation of gas, fluid, or ingesta, progressive shock, and/or colic, surgical exploration should be considered.[4]

Preoperative Stabilization

For adult cattle with dehydration secondary to diarrhea or grain engorgement, alkalinizing fluids such as Lactated Ringers and similar polyionic fluids are indicated to correct the expected metabolic acidosis.[5] These fluids have sodium and chloride concentrations that are typically at the low end of or below the reference range for bovine serum sodium and chloride concentrations. In contrast, dehydrated cattle with functional or mechanical obstructions of the gastrointestinal tract frequently need fluids with sodium and chloride concentrations that are greater than typical serum concentrations of these electrolytes, such as isotonic or hypertonic sodium chloride.[5] Potassium supplementation of intravenous fluids may often indicated in cattle with gastrointestinal disease.[5] Cattle dehydrated by the metabolic consequences of urinary tract obstruction also frequently benefit from sodium- and chloride-rich fluids delivered orally or parenterally; however, potassium supplementation of fluids should be used with extreme caution in animals with urinary tract obstruction.

Hypertonic saline, at a dosage of 4-5 ml/kg of 7.2% sodium chloride IV rapidly, provides rapid and convenient volume expansion for hypovolemic cattle.[6] This treatment should be followed by allowing the patient access to water to drink or by orogastric intubation of water. Excess water ingestion may result in rumen expansion of sufficient magnitude to hamper surgical exploration of the abdomen, particularly if significant abdominal distension already exists. Further, if sedation and recumbency or general anesthesia is anticipated, oral water loading may promote regurgitation. In such cases, following hypertonic saline with parenteral isotonic fluids may be a safer choice.

Preoperative flunixin meglumine (1.1-2.2 mg/kg IV) has been administered for analgesia during standing surgery. Results are inconsistent. In the author's experience, standing cattle often attempt to lie down when diseased bowel is exteriorized. In such cases, an assistant is often helpful in minimizing the amount of bowel that needs to be exteriorized for surgery; his or her support of the intestinal mass may also reduce painful tension on the mesentery. In such situations, is often helpful to tie the animal's head with a halter pulled toward the side of the surgical approach - if the animal lies down during the procedure, it will usually end up with the surgical side facing upwards. For debilitated animals, securing the animal in lateral recumbency at the onset of surgery may limit the disruption that occurs when the standing animal lies down. To facilitate closure of a flank laparotomy incision in an animal in lateral recumbency, pillows or padding can be placed under the down-side shoulder and down-side hip to induce concavity in the up-side flank. Pre-operative antimicrobial therapy is indicated if bowel incision is anticipated, if surgical time is anticipated to be prolonged, or if concurrent infection exists.

If standing surgery is to be performed, sedation should always be considered with great care when applied to the bovine patient, as excessive sedation may cause the animal to assume recumbency during surgery. For chemical restraint during standing surgery in an adult dairy cow, the author prefers the so-called "5-10-20" combination of 5 mg butorphanol, 10 mg of xylazine, and 20 mg of ketamine, administered IM.[7] In the authors' opinion,

sedation and analgesia imparted by this drug combination appears less likely to induce recumbency than xylazine or xylazine-butorphanol in combination.

Summary

Careful consideration of the animal's signalment, history, and intended use, along with a careful physical examination, remain the cornerstones to effective decision making when the practitioner is faced with acute abdominal crises in cattle. If surgical exploration is not undertaken immediately and medical treatment is elected, the practitioner should clearly delineate the parameters of the animal that are to be monitored to guide subsequent treatment decisions. Monitoring abdominal girth, comfort, fecal output, appetite, and abdominal contour are often useful in this regard. When fluid therapy is warranted, most cattle with functional or mechanical obstructions of the gastrointestinal require fluids enriched in sodium and chloride; isotonic or hypertonic saline are appropriate choices.

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HOW I TREAT THE CHRONIC ABDOMEN

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Introduction

The bovine patient with chronic abdominal disease is most often suffering from functional or structural impairment of normal forestomach and/or abomasal emptying. In contrast, chronic disorders of the large or small intestine are relatively rare and are typically characterized by a reduction in luminal diameter imparted by adhesions or progressively enlarging masses such as abdominal fat necrosis or neoplasia. Impaired forestomach or abomasal emptying typically results in a syndrome commonly referred to as vagal (or vague) indigestion. The prognosis for vagal indigestion (VI) is commonly deemed guarded, as many of the underlying causes are not reversible nor readily corrected by surgery. However, a select subset of VI cases can be expected to have a satisfactory outcome; the challenge lies in identifying those "candidates for success" animals by routine means, such as physical examination. In this seminar, the causes of VI will be reviewed, published data on outcome (where available) will be provided, and the author's approach to management of these cases will be described.

History and physical examination

Cattle with VI have been considered to suffer from an underlying, primary disease process that causes interruption of normal forestomach motility (functional problems) and / or mechanical obstruction of the normal flow of ingesta (structural problems). Inflammatory processes involving the cranial abdomen are relatively common precursors to VI. These processes include, but are not limited to, traumatic reticuloperitonitis, hepatic abscesses, perforating abomasal ulcers, and compromise to the abomasal wall and/or abomasal innervation imparted by a previous bout of abomasal volvulus or right displaced abomasum.[1,2] In these cases, functional obstruction is thought to result from 1) inhibitory sensory signals sent from the inflamed locus to the gastric centers of the brain, resulting in disturbed central neural coordination of motility; or 2) direct involvement of branches of the vagal nerve in the disease process.[2] Structural obstruction to normal forestomach and abomasal emptying may also be the consequence of inflammatory processes that cause the development of abscesses or adhesions. Structural obstruction may also occur from intraluminal masses (e.g. papillomas of the forestomach mucosa[3]), foreign bodies, or extraluminal compression (e.g the gravid uterus).

Various classification schemes have been proposed for VI, beginning with the 4 classes or types proposed by Hoflund.[4] Since each carries multiple potential primary causes, each can be considered a disease syndrome. A simplified classification scheme has been proposed by Ducharme and Fubini:[5] Type 1: Failure of eructation; Type 2: Failure of omasal transport; and Type 3: Failure of abomasal outflow. Failure of eructation is characterized by accumulation of free gas in the rumen owing to:

- obstruction of the esophagus or cardia;
- disturbance of the eructation process by dietary factors, physiologic immaturity, or inflammatory disease in the thoracic or abdominal cavities; or
- unknown causes.

Failure of omasal transport causes impairment of emptying of the rumen and reticulum; a variety of functional and structural causes have been proposed. Failure of pyloric outflow typically results in reflux of abomasal secretions (HCl) into the forestomachs, typically producing hypochloremic metabolic alkalosis. As for omasal transport failure, multiple inciting causes of functional or structural impairment of abomasal emptying have been identified.

A relatively common cause of failure of abomasal emptying is a preceding bout of right displacement of the abomasum (RDA) or abomasal volvulus (AV).[1,2]

Types 2 and 3 VI are characterized by subacute to chronic gastric distension and abdominal distension caused by retention of ingesta in the forestomachs and/or abomasum. These syndromes typically occur secondary to a preceding disease process that may or may not have produced noticeable clinical signs, such as occult traumatic reticuloperitonitis or hepatic abscess formation. Cattle with these types of VI show reductions in appetite, fecal output, and milk production. Rectal temperature, hydration status, fecal appearance, and heart rate are variable, depending on the inciting cause and the subsequent clinical course. Bradycardia may be apparent in some cases. Expansion of the rumen results in enlargement of the dorsal sac, seen from the rear of the animal as distension of the left upper quadrant of the abdomen. Expansion of the ventral sac of the rumen results in distension of the lower right abdominal quadrant. The resulting abdominal shape is commonly referred to as a "papple" (pear and apple) shape. Rumen contraction frequency may be increased. The failure of ingesta to exit the stomach in normal fashion typically results in rumen contents being mixed into a froth.

