



# **Neonatal calf diarrhoea in suckler beef herds**





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# contents



Background	1
Prevention of a NCD outbreak	2
Investigation of a NCD outbreak	8
Control of a NCD outbreak	17
Treatment of the scouring beef calf	19

## List of abbreviations

AEEC	Attaching and effacing <i>E. coli</i>	IV	Intravenous
BJD	Bovine Johne's disease	GGT	Gamma-glutamyl transferase
BVD	Bovine viral diarrhoea	NCD	Neonatal calf diarrhoea
ELISA	Enzyme linked immunosorbent	NVD	National vendor declaration
ETEC	Enterotoxigenic <i>E. coli</i>	PCR	Polymerase chain reaction
FPT	Failure of passive transfer	STEC	Shiga toxin-producing <i>E. coli</i>
IgG	Immunoglobulin G	TMS	Trimethoprim sulfonamide

## Background

Neonatal calf diarrhoea (NCD) occurs mainly in calves less than six weeks of age, although calves up to four months of age may be affected. Outbreaks of NCD can be costly for beef producers as treatment of affected calves is time consuming, and the death of a calf represents the loss of profit from that cow–calf unit for the year. Additionally for survivors there may be impacts on subsequent calf growth rate and weaning weight, loss of genetic potential and a decreased capacity to improve and maintain the herd. A 2003 study of Australian suckler beef herds demonstrated that the cost of NCD ranged from \$0.50 to \$68.60 per breeding cow.

NCD results from a combination of an adverse environment, poor host immunity and challenge from infectious agents. Although a very serious impairment in any one of these factors can lead to disease, NCD outbreaks usually reflect a problem in all three areas. Consequently it is important to address the problem at a herd level by assessing risk factors and implementing preventive management.

Rotavirus and cryptosporidia are the most common pathogens associated with NCD in suckler beef herds in southern Australia. The other major pathogens are enterotoxigenic *E. coli* (ETEC), Salmonella and coronavirus, and less common pathogens include coccidia, yersinia and clostridia. Intestinal nematodes are not addressed by this document as they only cause disease in calves less than six weeks old when there is severe challenge. Farmers and their employees should be informed of the zoonotic potential of some calf scour pathogens and advised on appropriate precautions.

### Rotavirus and coronavirus

These viruses are both transmitted faeco-orally and coronavirus is also spread via the respiratory tract. Both are shed intermittently by adult cows, with increased shedding around the time of calving. Many calves are sub-clinically infected and will shed virus in their faeces. Recovered calves can be reinfected.

Rotaviruses can survive in fresh water for more than two weeks at 23°C and for months in water or soil <5°C. They are stable in faeces and effluent for up to nine months and can remain in calving areas from

year to year. Bovine rotavirus is transmitted by cats, dogs and feral animals (deer, pigs, foxes and rabbits). Coronaviruses are more fragile.

The incubation period is short with clinical signs occurring one to three days after transmission. Consequently maternal shedding and poor colostrum transfer can lead to clinical disease in calves less than four days of age.

### Protozoal infections

Protozoa are transmitted faeco-orally. While cryptosporidia are most likely to be associated with calves less than six weeks of age, coccidiosis should be considered in calves over three weeks old when the environment is poor. Giardia has been associated with NCD in poor conditions, but is more commonly a secondary pathogen.

Adult cows are a potential source of all protozoal infections, however emphasis should be placed on providing good quality water, a clean environment and minimising access to other species, including wildlife. Outbreaks of coccidiosis are associated with stress, especially post weaning, and in dry conditions when cattle are fed off the ground and congregate around watering points. Chronically leaking troughs can create a favourable environment for coccidia to survive. Reduced stocking density and improved hygiene will minimise spread.

Coccidial and cryptosporidial oocysts can survive for months or years in water and soil in cool conditions, however drying will dramatically reduce their viability and infectivity. The prepatent period is three to six days for cryptosporidia and 15–20 days for coccidia. Cryptosporidia sporulate within the intestine and immediately infect adjacent cells, consequently calves may improve clinically and then relapse.

Calves one week to four months of age can shed significant numbers of cryptosporidial oocysts. Peak shedding occurs between one and three weeks of age. The number of oocysts in water/environment can be minimised by preventing access of this age group to watercourses. Viable oocysts can also be found in run-off water resulting in contamination of watercourses.

Cryptosporidia infections are more common when there are other animal species on the property.



## Salmonella

Salmonella is primarily transmitted faeco-orally but may also be shed in saliva and nasal secretions. It may be introduced onto a property by contaminated feed or water, infected livestock or other animals and birds. The bacteria can survive in the environment for several years. There is increased shedding and risk of clinical disease in periparturient cows.

Salmonellosis is often associated with recent stock introductions or stressors such as drought, droving or rapid dietary change. The incubation time is dose dependant and is usually 24–48 hours. Disease may be observed in two-day-old calves, but is most common in those four days and older.

## E. coli

*E. coli* are normal flora of the bovine gastrointestinal tract. They survive in soil for more than six months and in water for at least three months.

Enterotoxigenic *E. coli* (K99+) is the most common strain causing NCD. Calves are only susceptible to ETEC during the first 14 days of life and are most at risk in the first three days. Therefore emphasis should be directed on minimising exposure to faeces during this time. Adult cows shed the bacteria, but there is no documented evidence of an increase associated with calving. Some properties have recurrent ETEC problems and vaccination should be considered in this situation.

Attaching and effacing *E. coli* (AEEC) and shiga toxin-producing *E. coli* (STEC) can be pathogenic in calves, causing disease up to four weeks of age. Most diagnostic laboratories do not routinely screen for AEEC and STEC and a diagnosis should be supported by compatible histopathology as healthy calves may shed these strains.

### Key points to consider

- Healthy calves are often subclinically infected with enteric pathogens and amplify environmental contamination.
- All major enteric pathogens are commonly carried by asymptomatic adult cows and shedding of many pathogens species tends to increase around calving.

- All major enteric pathogens can survive in the environment and water sources for weeks to months (and in most cases over a year) in cool, damp conditions.
- Many other animals, birds and flies are potential vectors of cryptosporidium and Salmonella.
- Feral animals and domestic pets are a potential reservoir of rotavirus.
- Calves over six months of age, yearlings, dry cows and sheep post-weaning, present minimal risk to young calves. Strategic grazing of paddocks with these animals prior to use by young calves can be part of a preventive strategy to lower environmental contamination.

## Prevention of a NCD outbreak

NCD can be difficult to control and producers should implement management strategies to minimise the risk of an outbreak. These management strategies will also promote general calf health and reduce the risk of transmission of diseases such as bovine Johne's disease (BJD). Potential losses and the cost benefit of control measures should be considered when advising producers on management changes. Significant planning by the producer and integration into an annual management plan will be required.

Although management strategies can be applied generically without a diagnosis, it is advisable to have a thorough knowledge of the property and its NCD history. Producer compliance is often better when preventive efforts are directed at a tangible pathogen rather than an abstract 'bug'. Identification of specific pathogens will also allow targeted interventions such as vaccination. A risk assessment of current management practices should highlight weaknesses in the system. Heifers' calves are most susceptible to NCD and on many properties preventive management may only need to be applied to this group. Heifers have poorer mothering ability, lower colostrum quality and an increased risk of dystocia. Pregnant heifers are often grazed at a higher stocking rate to improve observation, exposing their calves to a greater environmental pathogen load.

## Principles of prevention

The susceptibility of a herd to NCD can be reduced by **minimising exposure of susceptible calves to pathogens** and by **promoting calf immunity**.

These principles can be encompassed in three management procedures:

1. Minimise contact between young calves and potential reservoirs of infection
2. Maximise colostrum intake, calf welfare and nutrition in the first six weeks of life
3. Prevent the introduction of new NCD pathogens into calving herds

### 1. Minimise contact between young calves and potential reservoirs of infection

#### Confidence: moderate

Recommendations are based on sound epidemiological principles, plus research and advice from pasture based industries, but have not been evaluated in Australian pasture based systems.

To minimise exposure of calves to enteric pathogens in a moderate to intensively farmed situation, it is necessary to minimise calf age range in mobs of cows with calves less than six weeks old, and to change paddocks when there is a build-up of manure or mud. This requires careful long-term planning to determine optimum patterns of paddock use and may require creation of temporary paddocks with electric fences. In a more extensive situation many of these suggestions may not be practical. The benefit of the strategies will also depend on the causative pathogen(s) on the property.

The following management strategies should be recommended to producers:

- **Target a limited joining (eg 60 days)**

- A short joining period may lower pathogen levels in calving paddocks and limit the age range of calves.

- The key goal of management is to maximise the total liveweight of calves weaned.

- **Select calving and nursery paddocks 12 months in advance**

- Use different paddocks from year to year.
- Do not use paddocks in which NCD has occurred in the previous 12 months.
- Spell (cut for hay or silage) calving and nursery paddocks for at least three months before use or graze with low-risk stock (calves less than six months old, yearlings, dry cows or sheep).
- Calve heifers and cows in separate paddocks.
- Plan a rotational strategy with multiple calving paddocks. Do not leave calving cows in one paddock for more than three weeks. Move more frequently if paddocks are wet and boggy. If there is no alternative in wet conditions, decrease stocking rate.

- **Minimise exposure of newborn calves to sources of infection**

- Drift off cows and newborn calves within 24 hours of birth into a separate 'nursery group'.
- Start a new nursery group every three weeks.
- Do not merge groups until the youngest calf is six weeks old.
- Provide clean water, preferably in troughs.

- **Avoid manure build-up**

- Move supplementary feeding sites around paddock or use bale feeders or troughs.
- Do not put cows into calving paddocks more than two weeks before the start of calving.
- Provide gravel or woodchips around water troughs if wet and muddy and apply 1kg lime/m<sup>2</sup> every week. Fix leaking troughs.
- Use multiple watering points to spread cattle in extensive country.

## 2. Maximise colostrum intake, calf welfare and nutrition in the first six weeks of life

### Confidence: high

The role of adequate colostrum intake in disease prevention in neonatal calves, and the increased rate of FPT associated with dystocia, is well established.

#### • Ensure calves receive adequate colostrum

Beef breeds often produce a low volume of colostrum with a high concentration of immunoglobulins. Calves should suckle at least 5% of bodyweight of colostrum in the first 12 hours and 10% of bodyweight by 24 hours. Adequate passive transfer is dependent on four factors:

- i The vigour of the calf
- ii The amount of colostrum available
- iii The maternal behaviour of the dam
- iv The conformation of the dam's udder

Dystocia is the most common cause of failure of passive transfer (FPT). Therefore, it is important to manage cow condition score, heifer liveweight and stature, and sire selection, and to supervise heifer and twin calvings to maximise the number of live calves born.

If dystocia rates exceed 10% in heifers and 2% in cows:

- Address the underlying reasons, as detailed in *More Beef from Pastures: The producer's manual*, and correct for the next calving season.
- Ensure calving areas permit regular checking of calving cows and easy movement if yarding is needed.
- Supplement assisted calves with colostrum by stripping the cow at calving and administering 1L/20kg with a tube feeder.

- Supplement all calves that have not suckled within six hours of birth.

Optimise pre-calving nutrition to ensure an adequate volume of colostrum. Poor nutrition has minimal effect on total immunoglobulin produced, but will decrease volume, resulting in high viscosity colostrum that is difficult for calves to suck. Early bonding between the cow and calf will maximise the chance of sufficient colostrum intake. Cattle should be selected and handled to facilitate early attachment. Calves from heifers are most at risk of abandonment. Reduce this risk by handling replacement heifer calves at weaning and use management procedures at calving that are appropriate to the temperament of the heifer group. It is possible for producers to interfere too much and cause problems. This may be a staff training issue.

Cows with poor conformation (pendulous abdomens or udders or large 'bottle' teats) should be preferentially culled.

*Dystocia is the most common reason for FPT in beef calves. Where dystocia rates are higher than acceptable, assisted calves should be supplemented with colostrum at the time of calving.*

### Travelling stock

When travelling, calving cows may be yarded at high relative stocking rates, which may increase the risk of mismothering and transit, nutritional and handling stress. Drovers should be encouraged to:

- set up a nursery and separate calving cows
- tube feed all new born calves with stored colostrum (1L/20kg)
- move calved cows to a fixed paddock



## Vaccination

### Confidence: moderate

The role of vaccination to prevent ETEC infection is well established, but studies into the efficacy of inactivated salmonella vaccines are equivocal.

Before recommending a vaccination program, the relevant pathogen should be shown to be a significant problem on the property and the cost benefit evaluated. Vaccination is not a panacea for a specific pathogen and is only worthwhile if the producer is prepared to address other significant risk factors, especially nutritional and environmental factors. It is not a replacement for poor management.

Two vaccines directed at preventing calf scours are available in Australia: an inactivated and adjuvanted *E. coli* whole cell vaccine to aid in the control of ETEC, and an inactivated whole cell Salmonella vaccine to aid in the control of Salmonellosis caused by *S. dublin* or *S. typhimurium*. In the United States, Europe and New Zealand, rotavirus and coronavirus vaccines are also available.

### *E. coli*

Under natural conditions less than 10% of beef cow colostrum contains antibodies to ETEC (K99+). As ETEC scours occurs during the first three days of life the neonate does not have time to mount a protective immune response to vaccination. Protection is afforded by vaccinating cows in late gestation to ensure high concentrations of colostral antibodies. The protective efficacy of ETEC bacterins is well documented, however good management is needed to ensure calves receive maternal antibodies.

The decision to vaccinate should be based on the cost benefit for each farm and will be influenced by:

- Prior history of ETEC scours
- High stocking density or use of a common calving area
- Projected calving during the wet season
- Large numbers of calving heifers

## Salmonella

No specific data is available regarding the efficacy in suckler beef herds of the Australian Salmonella vaccine described above. Vaccination of cows will provide partial protection against Salmonella challenge, though the level of colostral protection is questionable and the duration of immunity is relatively short. However, as many calves are exposed to Salmonella in the first week of life, colostral protection may be useful in an endemically infected herd.

Where Salmonellosis is a problem it is important to eliminate environmental and nutritional stresses, and to focus on hygiene and increased colostral intake. Where vaccination is justified, producers should be advised that vaccination may not result in complete cessation of the problem. Vaccination may be considered as a long-term strategy to reduce shedding by affected animals.

To maximise colostral protection from Salmonella and ETEC vaccination, pregnant cows should be vaccinated three and eight weeks prior to calving, followed by annual boosters pre-calving.

### Rotavirus and coronavirus

Vaccines against these viruses are not currently available in Australia.

Overseas, two immunoprophylactic approaches have been used against rotavirus and coronavirus infections in calves. The first involves oral vaccination of neonatal calves with a modified live vaccine. The vaccine must be administered immediately after birth, before the calf has sucked, as most cow colostrum contains virus-neutralising antibodies that interfere with the vaccine. There are conflicting reports of efficacy with this type of vaccine.

The second approach involves intramuscular vaccination of cows prior to calving with either modified live or inactivated vaccines. This stimulates a high level of specific antibodies in colostrum and milk during the first few days of life but levels are not protective by three to seven days after parturition.

Passive immunity to bovine rotavirus results in (1) a delay of a few days in the onset of clinical signs and/or (2) a reduced severity of clinical signs, and/or (3) a reduction in the length of the period of viral shedding associated with infection. However, field trials involving bovine rotavirus or bovine rotavirus-coronavirus-vaccinated cows, have not demonstrated a consistent benefit.

## Promote calf immunity

### Confidence: moderate

Minimising stress to prevent disease is a well researched principle, although there are few studies critically evaluating its link with calf scours.

Stress, nutrition and crowding all affect calf health in the first six weeks of life. To optimise calf immunity:

- Ensure that cows calve in body condition score 2.5–3.5 (BCS: 1=thin 5=fat). Monitor cow condition before and after calving.
- Match pasture supply and stock numbers to ensure adequate feed for lactating cows.
- Ensure cows receive adequate trace minerals, especially selenium and copper.
- Provide a sheltered, dry, draught-free area for calves from assisted calving during wet cold weather.
- Provide calving and nursing areas that:
  - are sheltered from the prevailing wind and have shade
  - are well-drained so calves remain dry
  - allow calves access to water within 300m
  - have multiple sheltered areas for calf camps so newborn calves can lie undisturbed

During the first two weeks of life calves spend most of their time sleeping or suckling and under crowded conditions their resting and feeding patterns may be altered. Crowding can be due to a high stocking rate or environmental factors causing calves to concentrate in a small area. Crowding is increased when producers bring springing cows into close paddocks for observation.

## 3. Prevent introduction of new NCD pathogens into calving herds

### Confidence: high

The role of biosecurity in disease prevention is well established.

Biosecurity is important to minimise introduction of pathogens by people, equipment, animals, colostrum, milk or contaminated watercourses.

The following practices should be recommended to producers:

- Do not replace dead calves with bobby calves from another property. Young calves are a high risk for introducing new diseases.
- Avoid the introduction of recently purchased animals into calving groups or mobs with calves less than six weeks old.
- If calves are routinely introduced on a large scale, establish protocols to minimise the risk of disease introduction.
- Keep calving cows and young calves away from paddocks containing watercourses arising from adjoining properties.
- Minimise access of visitors and vehicles from other properties into calving and nursing areas without disinfection.