Ancillary diagnostic tests may help define the underlying cause, but these are not always available to the clinician in ambulatory (field) practice settings. These include 1) a complete blood count, which may reveal underlying inflammatory disease; 2) serum chemistry analysis, which may help differentiate type 2 from 3 VI; 3) rumen chloride measurement, which can aid in confirming type 3 VI; 4) abdominocentesis; and 5) reticular radiography and/or ultrasonography. In many cases, when the expense of these tests is considered in light of the uncertain or guarded prognosis, testing is not conducted, and the animal is euthanized, salvaged for slaughter, or treated medically.

Treatment

Failure of eructation (type 1) is evaluated by passage of an orogastric tube and analysis of rumen fluid. Acute obstruction of the esophagus can be remedied by sedation and gentle passage of the orogastric tube. Small volumes of obstetrical lubricant can be infused to aid in relief of the obstruction. Rarely, rumenotomy is needed to relieve the obstruction; retrograde esophageal intubation and gavage can aid in removing the obstruction. Acute ruminal acidosis and frothy bloat are diagnosed by assessment of rumen fluid. Treatment of these conditions has been reviewed.[6]

Idiopathic failure of eructation typically presents as an animal with chronic or recurrent bloat. Such animals should be evaluated for esophageal patency by orogastric intubation. When no intraluminal obstruction is encountered, extraluminal compression of the esophagus (e.g. by mediastinal diseases) or neurogenic paresis of the eructation process should be considered as potential causes.[1,6] In a teaching hospital setting, the author has pursued documentation of underlying thoracic disease with specific ancillary tests (complete blood count, thoracic radiographs) with no consistent findings appreciated. In calves documented or suspected to have pneumonia that precedes bloat, the author has used a 1-2 week course of a macrolide antibiotic and an anti-inflammatory agent with some success. Placement of a small rumenostomy may enable release of gas for the short- to intermediate term. Rumenostomies are typically left in place for 2-3 weeks by placement of a tube or open syringe case into the stoma. Removal of the tube results in gradual closure of the stoma site. If bloat recurs after healing of the rumenostomy, valuable animals may be candidates for exploratory laparotomy and rumenotomy. Coarse foreign bodies (ropes, masses of twine, tinsel) have been retrieved from the rumenoreticulum of animals with chronic or recurrent bloat, and removal of these abrasive objects has led to recovery.

Cattle that develop signs of VI after surgical correction of RDA or AV warrant a poor

prognosis for recovery of production.[1,2] Focal or diffuse peritonitis, thrombosis of major vessels in the gastric wall, necrosis of the abomasal wall, and/or lesions of the vagal nerve account for the poor outcome.[2] The cause for VI as a rare consequence of simple RDA (i.e. without volvulus) was not apparent in a Canadian study.[2]

Medical management of type 2 or 3 vagal indigestion consists of administration of a magnet, antimicrobials, anti-inflammatory agents, oral or parenteral fluids, and / or laxatives. Medical management of vagal indigestion is most clearly indicated for indigestion of advanced (late) pregnancy, wherein the cow's gravid uterus creates a mechanical obstruction of the abomasum and/or proximal small intestine. Using the classification scheme proposed by Ducharme and Fubini,[5] this disease is categorized as a type 3 vagal indigestion, as rumen chloride concentration is increased in affected cows.[7,8] These cases typically resolve with natural or induced parturition, although fatalities may occur from impairment of respiration brought forth by the distended forestomachs. In a case series of indigestion of late pregnancy from India, cattle and buffaloes experienced normal lactation following parturition and gave birth to normal calves.[8] Recurrence of indigestion of late pregnancy appears to be uncommon.[8]

If VI cases subsequent to RDA or AV are excluded, the likelihood of successful surgical correction of VI is difficult to predict, owing to the variety of underlying causes. If traumatic reticuloperitonitis is the underlying cause, rumenotomy with removal of the foreign body and drainage of the perireticular abscess may result in recovery and return to productivity.[1] Cattle with VI associated with hepatic abscesses may require an initial rumenotomy to detect the abscess(es) and a second ventral surgical approach for drainage with a tube.[9] Excluding those with indigestion of advanced pregnancy, cattle with type 3 VI may have abomasal impaction, adhesions in the pylorus or duodenum, lymphosarcoma of the abomasal wall, or, rarely, foreign bodies lodged in the pylorus. The likelihood of successful surgical management of abomasal impaction, disorders of the pyloric area, and duodenal lesions can be subjectively judged as low. In contrast, type 2 VI caused by foreign bodies lodged in the reticuloomasal orifice is potentially correctable by rumenotomy and removal of the obstruction. Feed bags, clothing, plastic bags, and trichophytobezoars may become lodged in the reticuloomasal orifice; creating type 2 VI.

If rumenotomy is elected, the author prefers to orient the vertical incision as far cranially in the left paralumbar fossa as possible, so as to enable palpation of the reticulum and reticuloomasal orifice in larger cows. The incision is made large enough to accommodate the shoulder and arm. A line block of lidocaine is usually sufficient for this purpose. The animal is restrained in the standing position with the head secured in a halter and pulled toward the cow's left side. A standing paralumbar celiotomy is performed. In cattle with VI, immediate exploration of the abdomen in the face of ruminal distension often creates discomfort for the animal, which carries the risk of the animal assuming recumbency with an open laparotomy incision. The author prefers to forego abdominal exploration upon entry into the abdomen; instead, the rumenotomy is conducted first. The author prefers to suture the rumen wall to the skin rather than use a rumen board (Weingarth apparatus). The seromuscular layer of the rumen is sutured to the skin in a simple continuous pattern using heavy (#3) nonabsorbable suture material. Maintaining traction on the suture ensures that a good seal is made between the rumen wall and the skin. The rumen is then incised, the rumenoreticulum is emptied of ingesta, and the rumenoreticulum is explored. The omasum normally creates a palpably firm, spherical indentation in the cranial aspect of the medial (right) rumen wall. The surgeon can slowly move his or her fingers toward the cranial aspect of this indentation to find the reticuloomasal orifice, which presents as an elongated oval-shaped orifice with its longest axis in the dorsoventral plane. Normally, 3-4 fingers of

the right hand can be introduced into the lumen of this orifice. It should be explored carefully for masses or strands of foreign material, which, if detected, are removed by gentle traction.

The reticulum should be carefully explored for foreign bodies free in its lumen or embedded in its walls. Normally, the reticulum can be grasped and retracted into the antrum of the rumen. A reticulum that cannot be retracted in this manner is likely adhered to the diaphragm. An immobile reticulum with a fluctuant, round structure adhered to its cranial wall is characteristic of a perireticular abscess. These can be aspirated with a needle and syringe introduced into the rumen. Such abscesses can be lanced into the reticulum, using a scalpel blade inserted through the adhered reticular mucosa.