### Sourcing colostrum for beef properties

Ideally obtain colostrum from beef cows on the home property. Occupational health and safety concerns can be reduced by proper cow restraint and use of a 'tail jack'.

If colostrum must be introduced:

- Ensure it comes from a herd with a known negative BJD and enzootic bovine leucosis status. Facilities for pasteurisation of colostrum are not readily available and pasteurisation at temperatures sufficient to kill *M. paratuberculosis* is likely to result in a significant decrease in IgG levels.
- Collect colostrum from the first milking and ensure adequate immunoglobulin concentration using a colostrometer.

- Clean teats prior to milking.
- Store each cow's colostrum separately and refrigerate or freeze within one hour.

A commercial colostrum supplement containing 4g/L of bovine IgG is registered in Australia. Calves require 100–125g of IgG to provide adequate passive transfer.

## Assessing the effectiveness of preventive strategies

Producers should be encouraged to monitor and record the parameters in table 1 to assess the success of preventive measures and to alert them to a potential NCD outbreak. If parameters are outside desired levels a detailed re-evaluation is required to determine the potential causes of the breakdown.

Table 1: Recommended monitoring of calving herds

Parameter	When to measure	What to record	Desired level	Use of information
Number of sick and scouring calves	Check heifer herds and herds with a past history of NCD daily until all calves are three weeks old  Check other cow herds at least every three days	<ul style="list-style-type: none"> <li>• Mortality</li> <li>• No. treated</li> <li>• ID of calf and dam</li> <li>• Age of calf</li> </ul>	Mortality = 0  Retrospective no. treated <4%*	During calving will alert to potential start of outbreak  After calving can be used to assess success of preventive strategies
Number of cows dry	Branding or weaning (standard time each year)	Increase since pregnancy testing	<5%	Retrospective assessment of effectiveness of preventive strategies
Dystocia %	Weekly during calving	<ul style="list-style-type: none"> <li>• Number assisted</li> <li>• Total cows calved</li> </ul>	Heifers <10% Cows <2%	High dystocia % will result in increased FPT which will increase potential for NCD unless corrective action is taken
Mismothering	During routine checks of calving herd	Calves from unassisted births not sucking within six hours of birth or abandoned	<2%	Increased levels indicate a management problem
Introductions of new animals	As required using NVD	Origin and route of introduction		Assess risk of introduction of new calf scour pathogens

\* When monitoring during calving, calves requiring treatment should prompt assessment of risk factors and initiation of an appropriate control strategy, as disease can spread rapidly if pathogen build-up is not addressed.

## Further reading

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## MLA publications

- More Beef from Pastures: The producer's manual – Module 2: Tactical stock control
- More Beef from Pastures: The producer's manual – Module 4: Pasture utilisation
- More Beef from Pastures: The producer's manual – Module 6: Weaner throughput
- More Beef from Pastures: The producer's manual – Module 7: Herd health and welfare
- Tips & Tools: Prevention of calf scours in suckler beef herds

## Investigation of a NCD outbreak

NCD should be approached as an enterprise level problem, and requires a farm visit and examination of affected animals *in situ*. It is important to systematically identify environmental, management and nutritional factors contributing to the problem and to address them as part of a disease control and prevention strategy. Failure to do so will limit the effectiveness of disease prevention efforts. Implementation of pathogen specific interventions, such as antimicrobial therapy and vaccination, should be guided by further diagnostic investigation, while taking into account the cost benefit of such strategies.

The investigation should include the following steps:

1. Define the problem
2. Examine the affected group and identify risk factors
3. Estimate the cost of the outbreak
4. Examine individual calves
5. Take appropriate samples
6. Interpret all data and make appropriate recommendations
7. Monitor response to recommendations and reassess as appropriate

### 1. Define the problem

#### Confidence: moderate

There is little information on mortality and treatment rates in southern Australian beef enterprises. Figures have been taken from those used in the USA and Canada.

At the onset of the investigation it is important to define the disease duration, progression, morbidity, mortality, age affected and response to treatment. This information may be difficult to obtain depending on the availability of producer records and may require prospective investigation.

The veterinarian should be concerned about an NCD outbreak if any of the following apply:

- % calves requiring treatment during past month >4%
- Neonatal mortality (calves born alive but dying between one and 28 days of age) >3%
- Older calf mortality (calves born alive but dying between one and six months of age) >2%

or if these figures are not clearly defined:

- Pre-weaning mortality (calves born alive but dying between one day of age and weaning) >5%

On large, extensive operations, higher trigger levels may be reached before an investigation is initiated.

Producers often approach veterinarians for over-the-counter advice regarding calf scours. Long term effective management and prevention is more likely to be achieved through on-farm consultation and a review of management procedures. If any of the above trigger points are exceeded, a farm visit is likely to be beneficial to:

- Identify risk factors associated with the outbreak
- Determine aetiological agent(s) and appropriate therapeutic interventions; and
- Plan for the following season to avoid recurrent problems.

## 2. Examine the affected group and identify risk factors

### Principles of an on-farm investigative protocol

- Examine the affected mob *in situ* and observe potential predisposing factors.
- Question the producer regarding joining, calving, paddock and nutritional management, dystocia rates and biosecurity risks.
- Avoid mustering the mob to examine or collect samples from calves as this will increase stress and risk of disease transmission.

An NCD outbreak often results from predisposing factors associated with herd management during the previous year and the management of calving cows.

Risk factors are detailed in table 2.

It is important to identify predisposing management factors and assess:

- the calving paddock, for environmental stresses and areas of pathogen build-up
- calf and cow body condition and current nutrition of the herd
- the make-up of the affected group

Determine the age of calves affected and the proportion of calves in the following four groups:

- Unaffected
- Scouring, but still suckling, bright and alert (can't catch)
- Scouring and ≤8% dehydrated (can catch with some exertion)
- Scouring and >8% dehydrated (easy to catch/collapsed)

The following presentations may help to pinpoint differential diagnoses:

- If most affected calves are less than three days old and all are less than 14 days there is a strong possibility of ETEC.
- If a wide range of age groups is affected, possibly including yearlings or adults, Salmonella or yersinia should be considered. Both may be associated with the presence of blood and mucous in the scour.
- If all affected animals are over three weeks old with most over five weeks, and a high proportion have perineal faecal staining, rectal straining and excessive tail swishing, or an increased incidence of rectal prolapse, coccidiosis is a strong diagnostic possibility.
- If calves are dying suddenly with no signs of dehydration, consider toxicity, toxæmia or septicaemia (Salmonella, clostridial disease or *E. coli*).
- Other than these syndromes there are few characteristic clinical or age-related signs that can be used as aetiological pointers.



Table 2: Risk factors associated with NCD

Increased exposure to enteric pathogens	Decreased colostrum intake, calf welfare and nutrition	Increased risk of introducing pathogens
<b>Major risks:</b>	<b>Major risks:</b>	<b>Major risks:</b>
Joining period >60 days	Dystocia rate in heifers >10%	Any introduction into calving/nursery group
Same calving paddocks used each year	Dystocia rate in cows >2%	Nursery group exposed to potentially contaminated water
Calving paddock used continuously during calving period	>25% of herd are first-calf heifers	Pathogens in colostrum sourced from a dairy property (eg Salmonella)
Calving and/or nursing areas poorly drained	>10% of cows have pendulous abdomens or udders	
Well defined areas for calving: cow camps	ETEC diagnosed and producer not vaccinating	
High-risk stock in the calving and nursery areas within three months of calving	>10% of cows calve in condition score <2.5 or >3.5	
Newborn calves remain with calving cows >24 hours	Calving and/or nursing areas exposed to prevailing winds	
Less than four paddocks used for cows with young calves and same ones used every year	Less than four paddocks used for cows with young calves and same ones used every year	
Age range of calves in nursery group >4 weeks	Calves do not have water accessible within 300m	
Cows and calves can defecate in water source	Young calves are camping in crowded area	
Cows fed on ground in same region of the paddock each time		
<b>Moderate risks:</b>	<b>Moderate risks:</b>	<b>Moderate risks:</b>
Water source exposed to faecal run-off from cattle, especially calves upstream	Mismothering >2%	Pathogens in colostrum sourced from another beef property
Cows or heifers in calving paddock(s) >2 weeks before calving	Feed shortage before or after calving	Visitors or vehicles from other properties entering calving and nursing areas without thorough cleaning and disinfection
Heifers and cows calve in same paddock	<10% shade	
Single watering points encourage cattle to congregate in small area	Copper or selenium deficiency	
	Cows calve in cold, wet and windy weather or hot weather	

### 3. Estimate the cost of the outbreak

It is important to estimate the size and potential cost of the outbreak and its control to determine appropriate diagnostic, control and prevention strategies (see table 3). When calculating potential costs in the midst of an outbreak, assume morbidity and mortality will remain the same as existed during

the previous two weeks. This is a conservative estimate as uncontrolled pathogen build-up may lead to increased disease prevalence. An investment in investigation and control of an outbreak of NCD can be expected to yield ongoing on-farm benefits, especially if the aetiology and risk factors can be identified and successful interventions implemented.

Table 3: Economic losses associated with NCD

Easily measurable losses	Less obvious losses
<ul style="list-style-type: none"> <li>• Cost of calf death (sale value of finished animal minus cost of pasture and supplement consumed, parasite control and vaccination)</li> <li>• Cost of treatment (including labour)</li> <li>• Difference between value of culled dam and cost of pregnant replacement</li> </ul>	<ul style="list-style-type: none"> <li>• Impact on growth rate in affected calves</li> <li>• Loss of genetic potential</li> <li>• Decreased capacity to improve and maintain herd</li> <li>• Cost of preventive measures</li> </ul>

### 4. Examination of the individual calf

Select a minimum of six calves, representing the range of presentations, for a full clinical examination. Neonates are prone to sepsis, which may be reflected by scleral injection, hypopyon, omphalitis and septic arthritis. A high proportion of infected navels may reflect FPT or a poor calving environment. Septic arthritis may be indicative of a secondary bacteraemia. Fever is an unreliable indicator of sepsis in neonates. Depressed mentation is common and may reflect metabolic acidosis, hypothermia, hyperthermia, electrolyte derangements (hypo- or hypernatraemia, hypo- or hyperkalaemia), hypoglycaemia or septicaemia.

Mentation, suckle reflex, ability to stand and hydration should be assessed in individual calves to determine appropriate treatment. The dam should be examined for adequate milk supply.

### 5. Collection of laboratory samples

The following samples can be collected to identify aetiological agents and predisposing causes:

- Faecal samples, plus post-mortems where appropriate, to identify aetiological agents
- Serum samples to determine the role of FPT
- Blood or liver samples to determine any underlying mineral deficiencies

The appropriate testing strategy will depend on the differential diagnosis, the likelihood of establishing an aetiological diagnosis that will result in changes to treatment, control or preventative strategies and the cost of the proposed diagnostic protocol.

Table 4 provides 'rules-of-thumb' to help determine at which point diagnostic test(s) are likely to provide a cost benefit for the producer.

Table 4: Common diagnostic tests for NCD and criteria for determining their cost benefit

Proposed test	Indications	Economic benefit likely if:
Enterotoxigenic <i>E. coli</i>	Affected calves <4 days old	One calf has died or >10 likely to be affected
Salmonella	<ul style="list-style-type: none"> <li>Blood in scour</li> <li>Severe clinical signs</li> <li>Calves dying</li> </ul>	Projected cost of disease >2.5 times cost of laboratory tests
Full calf scours investigation	Calves dying	Projected cost of disease >4 times total cost of laboratory tests and post-mortem fees
Failure of passive transfer	<ul style="list-style-type: none"> <li>Affected calves &lt;3 weeks</li> <li>Dystocia in affected group &gt;5%</li> </ul>	>5 calves affected

## Faecal sampling

### Confidence: moderate

Six faecal samples are likely to give an indication of the most common pathogens contributing to NCD in a herd. More would be useful to establish the prevalence of different pathogens, but in many situations the cost benefit is not warranted.

It is important to collect sufficient, appropriate samples at the first visit to maximise the chance of a diagnosis. Collect a minimum of six samples from calves exhibiting a 'typical' presentation for the outbreak. Select calves at different stages of disease but preferably half should be in the early stages prior to initiation of treatment. These animals often have watery diarrhoea with little faecal staining around the perineum and tail, and may be difficult to detect unless depressed or not suckling. If only two or three calves are affected, samples from non-affected calves may help to assess pathogen prevalence on the farm, as cohorts may be sub-clinically infected. Routine collection of samples from unaffected animals is unlikely to prove useful as a proportion of unaffected calves will shed aetiological agents. A single faecal sample from an individual animal has limited diagnostic value due to the potential for discord between clinical signs and faecal shedding.

Rectally stimulate calves to obtain a good sample using a new disposable glove for each calf, and obstetrical lubricant that does not contain antiseptic.

For a full diagnostic work-up collect at least 5–10g of faeces into a leak-proof container. Where possible provide a yellow top container that is at least half full. Pre-number containers with indelible pen, and write all information, including cow and calf ID on an accompanying sheet of paper to minimise faecal contamination.

Where it is necessary to yard the mob to sample calves, minimise the risk of stress and disease transmission by holding cows and calves for as short a time as possible and by drafting healthy calves and their dams into a larger holding area. Moving the affected mob will spread the pathogen load around the farm. Prevent contact with other calves less than six weeks of age. If an affected mob is run through a crush that is also used for calving cows, the area must be thoroughly cleaned after use. If the crush floor and yards are concrete, scrape and clean with bleach or Virkon™ (see table 7). If the crush floor is soil, replace the top 10cm and add 1kg lime/m<sup>2</sup>.

### Post-mortem examinations

Post mortem examination of fresh, dead calves will provide the most valuable aetiological information.

#### Tip

*Calf post-mortems will be more rewarding when a severely affected calf is euthanased. Autolysis within the gut occurs within five minutes and it is likely that the samples will be non-diagnostic within a few hours of death.*

Gut autolysis starts within five minutes of death and samples taken for histological examination are likely to be non-diagnostic within a few hours. Despite this, a gross evaluation may confirm or challenge previous necropsy findings.

If mortality is >3% or previous post-mortem findings have not been rewarding, consider euthanasia of a calf in the early stages of disease. Producers will often present a cachectic calf that has had a protracted illness. These animals are unlikely to be of high diagnostic value due to debilitation and secondary infection, and may lead to disillusionment with the diagnostic process. As a single necropsy may identify an isolated, unrelated case, results should always be considered in conjunction with other necropsies and faecal testing of other animals in the group.

**A necropsy is not a sample collecting exercise.** It should be approached in a systematic manner in the same way as a clinical examination. Describe and interpret what is found, even if tissues appear normal. Start by describing body condition. Examine calves that have died within 48 hours of birth for signs of calving difficulties such as meconium staining inside the back legs, swollen head or tongue and scleral haemorrhages. Examine all body cavities and organs systematically. Examine the brain and meninges for evidence of developmental defects and meningitis and fix tissue for histopathology. Assess the nutritional status of the calf by examining fat reserves around the kidneys and coronary band.

The whole gastrointestinal tract should be examined from mouth to anus. Special note should be taken of:

- Erosions, proliferative lesions, ecchymoses or petechial haemorrhages
- Congestion, oedema, inflammation or emphysema
- Fluid distension
- Enlarged lymph nodes
- Nature of intestinal and colonic contents

### *Histological samples*

Table 5 lists the parts of the alimentary tract that should be examined and sampled. **This is not intended to reflect a protocol for conducting a complete necropsy.** Collect samples from the major organs, even if they will not initially be processed. Intestinal samples should be 3–4cm long, as short sections will curl. Ensure gut lumen is open prior to dunking sample in formalin or syringing formalin through the section. Be careful not to traumatise the gut. Do not cut intestines lengthwise as this will smear off the intestinal lumen cells, although a small lengthways cut at one end may facilitate formalin perfusion. Samples from other organs should be about 1cm x 2cm to allow adequate fixation. It is better to take several small samples than one large one.

#### **Tip**

*Syringe formalin through intestinal samples for histology, to allow better distribution and rapid fixation of important tissues.*

Table 5: Alimentary tract post-mortem of the calf with NCD

Location	Specifically observe	Possible aetiology	Samples required
Mouth	Oral erosions	Pestivirus	If observed collect and fix tissues
	Proliferative lesions	Bovine papular stomatitis	
Oesophagus	Erosions	Pestivirus	If observed collect and fix tissues
Abomasum	Inflammation and emphysema in rugal folds	Clostridia	Collect tissue for histopathology when gross pathology is observed  Abomasal contents (on ice) for detection of clostridia and clostridial toxins
Small intestine	Inflammation and fluid distension	Rotavirus Coronavirus Salmonella Cryptosporidia	Collect sections of duodenum, mid jejunum, and ileum for histopathology
Mesenteric lymph nodes	Enlargement	Rotavirus Coronavirus Salmonella Cryptosporidia	Collect samples for culture (Salmonella) and histopathology*
Caecum	Inflammation	Coronavirus Salmonella Coccidia	Contents on ice for culture  Tissue for histopathology if gross lesions
Spiral colon	Inflammation	Salmonella Coronavirus Coccidia	Collect tissue sections for histopathology
Rectum	Inflammation	Coccidia Pestivirus	Tissue for histopathology  <b>Always</b> submit contents for culture and pathogen detection assays (rota, corona, cryptosporidia).