If available, fresh rumen fluid from a donor cow and/or a small amount of good-quality roughage is placed into the rumen prior to its closure. An inverting, single- or double-layered closure is performed using absorbable suture. Large pieces of ingesta are manually removed from the rumen wall and skin. The rumen wall and skin are lavaged copiously with saline. The author prefers to carefully apply surgical disinfectant soap to the skin surrounding the incision, taking care to avoid contact of the soap with the rumen wall. The soap is wiped away from the incisional edge with gauzes laden with clean water. A clean gown, sterile sleeves, and sterile gloves are donned, and the suture line connecting the rumen and skin is removed by cutting the suture in several places and removing it from the incision. The abdomen is then explored. The empty rumen facilitates movement of the surgeon's hand and arm ventrally and cranially to explore the right side of the cranial abdomen. Adhesions from perforating ulcer, abomasal impaction, and lesions of the pylorus may be palpated but cannot be visualized. By conducting rumenotomy first, potentially curable primary disorders can be identified and treated (e.g. foreign bodies in the reticuloomasal orifice hardware, perireticular abscesses).

Exploration of the abdomen after closure of the rumenotomy enables greater freedom of movement of the surgeon's arm, with less apparent discomfort to the animal. In those very rare cases where a second surgery is elected – for example, to drain a hepatic abscess – conducting the rumenotomy first reduces the rumen volume and weight, thereby lessening the compromise to respiration and venous return imparted by dorsal recumbency.

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HOW I TREAT WITH FLUIDS

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Introduction

There appears to be a diverse set of options for successful administration of oral and/or parenteral fluid therapy in cattle. In the author's experience, the logic of selecting fluids for treatment – specifically, the composition, volume, and route of administration – varies according to the setting (in clinic versus in field), size of animal, severity of the clinical problem, presumptive or documented causes of disease, and availability of ancillary diagnostic tests. In this seminar, the author's preferences and rationale for fluid therapy will be discussed. There have been excellent reviews of this topic published recently.[1-3]

Evaluating the calf for fluid therapy

Fluid therapy in calves is most typically instituted as a means to counter losses of water and electrolytes in undifferentiated calf diarrhea. Affected animals show variable degrees of dehydration, poor peripheral perfusion, obtunded demeanor, weakness, and ataxia. These signs are attributed to hypovolemia, metabolic (a.k.a. strong ion) acidosis, negative energy balance, hypoglycemia, electrolyte imbalances in the extracellular fluid (ECF), and D-lactic acidosis.[1] However, these clinical signs are not necessarily specific for the common biochemical consequences of undifferentiated diarrhea; shock from sepsis can produce similar signs.[2] When examining these animals, the author conducts a complete physical examination, remaining mindful of a helpful mnemonic for common causes of illness in calves: "SHADES." The author uses this as a guide to remembering common primary and secondary conditions in calves:

S: Sepsis, from a localized or disseminated site of infection. Common examples include omphalophlebitis, pneumonia, salmonellosis, and disseminated (bloodborne) sepsis. Disseminated bacterial infections occur most commonly secondary to partial or complete failure of passive transfer and/or translocation of gut bacteria across a compromised gastrointestinal mucosa.[4] The presence of distended scleral vessels, slow capillary refill time, and fever are supportive of this cause; however, rectal temperature appears to be highly variable in septic calves in shock, and the lack of fever does not necessarily rule out this problem.

H: Hypoglycemia. The author's practice is located at 5300-6100 feet (~1600-1850 m) above sea level in a climate wherein freezing nighttime temperatures can be encountered 7 months out of the year. Hypoglycemia is speculated to occur as a result of malabsorption of nutrients across the damaged intestinal lining, expenditure of energy in maintaining core body temperature, insufficient intake of milk or milk replacer during a bout of diarrhea, and maternal abandonment. Hypoglycemia appears to be more common in neonates under a week of age than in older calves. The clinical signs of hypoglycemia appear to overlap considerably with acidosis. In the author's experience, it is very difficult to accurately predict blood glucose concentration in patients with septic diseases.

When possible, the author prefers to use a portable glucometer for assessment of blood glucose concentration in calves that are under a week of age or hypothermic (rectal temperature less than 99.5° F / 37.5° C). If this is not available, the potential for hypoglycemia is addressed with warmed dextrose supplementation of parenteral fluids (described below) or administration of a warm, dextrose-containing oral electrolyte replacer (OER).

A: Acidosis. In scouring calves, metabolic acidosis occurs secondary to hypersecretion of base-rich fluids into the proximal intestine, generation of L-lactate by hypoxic host cells, and absorption of D-lactate from the gastrointestinal tract. D-lactate is generated by anaerobic gut bacterial catabolism of non-absorbed nutrients. Variable degrees of weakness, ataxia, diminished palpebral reflexes, loss of suckle reflex, and inability to stand may

be apparent. The degree of acidosis is estimated by the severity of CNS depression, ataxia, ability to stand, and in dairy calves, and strength of the suckle response, with correction for calf age.[6,7]

D: Dehydration. Although water and electrolyte loss in diarrhea is the most common cause of dehydration, it may also occur secondary to limited water intake from any primary disease. Insensible water losses can be significant when calves become ill in hot weather and shade is not provided.
During physical examination, the degree of enophthalmos and skin elasticity of the neck are used to predict the magnitude of dehydration.[5] This estimate is then utilized to predict the amount of fluid needed to restore normal hydration by the following formula:[2]

Fluid volume for restoration of hydration (L) = estimated dehydration % x bodyweight (kg)

E: Electrolyte abnormalities: As for dehydration and acidosis, diarrhea is the most common inciting event. Electrolyte abnormalities are difficult to accurately predict without laboratory tests. Common changes include variable hyponatremia if the calf loses sodium and water in diarrhea and is only offered water to replace those losses, hypernatremia if sustained water deprivation occurs or if oral electrolytes are improperly formulated and force-fed, and variable ECF potassium concentrations, with hyperkalemia appearing more common in acute diarrhea and hypokalemia being more commonly encountered in subacute to chronic diarrhea.[2] Hyperkalemia is typically responsive to correction of acidosis and hypoglycemia.[1]

S: Suffocation. The author's intent here is to consider birth asphyxia as a potential cause of compromise to the neonate. Typically, there is a history of dystocia and abnormal neonatal mental status that was appreciated soon after parturition. The author uses this final S as a reminder to consider *in utero* conditions that may result in weak - or stillborn - calves. Infectious and toxic causes of *in utero* fetal compromise are therefore brought into consideration; these are more strongly supported as differential diagnoses when multiple weak or stillborn calves are reported in the herd history.

Oral versus intravenous fluid therapy
The ultimate decision as to whether or not to begin fluid therapy with parenteral fluids or oral fluids is based on severity of clinical signs and the presence of bloat. Calves that are capable of standing, show mild depression, and have an intact suckle response are considered candidates for initiation of fluid therapy via OERs. However, oral fluids with dextrose are not used in calves with signs of bloat, because in theory, placement of soluble carbohydrates into the forestomach or abomasal lumen could augment gas production by luminal bacteria.[2] Calves that are unable to stand or severely obtunded are resuscitated with intravenous fluids, then transitioned to OER therapy as soon as is practical if they improve to the point of standing and suckling. Calves that do not respond as expected are re-evaluated for hydration status; if successfully rehydrated, the poor-responding calf is reassessed for sepsis and hypoxia (suffocation in the mnemonic above). Once rehydrated, poor-responding calves may also be candidates for non-steroidal anti-inflammatory drugs, administration of which may relieve visceral pain.