\* Mesenteric lymph nodes can be used to detect an inflammatory response as autolysis is always faster in gut

Add 10% neutral buffered formalin to sample pots in the field to minimise post-mortem change. Ideally fix samples overnight at a 1:10 ratio of tissue:formalin and then place in a fresh container with formalin-soaked gauze to send to the laboratory. Formalin should not be drained from large specimens. If the client is paying for each sample separately, put each section in a different pot to allow sequential processing.

### Tip

Add formalin to samples in the field, and change before sending to the laboratory.

### Fresh samples

Samples of rectal contents should be tested for enteric pathogens. Fresh samples of mesenteric lymph nodes, spleen and liver are useful for Salmonella culture.

If the case is suggestive of enterotoxaemia and there are sections of haemorrhagic intestine, submit intestinal samples for histopathology and intestinal contents for the presence of toxin. Demonstrating the presence of *C. perfringens* toxins, or the capacity to produce toxins, provides support for, but does not confirm, a clinical diagnosis, as isolates from normal calves often produce toxin. Diagnostic laboratories in Australia do not routinely perform quantitative



clostridial counts. A diagnosis of clostridial enteritis is therefore based on a cumulative picture created by the case history, clinical presentation, gross pathology, histopathology and ancillary testing (toxin production).

## Laboratory tests

### Confidence: high

The role of the five major pathogens is well established worldwide. It is possible that other viruses may be involved but commercial diagnostic tests have yet to be developed for these.

### Establishing the pathogens involved

As indicated on page 9, it may be possible to target tests for ETEC, Salmonella, yersiniosis or coccidiosis from the presenting syndrome, however a limited panel of tests may result in a misdiagnosis. Faecal testing for rotavirus, cryptosporidia, coronavirus and Salmonella is most likely to determine the aetiological agent. ETEC should be included if calves under three days of age are affected.

If initial samples test negative for all major pathogens and the severity of the outbreak warrants further investigation, ancillary tests or necropsy should be considered depending on clinical signs. Further tests may include faecal float for coccidiosis, intestinal worms or giardia and antigen capture of BVD from the spleen. If ante-mortem bloating is a feature clostridial abomasitis should be considered.

Table 6: Advantage and disadvantages of common tests for FPT

Test	Advantages	Disadvantages	Suggested use	Level indicative of FPT
<b>Serum protein</b>	Cost-effective and accurate in healthy calves	Affected by dehydration	Group screening test to assess or monitor healthy calves	<5.0 g/L <5.5 g/L sick calves
<b>Whole blood immunoassay<sup>1</sup></b>	Rapid calf-side test	Costly	Group screening or assessment of individual calves	
<b>GGT</b>	Reliable in sick calves at 50 IU/L cut-off	Rapid decrease with age; variable endpoint	Assessment of sick calves	<i>Healthy calves</i> 4 days <100 IU/L 5–7 day <75 IU/L 8–10 days <50 IU/L  <i>Sick calves</i> <50 IU/L calves aged <21 days

<sup>1</sup> Midland Bioproducts quick test kit

### Tip

For the best chance of establishing all potential aetiological agents, faecal samples should be tested for rotavirus, cryptosporidia, coronavirus and Salmonella, plus ETEC where indicated.

Isolation of *E. coli* from the GIT does not constitute a diagnosis unless the isolate is demonstrated to possess virulence attributes that correlate with the clinical presentation and histopathological findings. *E. coli* possessing virulence attributes (enterohaemolysins, adhesins and shiga toxins) may be isolated from normal calves.

### Evaluation of passive transfer

Blood from 10 calves, two to seven days of age (up to 14 days if necessary), should be tested to assess the adequacy of passive transfer (serum IgG >10g/L (1,000mg/dL)). Table 6 presents the advantages and disadvantages of several common methods.

Initially, sample sick calves when they are being treated. If more than 25% of sick calves have FPT, the problem should be confirmed in healthy newborn calves as immunoglobulins may be lost from the gut with inflammatory diarrhoea. If FPT is a herd problem contributing risk factors should be assessed.

Direct tests for IgG are expensive, therefore screening tests in calves estimate serum IgG content from the total globulin level of other proteins whose transfer is associated with the absorption of IgG.

### *Determination of mineral deficiencies*

Severe mineral deficiencies may predispose to NCD. In districts with a history of deficiencies, a screening blood test is likely to identify the problem. Liver biopsies will provide more accurate measurements of copper and selenium tissue stores.

### **Sending samples to the laboratory**

Calf scour samples are potentially zoonotic and should be packaged to IATA 650 standard. Containers with liquid should be sealed inside a second leak-proof container. Containers with faecal samples should be placed in a zip-lock bag and shipped in an esky with an iceblock. The outside of the packaging should be clean.

Submission forms should contain complete epidemiological details and be placed in a plastic envelope separate from the samples. Provision of adequate information will allow the laboratory to correlate the results with the clinical presentation to provide a more meaningful assessment of significance. Quality data recording also facilitates national disease monitoring by government and industry groups. This can allow targeted prioritisation of resources and research.

## **6. Interpret all data and make appropriate recommendations**

The information should be consolidated and analysed to determine the major factors that have resulted in the NCD outbreak.

### *Analysis of risk factors*

Rank potential risk factors by their likely contribution to the problem, including their relationship to any established aetiological agent, and the costs, benefits and practicality of change. Determine the five most important factors for discussion with the producer. Establish key control points at calving and between calves.

### *Interpretation of laboratory results*

The major enteric pathogens can be shed by apparently normal calves, and the relevance of these pathogens in a faecal sample may be questioned. A causal relationship may be inferred by a high prevalence of a known, or multiple known, enteric

pathogens. Correlating the presence of a pathogen with compatible clinical signs, response to treatment, gross pathology and histopathology, provides support for a more definitive diagnosis. Multiple pathogens are frequently incriminated in NCD. Identification of specific pathogens can facilitate prevention, treatment, and control efforts.

### *Reporting to the producer*

The outcomes of the investigation should be reviewed in consultation with the producer and a prioritised and agreed list of management changes to control the outbreak, and prevent recurrence in subsequent years, should be determined. The producer should also be provided with advice on appropriate treatment of affected calves. If the cause is primarily viral it is useful to indicate to the producer why antimicrobial therapy is not effective and to redirect efforts to supportive fluid therapy and preventive management interventions. Major recommendations are best presented as a clear single-page written report containing:

- the outcomes of the investigation
- key points, time lines and delegation of responsibility for
  - control strategies
  - management changes
  - treatment
- arrangements for follow-up

## **7. Monitor response to recommendations and reassess as appropriate**

Table 1 lists parameters for monitoring NCD. Specifically monitor mortality rate, the percentage of calves requiring treatment and the response to treatment. New cases are likely to continue in the affected mob for several weeks as the proportion infected may be high before clinical cases occur. Spread to unaffected mobs may indicate a need for further control measures. Poor response to treatment or an unexplained high mortality rate may warrant further diagnostic investigation. **Further reading** Weaver, D. M., J. W. Tyler, et al. (2000). "Passive transfer of colostral immunoglobulins in calves." *Journal of Veterinary Internal Medicine* **14**(6):569–77.

## Control of a NCD outbreak

### Confidence: moderate

Recommendations are based on sound epidemiological principles, plus research and advice from pasture based industries, but have not been evaluated in Australian pasture based systems.

In most cases, initial control measures will need to be implemented in the absence of a definitive diagnosis, however an established aetiology is useful to target specific control measures.

Control measures should aim to:

1. Protect newborn calves and young calves in unaffected mobs from infection
2. Isolate the affected mob
3. Minimise transmission within the affected mob
4. Minimise spread of zoonotic pathogens to humans.

If the outbreak only involves a specific group of cows, such as heifers, control measures may only be required for this mob and the cohort animals yet to calve.

### 1. Protect newborn calves and young calves in unaffected mobs

Any predisposing risks identified during the investigation should be minimised in cows yet to calve.

#### Tip

*Moving pregnant cows to a new clean calving paddock is one of the best intervention strategies in the face of an outbreak.*

Decrease exposure to infection and maximise calf immunity:

- Move all pregnant cows to a clean calving paddock. If possible, change this paddock every three weeks or more frequently if wet and boggy.