Milk or milk replacer feeding is continued after resuscitation, with a target intake of 10% of bodyweight per day split into as many feedings as is practical (usually 1-2). The exception to this rule is the calf with bloat or abdominal distension from ileus; smaller volumes are offered after a 12 hour fast. To avoid concerns regarding impairment of milk curd formation by certain OER products, a rule of "milk in the morning" (until noon) and "lytes at night" (3-4 liters of OER in the remaining workday) can be implemented. Supplemental heat and deep bedding should be provided. Free choice clean water and OER are provided to affected calves whenever possible.

Calves with evidence of failure of passive transfer or sepsis are assigned a guarded prognosis at best. If treatment is elected, treated with IV transfusion of whole blood (10-20 ml/kg) or plasma (20-40 ml /kg) and parenteral bactericidal antibiotics (ampicillin or trimethoprim-sulfamethazine). Cost concerns

on the part of the owner may prevent the administration of whole blood or plasma to all calves that might need it. Colostrum management (including colostrum hygiene), maternity pen/pasture hygiene, and pregnant cow (particularly heifer) body condition scores should be critiqued if this problem is endemic. Target prevalence of failure of passive transfer has been recently defined by Meganck and colleagues as > 10% prevalence;[1] lower prevalence targets may be proposed, based on case definition and tests used to document this condition.

OERs are considered more likely to correct underlying acidosis if they contain 50-80 mM/L of an alkalinizing agent, preferably acetate or bicarbonate. The former is converted to bicarbonate in the liver. Although hypertonic oral electrolyte replacement fluids are considered likely to be retained in the stomach for longer periods than isotonic fluids, the preferred hypertonic solutions have a higher energy content (due to inclusion of dextrose, acetate, and/or glycine). Bicarbonate in OERs will raise the pH of the abomasum, which could 1) impact milk curd formation and 2) enable survival of certain bacterial pathogens in the abomasal lumen.[2] Unlike bicarbonate, acetate does not impact abomasal pH nor slow abomasal emptying and may promote additional ECF volume expansion by a co-transport system linked to absorption of sodium.[1,2,8] That said, the two bases were determined to be equally effective in resuscitating calves in a prospective Korean study.[8] In the author's practice, although acetate- and bicarbonate-containing OER products appear effective, the disparity in price (commercial vendor) per 2 L of OERs can vary from 2-4- fold among products with dextrose and bicarbonate versus those with dextrose, acetate, and glycine ($1.25 USD vs. $5.05 USD, respectively). While this cost disparity may be relatively minor for the small herd owner, it may be worthy of consideration in large herds.

Intravenous fluid therapy is initiated according to the rules stated above. Base (bicarbonate) dosages in acidotic calves is estimated using Naylor's scoring system[6] and plugged into the following equation: Total dose of base needed = Estimated base deficit (mMol/l x 0.6 l/kg x calf bodyweight in kg. Slow IV infusion of that estimated amount of base is accomplished with hypertonic sodium bicarbonate (8.4%, 1 mM/L), followed by administration of 2-4 L of either warm water or a hypertonic OER diluted to half of the label directions. Dilution is justified by the goal of providing adequate free water for absorption from the gut. Subesquent OER treatments are administered as per label directions. Hypoglycemia is detected by glucometer; calves that are hypoglycemic are administered an IV bolus of 50% dextrose at the dosage of 100mg/kg (0.2ml/kg of 50% dextrose), Meganck and colleagues[1] recommend a dextrose dosage of 150 mg/kg.

Calves that are bloated or that are suspected of salmonellosis are preferably treated in the clinic because of the potential for the need for decompression and protracted illness, respectively. These calves are administered 1-3 liters of isotonic (1.3%) sodium bicarbonate via an IV catheter unless otherwise dictated by serum biochemistry tests. Further, these calves are transitioned to a IV balanced electrolyte solution for maintenance fluid therapy on a case-by-case basis.

Fluid therapy in mature cattle

Dehydration in mature cattle is evaluated on a similar basis as done for calves, although *Bos indicus* breeds may have inherently reduced skin elasticity as compared to *Bos taurus* breeds, and emaciated animals may have variable degrees of enophthalmos.[3] Oral rehydration therapy is considerably less expensive and time-consuming than IV drips of polyionic fluids in mature cattle. The latter is typically reserved for those rare cases of valuable stock with metabolic disorders such as hepatic lipidosis or acute diarrhea. A variety of oral rehydration fluids can be used in mature cattle; Roussel[2] recommends the following electrolytes to be added to 5 gallons (19 L) of clean water for administration orally: 140 g NaCl, 25 g KCl, and 10 g $CaCl_2$. In the author's clinic, dairy cattle with abomasal displacement are often given oral fluids by orogastric tube immediately after surgery; the components include propylene glycol – 0.5 ml/kg; calcium propionate – 1g/kg; and potassium chloride – 0.4 g/kg. Moderate dehydration and ketosis are treated with intravenous hypertonic saline and

intravenous dextrose, respectively, as described below.

In a large retrospective study of cattle undergoing fluid therapy at the veterinary clinic at Texas A&M University, dehydrated cattle with metabolic acidosis were those with ruminal acidosis and acute diarrhea.[9] Lactated Ringer's solution is a commonly used, commercially-available fluid that can be administered to correct metabolic acidosis. Isotonic (1.3%) or hypertonic (8.4%) sodium bicarbonate solutions can be used to more rapidly correct acidosis, as in calves. Administration of parenteral hypertonic electrolyte solutions is typically followed by administration of oral water at 20-60 ml/kg; ambulatory cattle may drink similar volumes voluntarily.[10]

The majority of dehydrated mature cattle have normal pH values or tend toward metabolic alkalosis.[9,11] These cattle typically require fluids with sodium and chloride concentrations that are greater than serum concentrations of these electrolytes, such as isotonic or hypertonic sodium chloride.[9] Compounded fluids may be needed if commercially-sold intravenous fluids are cost-prohibitive or in short supply. In the author's clinic, intravenous fluids can be compounded by addition of reagent-grade salts to 2.5 gallon (9.5 L) containers of commercially-available distilled water. To create isotonic saline, 85.2 grams of NaCl can be added to this volume of water. If potassium supplementation is elected, 3.5 grams of KCl can be added to this volume of saline to provide 5 mM/l of potassium ion. Although the rehydration volume is the target when isotonic fluids are administered IV to mature cattle, in reality, the ultimate volume that is administered is often dictated by time constraints (e.g. time from admission until discharge from the clinic, or time that can be spent on-farm monitoring fluid drips).

If a solution with alkalinizing capacity is desired, a different fluid recipe is used. Again, using reagent-grade salts that are mixed in a registered veterinary pharmacy, 60.5 grams of NaCl, 3.5 grams of KCl, and 38.8 g of sodium acetate are added to the 2.5 gallons of distilled water. The resulting fluid has these approximate concentrations of electrolytes: Na – 140 mM/L; K – 5 mM/L; Cl – 115 mM/L, and acetate – 30 mM/L. To avoid hemolysis from inadvertent IV administration of pure water, the empty packet of pre-made electrolytes can be taped to the exterior of the fluid container to display that the fluids were compounded as planned.

Dextrose (100 mg/kg) and calcium (0.5 – 1.0 ml/kg of a 23% solution of calcium borogluconate) are added to the intravenous fluids of cows with sustained negative energy balance and/or heavy lactation. Oral phosphorus supplementation (0.4 g/kg of sodium phosphate in ~20 L water) is initiated when sustained infusions of intravenous dextrose are administered.