- Drift off newborn calves and their dams from the calving herd to a 'nursery' group in a fresh paddock. Ideally start a new group every three weeks.
- Do not put new calves into an affected group.
- Sick calves and newborn calves should not be handled by the same person without thorough disinfection between operations. Always have overalls and gloves specifically for treating sick calves and wear clean overalls and disposable gloves when handling newborn calves.
- Place supplementary feed in a new region of the paddock each time, or use troughs, feeders or racks to avoid faecal contamination.

Calves born to cows with dystocia have much higher risk of FPT. Strip colostrum while assisting with calving and feed to the calf (1L/20kg) by stomach tube to help reduce the risk of scours.

Assess calf immunity by evaluating FPT and the percentage of assisted calvings. In many cases a high proportion of FPT can only be addressed with long-term solutions such as:

- Breeding and nutritional strategies to minimise dystocia
- Management of heifers to ensure adequate nutrition prior to calving
- Ensuring adequate levels of copper and selenium

NOTE: If calving cows have a condition score  $\leq 2.5$ , or are not receiving enough feed, FPT may simply be due to low volume, high viscosity colostrum that is difficult for calves to suck. This is often a problem in heifers' calves. Provision of better nutrition to pregnant cows, especially springing heifers, may alleviate the problem.

#### Vaccinating in the face of an outbreak

When contemplating vaccination as a control measure, consider:

- The time left to calve
- The time until development of protective immunity
- The efficacy of the vaccine
- The likely cost benefit of the vaccination program

Two inactivated whole cell vaccines directed at aiding the control of calf scours are available in Australia: an *E. coli* vaccine to help prevent ETEC, and a Salmonella vaccine to help prevent Salmonellosis.

### **Confidence: moderate**

The use of vaccination to prevent ETEC diarrhoea is well established, but studies on the efficacy of inactivated Salmonella vaccines are equivocal. There are no studies evaluating the efficacy of the Salmonella vaccine available in Australia in beef herds.

Vaccination of previously unvaccinated pregnant cows in the face of an outbreak of ETEC is likely to be beneficial if the outbreak occurs at the beginning of the calving season. Two injections three weeks apart are required. Benefit has been reported by vaccinating all cows that are more than 10 days away from calving.

The efficacy of the Salmonella vaccine to prevent disease in commercial beef herds has not been established in Australia. Experimental studies with other Salmonella bacterin vaccines indicate that protective antibodies are produced within two weeks of the first injection, with peak antibody responses seen two to four weeks after the booster dose. Vaccination against Salmonella should always be accompanied by other management interventions to reduce pathogen exposure and increase host immunity (nutrition and environmental management) to obtain the best results. Due to the time required for stimulation of active immunity, vaccination of neonatal calves is unlikely to be of benefit.

Where vaccines that stimulate colostral immunity are used, calves that have not suckled within six hours of birth should be supplemented with colostrum.

## **2. Isolate the affected mob**

### **Tip**

*Salmonella and coronavirus are spread via nasal secretions so if these diseases are implicated increase the separation between affected and unaffected mobs.*

- An adjacent paddock is usually sufficient, however Salmonella and coronavirus are spread in nasal secretions. Increased separation is needed if these pathogens are implicated. If management allows, keep the mob in one paddock or area of the farm to minimise contamination of paddocks. Graze contaminated paddocks with stock older than three months for as long as possible following an outbreak.
- Do not use infected paddocks for calving cows or nursery mobs in the subsequent calving period if possible.
- Separate infected and non-affected mobs until all calves are six weeks or older. If handling is required, yard unaffected calves first.

## **3. Minimise transmission within the affected mob**

### **Tip**

*Fence off heavily used calf camps in affected paddocks.*

- If <10% of calves are affected, isolate scouring calves and their dams if practical. If >10% are affected, a high number of the remaining animals are likely to develop clinical disease or are already sub-clinically affected.
- Fence off heavily used calf camps in affected paddocks.
- If the area around water troughs is wet and muddy, apply soil, sand or woodchips plus 1kg lime/m<sup>2</sup>.
- Drift off cows with calves older than six weeks from the affected group, and quarantine them in one area for as long as feed allows.
- Ensure affected calves have no access to watercourses or surface water.
- Delay stressful management procedures (eg dehorning, castration, dietary changes including weaning and transport) for as long as practical to allow the outbreak to run its course.
- Use management procedures to minimise faecal build-up, ie when supplementary feeding use a different area each day.

## 4. Minimise spread of zoonotic pathogens to humans

### Tip

Wear overalls and disposable gloves when working with sick calves. Keep contaminated clothing away from family members.

Farmers and their employees should be informed of the zoonotic potential of calf scour pathogens, specifically Salmonella and cryptosporidia.

Recommendations for people working with calves include:

- Wear overalls and disposable gloves when working with sick calves.
- Always wash your hands after working with calves.
- Do not eat, drink or smoke while working with calves.
- Do not work with sick calves when you are on antibiotics, immunocompromised, infected with HIV or taking immunosuppressive medication.
- Keep contaminated clothing away from family members especially children, seniors and immunocompromised people.

### Further reading

Larson, R. L., J. W. Tyler, et al. (2004). "Management strategies to decrease calf death losses in beef herds." *Journal of the American Veterinary Medical Association* **224**(1):42–48.

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Radostits, O. M. and S. D. Acres (1980). "The prevention and control of epidemics of acute undifferentiated diarrhea of beef calves in Western Canada." *Canadian Veterinary Journal* **21**(9):243–9.

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neonatal mortality in the beef calf." *Veterinary Clinics of North America – Food Animal Practice* **10**(1):119–126.

### MLA publications

More Beef from Pastures: The producer's manual – Module 2: Tactical stock control

More Beef from Pastures: The producer's manual – Module 4: Pasture utilisation

More Beef from Pastures: The producer's manual – Module 6: Weaner throughput

## Treatment of the scouring beef calf

### Confidence: high

Treatment of scouring calves is well established. However there is a limited range of suitable electrolyte products available in Australia.

### Practical aspects

Management will depend on the severity of infection, percentage of calves affected and the ease with which cattle can be handled. If <10% of calves require treatment and separation of these calves and their dams is practical, do so to minimise disease transmission and facilitate monitoring and repeat treatments.

Cows and sick calves can be separated from the mob by restraining the calves and drifting unaffected cow–calf pairs into a different paddock, or by enticing the dams to follow a vehicle transporting the sick calves. It should be remembered that apparently unaffected cattle may shed pathogens and if there is limited availability of 'clean' paddocks, these should be reserved for pregnant cows. Yarding should be avoided as it will facilitate disease spread, increase calf stress and may result in mismothering.

### Tip

Construct a temporary treatment shelter in the perimeter of the paddock for dehydrated calves requiring repeat treatments.

On many properties the most practical solution is to leave less severely affected calves with their dams



and to construct a temporary treatment shelter in the perimeter of the paddock for calves that require more intensive treatment. Both groups of calves should have easy access to clean water. Confined calves should receive 12–24 hours of intensive therapy to ensure rapid return to their dams. The shelter should protect from drafts and provide shade, but allow contact (nose to nose) with the dam to minimise separation stress. It could be simply constructed with three gates, movable yard sections or large square hay bales, with tin or tarpaulin to provide shade and shelter. Move the treatment shelter weekly to an adjacent area to prevent pathogen build-up. Fence off the surrounding area so that the isolation area gets slightly bigger each week.

If calves require treatment for longer periods, an isolation area that keeps calves with their dams, but allows for easy catching and treatment, will be needed. Unless calves are severely dehydrated and require repeat administration of fluids, they should be kept with their dam to minimise stress and ensure adequate energy intake.

Table 7: Disinfectants for some bacterial and vira NCD pathogens

Disinfectant	Use concentration ‡
Sodium hypochlorite (bleach)	1,750ppm = 1.75g/L* solution (pH 6 to 7)
Povidine iodine	1% available iodine
Potassium monopersulfate (Virkon™)	1% solution

‡ A minimum 10 minute contact time is required \* Household bleach is 42g/L – use a 1:25 dilution

Calves that require treatment should be clearly identified with an ear tag, neckband, rattle or spray paint.

### Disinfection of treatment areas

Treatment areas and equipment should be made of plastic or metal to facilitate cleaning. Clean surfaces regularly as most disinfectants are inactivated by organic material. Scrubbing is preferred to high-pressure sprays which can aerosolise organisms allowing dissemination.

Separate equipment and clothing should be used to administer oral electrolytes to sick calves and colostrum to newborn calves. Salmonella and coronavirus are shed in saliva and can contaminate equipment used for oral medication. Equipment

should be washed with warm soapy water to remove the fat residue left by milk and colostrum and rinsed prior to disinfection.