In field settings, hypertonic saline, at a dosage of 4-5 ml/kg of 7.2% sodium chloride IV over 5-10 minutes, provides rapid and convenient volume expansion for hypovolemic cattle.[2,10] Oral water loading at 20-60 ml/kg should follow for cattle that do not drink voluntarily; 20 liters is a reasonable volume for an average-sized mature animal.[10]

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WHAT DOES ANTHELMINTIC RESISTANCE MEAN FOR WORM TREATMENT IN CATTLE?

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Introduction

Reports of Anthelmintic resistance in Australian cattle have grown since the mid 2000's.[1,2,3,4] With the aid of producers, veterinarians and independent researchers, from 2012-14, Merial undertook the largest national survey of anthelmintic resistance on Australian cattle properties to date.

Anthelmintic resistance in cattle nematodes

Worm Egg Count Reduction Tests (WECRT) were conducted on 36 properties throughout Australia to get an indication of the prevalence and severity of anthelmintic resistance in naturally acquired nematode infections in cattle.

Cattle were selected on the following criteria; young cattle (preferably weaners) aged 6-12 months, of a consistent line (age, size, weight, breed, sex, condition), with a similar grazing history and able to be maintained on similar paddocks for the duration of the test, and a targeted minimum pre-trial mean worm egg count (WEC) of >200epg, with the majority of animals having WEC's >100epg.

Where numbers and facilities allowed, cattle were randomly allocated into 5 treatment groups of 15 animals each; 1) abamectin/levamisole PO (abamectin 0.5mg/kg, levamisole 10mg/kg), 2) moxidectin PO (0.5mg/kg), 3) doramectin PO (0.5mg/kg), 4) eprinomectin PO (0.5mg/kg), and 5) untreated control group. Where facility constraints were a concern, injectable doramectin (0.2mg/kg) and injectable moxidectin (0.2mg/kg) could be used in place of PO products.

On day zero (0) cattle were individually weighed and treated according to group. Each pour-on treatment group were then separated for a minimum of 7 days to avoid cross contamination. On day 14 faecal samples were collected for individual WEC's and larval differentiation by group. Treatment efficacy was based on arithmetic means using Abbott's formula as follows: Efficacy (%) = 100 x (mean control group – mean treated group/mean control group).

An efficacy of <95% was used to identify the presence of resistance to a given treatment. Where larval differentiation results permitted, efficacy against the following nematode species was calculated; *Cooperia spp*. (n=32), *Ostertagia spp.* (n=23), *Trichostrongylus spp.* (n=9), *Haemonchus spp.* (n=21), *Oesophagostomum spp. (n=10), Bunostomum spp.* (n=6) and *Nematodirus spp.* (n=2).

75.0% (n=32) of properties had resistance in at least one species (*Ostertagia spp., Trichostrongylus spp., Haemonchus spp.* and *Cooperia spp*.) to at least one of the single active macrocyclic lactone (ML) treatment groups.

The prevalence of resistance in individual species to any of the single active ML's was; *Cooperia spp.* (n=32) 71.9%, *Ostertagia spp.* (n=23) 34.8%, *Haemonchus spp.* (n=21) 57.6%, *Trichostrongylus spp.* (n=9) 22.2% and *Oesophagostomum spp.* (n=10) 20.0%.

Of the 36 trial sites, 36, 30, 25, 17 and 36 included a treatment group for abamectin/levamisole PO, moxidectin, doramectin, eprinomectin and an untreated control group, respectively.

For all nematode species:

- 48.0% of properties tested had resistance to the doramectin treatment group (n=25), efficacy range 16.7 - 100%, mean 88.3%,
- 53.3% of properties tested had resistance to the moxidectin treatment group (n=30), efficacy range 30.7 - 100%, mean 88.7%,
- 41.2% of properties tested had resistance to the eprinomectin treatment group (n=17), efficacy range 56.8 - 100%, mean 90.7%, and
- 2.8% of properties tested had resistance to the abamectin/levamisole PO treatment group (n=36), efficacy range 94.7 - 100%, mean 99.5%.

What does anthelmintic resistance mean for worm treatment in cattle?

Although not a large data set, the overall findings were consistent with previous work

done looking at the prevalence of resistance in Australian cattle nematodes. Cotter *et al* (2015) identified resistance in either *Cooperia oncophora* or *Ostertagia ostertagi* to ivermectin on 89.5% (n=19) of properties tested[1]. Cotter *et al* (2015) also looked at the efficacy of fenbendazole and levamisole and identified resistance in *Ostertagia ostertagi* on 50.0% (n=16) and 67.0% (n=15) of properties, respectively[1].

Rendell (2010) identified resistance in either *Cooperia spp.* or *Ostertagia ostertagi* to ivermectin on 53.8% (n=13) of properties tested. Resistance to ivermectin in *Trichostrongylus spp.* was also identified on one property. Resistance to benzimidazole (BZ) was seen in 45.5%, 57.1%, and 9.1% in *Ostertagia ostertagi* (n=11)*, Trichostrongylus spp.* (n=7) and *Cooperia spp.* (n=11), respectively. While 100.0% of properties tested had resistance in *Ostertagia ostertagi* (n=3) to levamisole.

Throughout Merial's study, on properties where ML resistance was identified in *Cooperia spp.* and all three single active ML's were tested (n=10), eprinomectin maintained a higher arithmetic mean efficacy against *Cooperia spp.* (85.9%) compared to moxidectin (78.9%) and doramectin (69.1%). Although a small data set, the result was suggestive of varying potency between the single active ML's.

Worsening resistance in cattle nematodes will have several implications for the future treatment of cattle.

As, or ideally before resistance worsens to single active drenches, greater emphasis should be placed on the use of combination products to increase their efficacy and delay the onset of resistance to their individual actives[5]. The use of combination broad-spectrum active products is well understood and accepted in the management of sheep nematodes but currently less so in that of cattle.

This is of particular importance where *Ostertagia ostertagi* is found in significant numbers. The innate variable efficacy of benzimidazoles[6] and levamisole[7] against inhibited L4 *Ostertagia ostertagi* should prompt producers to move to combination products prior to the failure of ML's.

The addition of levamisole, with its superior efficacy against BZ/ML resistant *Cooperia spp.*[8], in combination with a ML, not only increases the resulting efficacy of the drench but will prolong the lifespan of ML's against *Cooperia spp.*, the recognised dose limiting species for ML's.

Failure of drench classes on properties will lead to downward pressure on productivity gains. This will place additional pressure on intensive production systems not only in lost production, but also secondary to the necessary adoption of integrated pasture management practise used to success in sheep production systems; resting and rotating paddocks, reduced stocking rates and genetic selection away from productivity traits towards inherited resistance.

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LAPAROSCOPIC ABOMASOPEXY FOR LDA CORRECTION USING JANOWITZ TWO STEP METHOD

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Introduction

Left displaced abomasum or LDA is a common occurrence in high producing dairy cows in Australia. Cows will usually present in the first month of lactation and commonly present because they are off their milk. Percussion of the left hand side of the cow over the ribs will normally create a high pitched ping. The presence of an abomasum on the left hand side can be determined by sampling the area of the gas cap with an 18g 1.5" needle and confirmation of a low pH.

There are several methods for the correction of an LDA however recently in Europe a less invasive technique involving laparoscope has used more routinely. With practice this method can be completed successfully without assistance although it is easier if there is an extra set of hands. This presentation aims to describe the two-step method of laparoscopic LDA correction.

Instruments Required

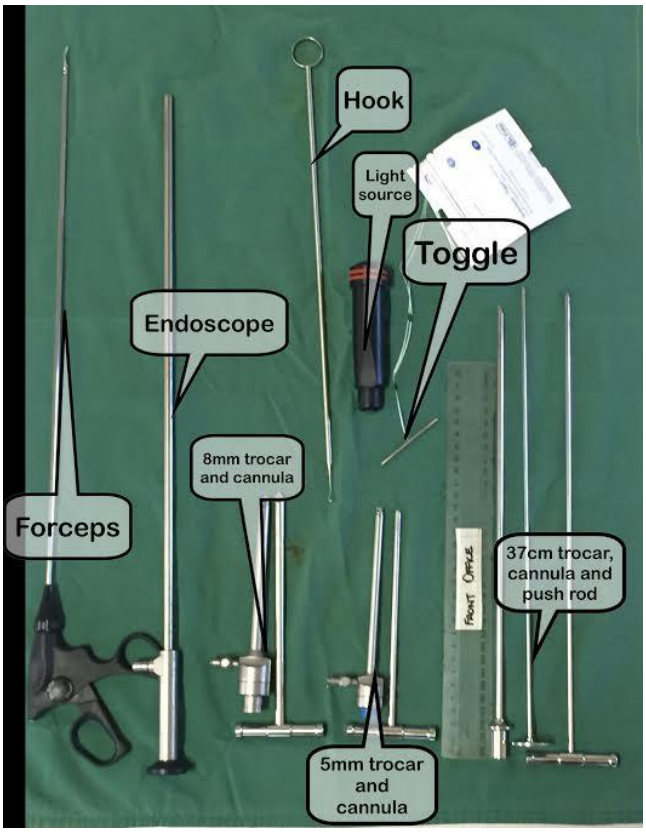

Image 1: Instruments required for laparascopic LDA correction.

Incisions for Cannulas

Two sites need to be prepared for the cannulas. The 8mm cannula is placed first in the paralumber fossa just behind the last rib. The sites can be scrubbed, blebs of lignocaine are placed at each site that is going to be incised. A stab incision is made at each site and a pneumoperitoneum is created by allowing air to flow in through the 8mm cannula (scope cannula). The 5mm cannula (instrument cannula) is then placed just cranial to the last rib.

Image 2: cannula placement for laparoscopic LDA

Toggle Placement

The endoscope is placed though the 8mm cannula so that the abomasum can be identified. Once identified the long cannula and trocar (37cm long) are placed in the 5mm cannula and the abomasum is penetrated at the top of the abomasum near the greater curvature. A short sharp push seems to be most effective. The trocar is removed and a specialised laparoscopic toggle is passed through the long trocar and pushed into the abomasum with the push rod. If you are uncertain that you have penetrated the abomasum a small feeding tube can be passed down to the cannula and fluid can be withdrawn using a syringe the pH can be measured to confirm abomasal fluid. Be careful if you do, as

delaying the placing of the toggle will cause the abomasum to deflate making the process more difficult or not possible.

The literature says that at this stage the suture material attached to the toggle is to be pushed into the abdominal cavity. I have found leaving the suture material outside the cow at this stage, is a better technique. On some occasions I have extended the length of the suture material so that once the cow has been rolled the suture material will remain accessible until pulled through from the ventral incision. I have also found it easier to retrieve once the cow has been rolled and to see if the abomasum is moving during the rolling process.

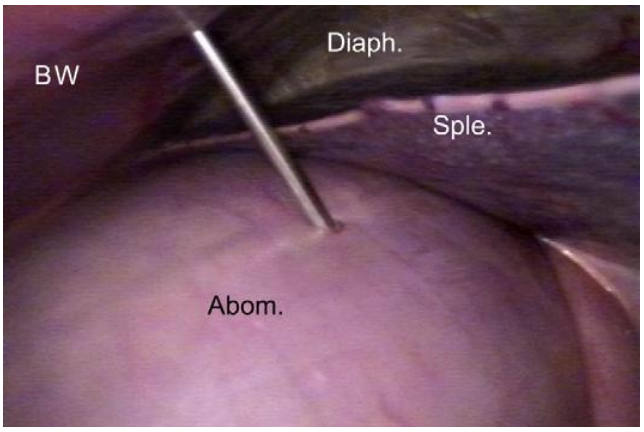

Image 3: Placement position of the toggle.[1]

Casting

The next step requires the cow to be placed into right lateral recumbency. The method of casting is of personal preference but I have found that the two half hitch method works for me with the half hitches on the left hand side. They seem to fall with right side down the majority of times. The cow is sedated with 1.5mL xylazine (20mg/mL) and 1.5mL ketamine (100mg/mL). The back legs are tied up to a nearby rail stopping just prior to half way. The front legs are also tied to a post just short of half way.

Toggle Retrieval

Again two sites need to be prepared for the cannula. The 8mm cannula goes just to the cow's right hand side of the umbilicus, this is the cannula for the scope. Be careful when placing this cannula not to hit the milk vein. The 5mm cannula is to be placed about 10cm caudal to the xiphoid and just to the right of midline. This is your instrument cannula.

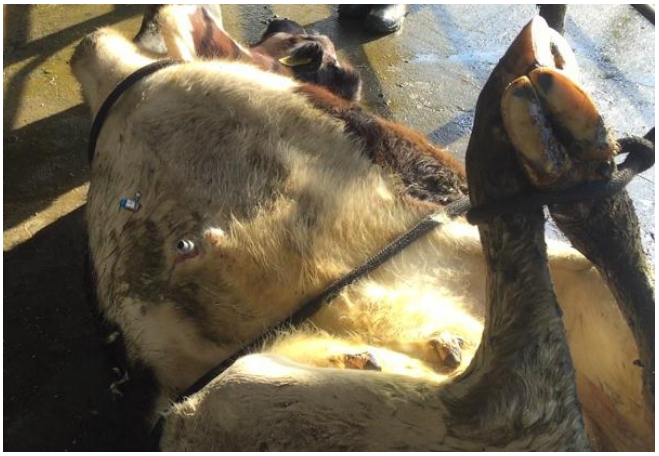

Image 4: cannula placement for toggle retrieval.

Using the endoscope identify the suture material from the toggle. It is usually sitting back towards the left hand side of the cow and in line with the umbilicus. Once identified pass the forceps through the 5mm cannula. To find the forceps on your scope I find it easier if you slide the forceps down the scope until they drop off the end of the scope. Pick up the suture material of the toggle and pull forceps and cannula all in one movement so as not to cut the suture material on the cannula. This technique requires practice and completion times will shorten after completing a few toggles.

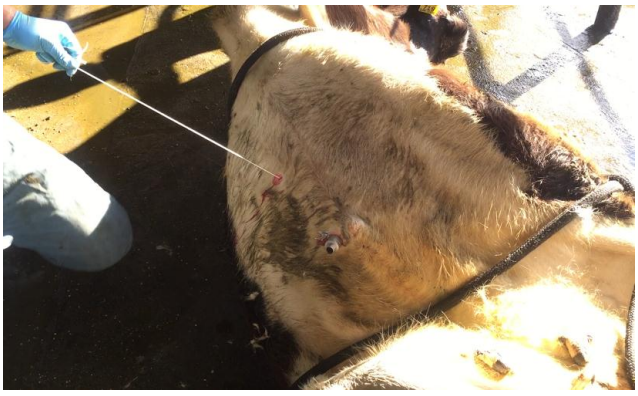

Image 5: retrieval of the toggle.