Apply disinfectants for at least 10 minutes before rinsing. Temperature, pH and water hardness will affect the efficacy of disinfectants. Always follow manufacturer’s directions. Suitable disinfectants are shown in table 7. Virkon™ also has a detergent action that facilitates cleaning.

Rotavirus is relatively resistant to many common disinfectants, such as chlorhexidine. It is not affected by soaps and washing with soap alone may actually spread the virus on the washed surface.

Cryptosporidia are extremely resistant to most veterinary disinfectants and it is therefore important to physically remove debris. They can survive for months in water but are susceptible to drying, so where possible use this to reduce the challenge exposure. Virkon™ inhibits, but does not kill, cryptosporidia.

### Evaluating the calf and determining treatment options

Dehydration, metabolic derangements and sepsis are common in scouring calves. Medical management should be directed at correcting fluid, electrolyte, metabolic and acid base derangements, and at prevention or treatment of sepsis.

Without laboratory support, field evaluation and therapeutic interventions should be directed at the most common derangements: dehydration, acidosis, sepsis and hypoglycaemia.

**Hydration** is estimated by measuring skin tent, evaluating mucous membranes and observing eyeball position (see table 8). Acutely affected calves may present with dehydration prior to the onset of diarrhoea if fluid is sequestered in the lumen of the gastrointestinal tract.

**Acidosis** is common and in the absence of laboratory support should be anticipated in depressed, dehydrated, scouring calves (see table 9).

**Sepsis** may be evident from pyrexia, scleral injection or the presence of fibrin clots in the anterior chamber.

**Scouring calves that are bright and alert, suckling and not dehydrated, often do not require treatment.**

The decision tree in figure 1 can be used to direct the implementation of therapeutic protocols. Early detection is important to correct deficits and prevent calves from losing their suckle reflex and becoming recumbent. Special attention should be paid to detect calves with very watery faeces. These calves

often do not have staining around their perineum and may be easily missed. Treatment of calves with fluids at this stage will maximise their chances of survival.

### Fluid therapy

Re-hydration and correction of electrolyte and acid base disorders are the founding principles behind the treatment of calf scours as more calves die from dehydration and acidosis than sepsis. Oral rehydration solutions are very effective when given early, if the composition and volume of the solution is appropriate to meet the calf's needs.

Oral administration of alkalinising electrolyte solutions is recommended for calves that are 5–8% dehydrated, can still stand and have a weak suck reflex. If the calf is dehydrated and recumbent, give IV fluids.

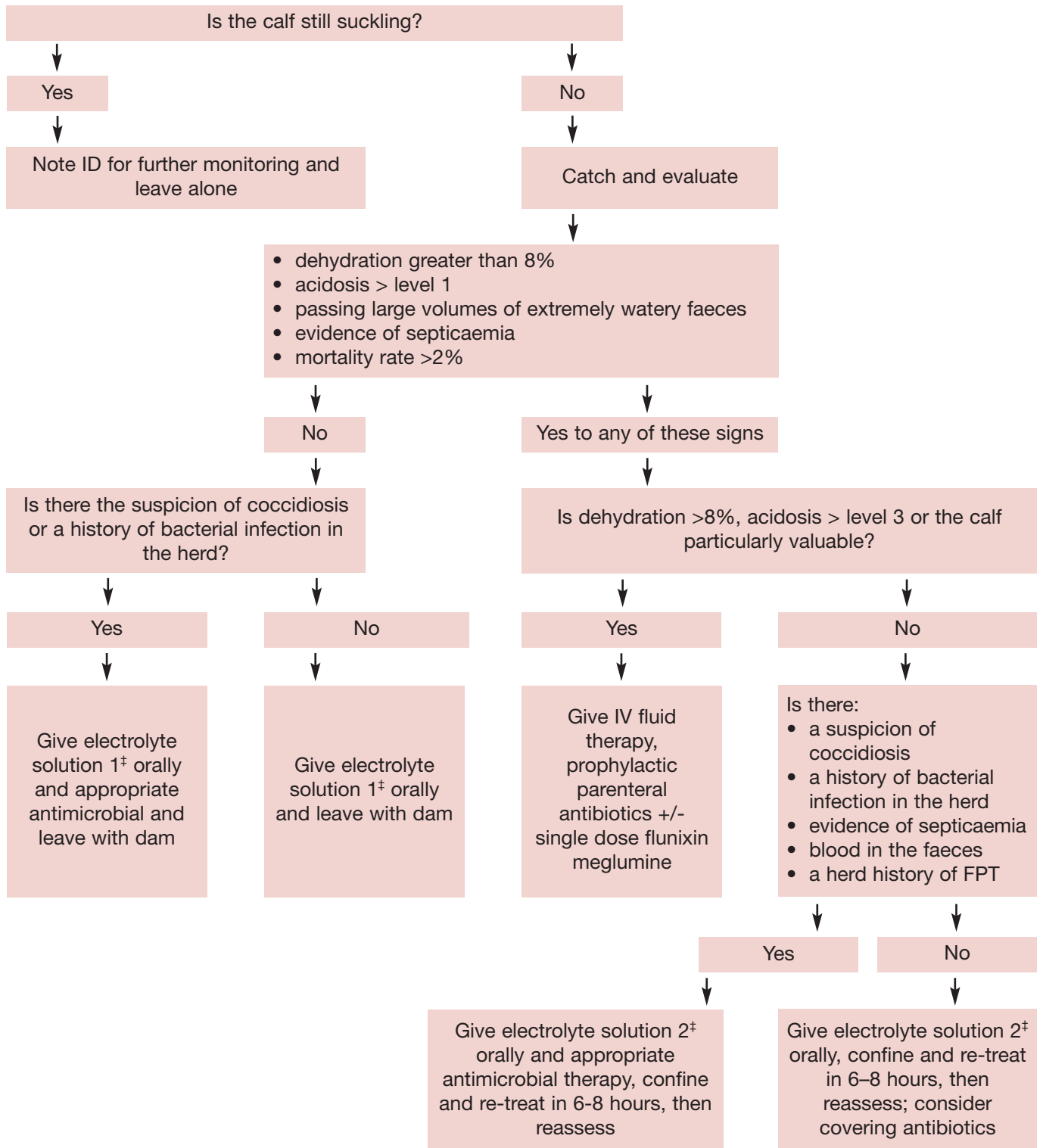
Table 8: Assessing dehydration in scouring calves

% dehydration	Eyeball sunkness	Skin tent time (seconds)	Mucous membranes	Fluid therapy required
0–4	None/slight	1–4	Moist	None
5–8	Slight separation between eyeball and orbit	5–10	Tacky	Likely to respond to oral electrolytes
9–10	Up to 0.5cm between eyeball and orbit	11–15	Tacky	Requires intravenous fluids
11+	Gap between eyeball and orbit is 0.5 to 1cm	>15	Dry	Requires intravenous fluids and repeated monitoring

Table 9: Assessing acidosis in scouring calves

Acidosis level	Clinical signs	Urine pH	Estimated base deficit (mmol/L)	
			≤8 days old	>8 days old
1	Bright and alert Strong suckle reflex Warm mouth	>6.5	0	5
2	Standing or sitting quietly Will move if disturbed Weak suckle reflex Slightly cold mouth	6.0–6.4	5	10
3	Depressed Unwilling/unable to stand Sternal recumbency No suckle reflex Cold mouth	<6.0	10	15
4	Collapsed Death imminent Sternal recumbency No suckle reflex Very cold mouth	<6.0	10	20

Figure 1: Decision tree to determine the appropriate treatment for a scouring calf



† See table 10 for properties of electrolyte solutions and table 11 for daily fluid volume requirements

## Oral electrolyte solutions

### Selecting the correct oral electrolyte solution

Oral solutions should:

1. Supply sufficient sodium to facilitate normalisation of extracellular fluid deficits. Recommended concentrations are 60–133mmol/L.
2. Provide agents that facilitate absorption of sodium and water from the intestine (eg glucose, specific amino acids such as glycine, or acetate or propionate).
3. Provide an alkalisng agent to treat metabolic acidosis (eg sodium bicarbonate, citrate, acetate and propionate).
4. Provide energy, as electrolyte solutions may be administered instead of milk or milk replacer for short periods of time.
5. Provide potassium, which is depleted by faecal loss. Most solutions contain between 10 and 30mmol/L.

Dehydrated calves are likely to be acidotic and the initial rehydration fluid should contain 40–80mmol/L of an alkalisng agent. Electrolyte solutions that contain >40mmol/L of bicarbonate or citrate have

marked adverse effects on milk clotting. Solutions containing bicarbonate may also alkalisng the gastrointestinal tract of milk-fed calves and promote bacterial overgrowth, ETEC attachment and toxin production. These solutions should not be fed to calves that are to be left with their dam and are bright enough to suckle within two hours.