Once the suture material has been pulled through cut the loop and tie the suture material level with the black line on the suture material. I use a small plastic "surfboard" with holes in it to tie off onto. Complete the roll and if you think that the cow requires antibiotics then consider the use of erythromycin as it also has the side effect of increasing gastric emptying time.

Lessons Learnt

- Don't leave the cannula sitting in the abomasum before placing the toggle. The abomasum will deflate making life a lot more difficult.

- If you miss the abomasum on the toggle you can always make another incision for your instrument cannula (5mm) and pull the toggle back out through the new incision

- If you hit the rumen when you first place the 8mm cannula behind the last rib. Pull out and try again it doesn't seem to be too much of a problem for the cow.

- Be careful not to over roll the cow this seems to make life fairly difficult and initially this was the reason I started adding a bit of an extension to the toggle suture material so I could always retrieve it.

- If you run out of power in your light while toggling you can use the light off your phone to complete the job.

- If you find adhesions usually you will end up doing surgery but consider the use of a surgical toggle as the toggle is already placed.

Surgical Toggle

I have completed a few of these now and they seem to work pretty well. In LDA's with adhesions the toggle may already be in place in the abomasum. It is simply a matter of making a small incision through the left paralumbar fossa. Adhesions can be broken down, abomasum deflated and pushed over to the right hand side of the cow. The suture material from the toggle can be placed on a large needle and be pushed though the ventral wall of the abdomen. You should be aiming for a spot about 10cm caudal of the xiphoid and slightly to the right hand side of the cow. Be carefully not to hit the milk vein! I have also used a similar technique for the correction of RDA's the incision for this is made in the right paralumber fossa and the toggle is tied just caudal to the xiphoid as above.

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FIRST ATTEMPT AT THE FREEZING OF BULL SEMEN STRAWS: A CASE STUDY

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Abstract
The process of collecting and freezing semen is a daunting undertaking for a recent graduate or inexperienced veterinarian. This case study aims to demonstrate the authors initial experiences in this field and reiterate that the process is not out of reach for the inexperienced mixed practitioner. The presentation gives a brief overview of the authors' methodology for the collection of a 3 yr old Aberdeen Angus bull and subsequent straw manufacture. Possible variations to the protocol are then discussed based on the absence of key equipment such as a; photometer, CASA system, automatic straw freezing machine, cold handling cabinet, and a straw filling and sealing machine.

Introduction
Artificial insemination is a reproductive tool that has led to significant advances in genetic improvement in the Australian beef and dairy industries. During routine Veterinary Bull Breeding Soundness Exam (VBBSE) visits, veterinarians are approached by beef cattle producers to freeze semen straws from their commercial sale bulls for sale or storage as an insurance policy in the case of loss of fertility. This is the case study of a recent (3 yr) inexperienced graduates' first attempt at freezing straws. The aim of this paper is to introduce the process of semen straw freezing in a mixed practice setting to those with little experience in the field. The various methods that may be employed to get to a successful outcome with equipment limitations are explored.

Cryopreservation theory
Cryopreservation is the preservation of cells at sub-zero temperatures by allowing movement of water and cryprotectant in and out of the cell without the formation of intracellular ice. [1,2] Intracellular ice formation leads to irreversible membrane damage which impacts cell survival and function.[3] The finer details regarding the changes that occur within the cell during cryopreservation has been reviewed in the literature and will not be discussed here. [4,5,6] Fundamentally, the veterinarians aim is to get the spermatozoa to the dehydrated storage state and back again with the least amount of damage. There are two inherent dilemmas within this aim: firstly some degree of damage is inevitable (approximately 50% loss is expected) and secondarily damage caused in one step in the freezing process will not be evident until the final stages when the semen is assessed, long after anything can be done to mitigate it. [3,4,9,10,11] The key to success is to be well prepared and follow the freezing protocol closely; timing is everything.

Case background
A three yr old Aberdeen Angus bull was presented for semen collection and freezing. The bull had been purchased 6 mo ago as a commercial stud sire. The purpose of semen freezing was to produce three hundred 0.5ml straws for commercial sale and to store semen as an insurance policy against loss of use. The bull had been tested 2 mo ago (VBBSE) and passed physical, semen motility and morphological assessments.

Preparation
The commercially pre-labelled straws were ordered 2 wks prior to the scheduled collection. The following information was included on the straws based on ICAR codes; date of manufacture, centre identification, centre licensing status, animal identification and breed code. [7]
The day before the collection the extender (Andromed®) was prepared according to manufacturer recommendation. Andromed is egg yolk free and thus suitable for the export market if required.
The following calculation was completed to determine the volume of extender required:

- 300 x 0.5ml straws = 150ml of extended semen
- Volume of the ejaculate assumed to be 10ml
- extended semen volume (150ml) – ejaculate volume (10ml)
  = 140ml of extender is needed (min volume).

The extender was diluted using HiClone® cell culture water in 80ml sterile containers. Both extender and water were warmed to 35° C using a water bath. The cell culture water (80ml) was slowly added to the extender (20ml). This was then repeated to give a total volume of 200ml extender. The diluted extender was then cooled to 4°C and stored.

Collection technique

The bull was identified and safely secured in the crush. A sterile disposable mare artificial insemination pipette was introduced into the prepuce and the prepuce was flushed with 200ml warmed sterile saline. The bull was collected using electroejaculation following 1-2 minutes of manual ampullae massage. The semen was collected with warm, dry and clean collection cones sequentially into 6 warmed sterile 10ml falcon tubes. This was done to attempt sequential fractioning of the ejaculate which allowed categorizing into good, average and poor quality of semen and allowed for sample isolation in the possible event of urination during ejaculation. In order to extend the lifespan of the spermatozoa work was rapid and efficient, from the time of collection to the addition of extender to the semen. Two collections were completed 30 min apart in order to get the required volume of semen.

Initial semen processing

The semen was assessed crush side at 37°C using phase microscopy and a warmed stage. Semen and extender was maintained at 37°C using a water bath. A sample of the raw semen sample was placed in a vial (4.95ml phosphate buffered saline/formalin with 0.05ml semen equating to a ratio of 1:100) for concentration and morphological assessment at the laboratory. The volume of each sample was recorded. Each sample was subjectively assessed for initial gross motility and concentration.

Table 1. Gross assessment of semen concentration

Grading	**Visual appearance**	**Concentration** (x 10^6 sperm/ml)
12	Thick, creamy and grainy	1200
11		1100
10	Whole milk	1000
9		900
8	Skim milk	800
7		700
6	Translucent	600
5 and below		500 and under

A 3mm diameter drop of semen was placed onto a warmed (37°C) new glass slide and covered with a warmed cover slip. The sample was observed under phase contrast microscopy at 400 x magnification and the percentage of sperm cells having progressive forward linear movement was assessed. Based on this subjective assessment the motility of the raw semen varied from between 0 and 90%. Concentration was assessed visually using the grading system outlined in Table 1. The concentration of the samples varied between grade 7 and grade 10 (i.e. 700 x 10^6 sperm/ ml and 1000x 10^6 sperm/ ml). The sample with 0% motility was contaminated with urine during ejaculation and was discarded. The volumes of each portion varied between 2 and 5 ml. Only vials with motility > 70% were used. The suitable vials were extended 1:1 with the warmed diluted Andromed®. The warmed extender was added to each vial slowly at a ratio of 1:1; extender added to semen using disposable pipettes along the side of the tube and then swirled gently to mix. Motility was assessed on each sample. The semen motility was found to have improved following the addition of extender. The process was repeated, until a ratio of 1:3 (semen : extender) was reached. Samples were discarded if at any point the motility of the sample dropped below acceptable levels (i.e. less than 70%). The partially extended semen vials were combined into a 120ml warmed sterile container, a final assessment was completed and the partially extended semen was left at room temperature for 20 min to begin the cooling process. The semen was assessed to ensure that motility had been maintained. This process was repeated with the second semen collection, which yielded 3 vials of 90% motility with volumes of 2, 4 and 8ml. The final total volume of the combined partially extended (1:3) semen was 100ml.

Semen cooling

Following 20 min at room temperature, the semen vials were wrapped in cotton wool and transferred to a foam box containing an ice brick. The semen was transported to the clinic and the box was placed into the refrigerator. This method ensured that the semen cooled slowly from room temperature to 4°C in a minimum of 2 hrs. The semen was kept in the foam box at 4°C overnight (approximately 12 hours) to equilibrate.[7] The straw filling equipment and consumables were set up in the cool room to reach a stable 4°C overnight.

Calculation of number of doses and dilution
In order to calculate the amount of extender to add to the pre-extended semen the concentration and morphology of the semen had to be determined. This was completed on the concentration vial prepared at collection (1:100), using a haemocytometer with a Neubauer chamber.
It was calculated that 511 straws were able to be made based on a conservative motile sperm dose (MSD) of 20 x 10^6 sperm/ straw (Figure 1). [8] The volume of extender required was calculated based on 320 x 0.5ml straws and the current semen: extender dilution of 3:1.

1. Number of sperm in ejaculate:
   Volume ejaculate (ml) x conc. spermatozoa (x 10^6 /ml) = total number sperm

   25ml x 500 = 12.5 billion sperm (12 500 x 10^6)

2. Number of PMMN (progressively motile morphologically normal) sperm
   Number of sperm x motility % x morphology %= total number PMMN sperm

   12.5 x 0.90 x 0.91= 10.23 billion PMMN sperm total

3. Calculate number of doses and dilution
   Total PMMN sperm (x 10^6)/ MSD (20 x 10^6 PMMN/straw) = # straws

   $$\frac{10\ 230 \text{ million PMMN}}{511 \text{ straws}} = 20 \text{ million PMMN/ straw}$$

- Aim to make 300 straws, dilute semen to allow for 320 straws for breakages/ filling issues.
  - 320 x 0.5ml straws = 160ml total volume.
  - Current volume 100ml (1:3 extended)
  - 160ml- 100ml = 60ml extender added

Figure 1: Calculations to determine number of doses and dilution required.

Straw filling
A Minitube® semi-automatic filling and sealing machine (SFS 133) was used to fill the straws. Glass sealing balls and pre-printed medium 0.5ml straws were used. All equipment was kept in the coolroom at 4ºC for the 12 hr prior to and during the procedure. The empty straws were packed into cartridges the night before to ensure they were adequately chilled. The semen was diluted to the final required volume of 160ml with the addition of 60ml chilled extender. Straws were then filled and sealed with glass sealing balls. The straws were examined to ensure that they had filled and sealed adequately and to ensure that the 1cm air bubble in the straw was in the middle of the straw. This is to prevent expansion during the freezing process resulting in bursting of the straw. The filled straws were packed onto freezing racks and left to stand for 20 mins at 4ºC.

Straw Freezing
The following freezing protocol was followed; 1: +4ºC to -120ºC Liquid Nitrogen (LN) vapour 12 mins, 2: -120ºC to -196ºC submersed in LN, 3: packed into goblets whilst submersed and stored in LN. A long foam box was filled with 10cm LN. The chilled filled straws were suspended 4cm above the LN, using a freezing rack and two metal blocks at each end of the box. The LN was fanned using the box lid. The lid was quickly placed onto the box sealing the vapour in. The straws were left in LN vapour for 12 min. The straws were carefully taken out of the freezing rack and submersed in 15cm of LN within a smaller foam box. The straws, now at -196ºC, were packed into goblets for storage whilst submersed in LN. The goblets were moved into the storage LN tank. A total of 310 semen straws were successfully processed.

Post-Freezing Assessment
The semen was thawed and assessed 24 hours and 7 days following freezing. The post-thaw motility was 50% at both assessments which is considered satisfactory. [,9,10,11]

Discussion
There are many possible variations to the standard semen freezing protocol that may be employed to reach a successful outcome when equipment is limited. Whilst specialised semen freezing equipment is excellent for improving accuracy and streamlining the semen freezing process, this equipment is beyond the reach of many mixed practitioners. The following variations are discussed based on the absence of equipment including: photometry, CASA system, automatic straw freezing machine, cold handling cabinet, and straw filling and sealing machine.
A photometer is used to objectively assess semen concentration by analysing ejaculate turbidity. Subjective determination of semen concentration without a photometer may be achieved using the visual grading system demonstrated in this case (Table 1). Sperm concentration may be assessed objectively using a haemocytometer with a Neubauer chamber as outlined previously.

A computer assisted semen analyser (CASA) system is used to objectively measure semen motility (velocity and type of movement), concentration, morphology (based on sperm shape only) and vitality (live/dead sperm count). [12,13] With operator experience and the correct technique, as described above and in the literature, subjective motility assessment can correlate with CASA. [14] A veterinary semen morphologist can provide a more descriptive assessment of morphology to include important vacuolations and some acrosome defects.
In the absence of a cold handling cabinet, transportable coolrooms are readily available for hire or may be available from clients.
Filling and sealing semen straws manually, although time consuming, is a method just as valid as using an automatic filling and sealing machine. A micropipette tip is placed onto the end of a 5ml syringe and is attached to the straw. The semen is drawn up into the straw using the syringe ensuring that approximately 0.05ml of air is left in the straw to allow for expansion during freezing. The straw is pushed onto a sealing ball using the syringe to hold the straw firmly, ensuring not to minimise touching the straw with the fingers.
Automated straw freezing machines aim to provide a uniform freezing pattern whilst consuming the least amount of LN, requiring less labour and producing more straws/hr than conventional manual methods. The requirement for such technology can be bypassed using tried and practised modified equipment. As discussed in the case study, a large foam box was altered in order to hold a straw freezing rack exactly 4cm above LN. If a freezing rack is not available, a substitute "boat" can be made using small foam blocks as "floaters" and the teeth of an inverted jigsaw blade as semen straw holders. With careful measurement, the straws can then be placed on the jigsaw blade, floating 4cm on top of the LN. The same box can also be used for the next freezing step: i.e. rapid submersion into LN.

Summary

Semen cryopreservation is a tool to aid in genetic improvement. This paper outlined that although not without challenges, bull semen freezing is a readily achievable skill for any new graduate or mixed practitioner with a background in VBBSE. This case is an example of what a new graduate can achieve with some VBBSE equipment and mentorship. The described technique was adapted from various sources and literature. The key to success is to be well prepared and follow the freezing protocol closely; timing is everything.

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