Oral electrolyte solutions are compared to the osmolality of plasma, which is 306mOsm/L. Solutions can be iso-osmolar (300–312mOsm/L), hyperosmolar (>312mOsm/L) or hypo-osmolar (<300mOsm/L). Large quantities of solutions <250mOsm/L may cause haemolysis and should be avoided. For short-term administration in standing calves, osmolality is unlikely to be clinically relevant, and sodium and glucose levels, together with alkalisng ability, are more important. Hyperosmolar solutions are likely to result in a better outcome in a severely dehydrated calf or when repeat administration is required, however they will induce hypernatraemia if they are the only source of fluids and should only be used repeatedly if the calf is consuming milk or water from other sources.

There is no ‘perfect’ rehydration solution that can be used in all situations. The producer may need to keep three products on hand:

Table 10: Properties of oral electrolyte solutions

Type 1	A solution containing acetate or propionate for use in calves that are still standing and can be left with their dams.
Type 2	A strongly alkalisng solution for calves that are severely affected and require repeat therapy and isolation.
Type 3	A solution that will provide enough energy for maintenance to use in calves that won’t suckle after 24 hours

### Administration of oral electrolyte solutions

Due to the difficulty in catching and treating beef calves, farmers should give four litres (or 10% of calf bodyweight if <40kg) at the initial treatment. If required, a second treatment can be given 6–12 hours later. For calves over 70kg, five litres can be given at a time. An oesophageal feeder is a quick and effective method for administering oral fluids and producers should be instructed on their use. If calves appear bloated and uncomfortable, give smaller

amounts more frequently. It is important to correct deficits and compensate for further losses within the first 12 hours. Cease treatment if reflux or excessive distress is seen.

#### Tip

As not all fluid will be absorbed in the intestine, the faeces of calves treated with oral electrolytes may become more watery and increase in volume, even when the calf is improving clinically.



### Calculation of the required daily volume

Calculate the initial volume required and reassess on a daily basis.

The total daily fluid requirement is shown in table 11 and includes:

- the volume required to correct deficit (% dehydration x body weight)

- estimated ongoing losses in diarrhoea (1–4L/day)
- maintenance requirements (3ml/kg/hour)

Only 60–80% of oral fluids are absorbed and this must be considered when calculating fluid requirements.

Table 11: Calculation of daily oral fluid requirement (L) and recommended frequency of administration (assuming 70% of fluid absorbed)

% dehydration		0%			5%			8%		
		Fluid required (L/day)								
Estimated loss through scours (L/day)		1	2	4	1	2	4	1	2	4
Weight of calf (kg)	30	4.6	6.0	8.8	6.7	8.1	11.0	8.0	9.4	12.3
	40	5.6	7.0	9.9	8.4	9.9	12.7	10.2	11.6	14.4
	50	6.6	8.0	10.9	10.2	11.6	14.5	12.3	13.8	16.6
	60	7.6	9.1	11.9	11.9	13.4	16.2	14.5	15.9	18.8
	70	8.7	10.1	13.0	13.7	15.1	18.0	16.7	18.1	21.0
	80	9.7	11.1	14.0	15.4	16.8	19.7	18.8	20.3	23.1
Minimum frequency of administration of divided daily fluid volume										
Twice daily		Three times daily			Four times daily					

### Intravenous fluid therapy

If a calf is depressed and unwilling to suckle, IV fluids are required. Ideally dehydration and acidosis should be slowly corrected over 24 hours, but few problems are usually seen if correction occurs over four hours. To determine the correct IV fluid therapy protocol:

- Select the appropriate combination of fluids
- Determine the amount of bicarbonate required to treat acidosis
- Determine the volume of fluid required to correct the deficit
- Provide additional energy
- Determine ongoing maintenance requirements

#### 1. Selecting the appropriate combination of fluids

In the field, the prime use of IV fluid therapy is to correct initial deficits. If the facilities and producer

nursing skills are good, or the calf particularly valuable, IV fluids can be used for maintenance and ongoing losses, but in most situations it is more practical to use oral solutions once the initial deficit is reversed.

Both large volume isotonic (or mildly hypertonic) and small volume hypertonic solutions may be used to successfully treat collapsed calves. Recommended combinations are:

- Normal saline with added bicarbonate. This is an 'all-in-one' solution to correct base and fluid deficits.
- Isotonic bicarbonate to correct base deficit followed by acetated Ringers (Normosol R) or lactated Ringers (Hartmann's) to correct volume deficit.
- Isotonic bicarbonate to correct base deficit followed by hypertonic saline and an isotonic oral electrolyte solution to correct volume deficit.

### Tip

When *hypertonic* solutions are administered they *must* be given concurrently with an *isotonic* oral electrolyte solution

Large volume solutions require facilities for the calf to be restrained and the fluids set up and left to run. Small volume hypertonic solutions can be administered over four minutes and consequently require minimal restraint of the calf. Hypertonic solutions **must** be given concurrently with an **isotonic** oral electrolyte solution.

## 2. Treatment of acidosis

Calves requiring IV fluids should always be treated for acidosis, especially if they are over eight days of age. Clinical trials show that bicarbonate is the most

effective alkalisating agent. Base deficit (see table 9) can be used to calculate the bicarbonate required using the following equation:

$$\text{mmol bicarbonate} = \text{body weight (kg)} \times \text{base deficit (mmol/L)} \times 0.5$$

The mmol bicarbonate = the volume (mls) of 1M 8.5% bicarbonate solution (see table 12)

Bicarbonate may be added to the total volume of normal saline or administered as an isotonic solution prior to other electrolyte solutions.

Isotonic bicarbonate solution can be made by adding 150ml of 8.5% bicarbonate solution to 850ml sterile water or 5% dextrose (or 850ml normal saline if these are not available). The latter solution is mildly hypertonic but of little consequence when only 1–3 litres are administered. The volume of isotonic bicarbonate solution required is shown in table 12.

**Bicarbonate should only be added to sterile water, 5% dextrose or normal saline. Precipitates may form with other solutions.**

Table 12: Bicarbonate requirements of calves depending on estimated base deficit

Weight of calf (kg)	Base deficit (mmol/L)	Bicarbonate requirements (mmol/L) = mls of 8.5% bicarbonate solution	Volume (L) of 1.3% bicarbonate solution (isotonic)
30	10	150	1.0
	15	225	1.5
	20	300	1.9
40	10	200	1.3
	15	300	1.9
	20	400	2.6
50	10	250	1.6
	15	375	2.4
	20	500	3.2
60	10	300	1.9
	15	450	2.9
	20	600	3.9
70	10	350	2.3
	15	525	3.4
	20	700	4.5
80	10	400	2.6
	15	600	3.9
	20	800	5.2

While it is important to make a reasonable attempt at estimating the base deficit, volume expansion will increase renal perfusion, allowing the kidneys to establish an appropriate sodium balance.

If bicarbonate is not available, acetated Ringers (Normosol R) or lactated Ringers (Hartmann's) are a poor second choice to correct acidosis, but do have mild alkalisating ability. These solutions are not suitable for a seriously acidotic calf, but are suitable for maintenance once the base deficit is corrected.

### 3. Determining the volume of IV fluid required

#### a) Large volume isotonic solutions

The volume of isotonic solution required is calculated as follows:

*Volume required (L) = estimated % dehydrated (see table 8) x body weight (kg)*

Isotonic fluids may be administered IV at 40–80ml/kg/hour. Where higher flow rates are used, the patient should be monitored for fast or laboured respiration that may indicate pulmonary oedema.

#### b) Hypertonic fluids

Intestinal loss of fluid, sodium, chloride, potassium and bicarbonate results in hypotonic plasma and extracellular fluid. Consequently, fluid moves from the extracellular fluid into cells, exacerbating hypovolaemic shock. Treatment with hypertonic saline reverses this flow and when used with an isotonic oral electrolyte solution, the osmotic gradient favours movement from the gastro-intestinal tract to the plasma.

Hypertonic saline may be administered at 4ml/kg over four minutes **concurrently with an isotonic oral electrolyte solution** (1L/10kg). Severely acidotic, collapsed calves should be treated with isotonic bicarbonate prior to administration of hypertonic saline. The oral electrolyte solution must be isotonic, with alkalisating ability being less important. Where acidosis is less severe a **strongly alkalisating isotonic** oral electrolyte solution may be used without IV bicarbonate. The oral fluid should ideally be given before the IV hypertonic saline, due

to the rapid expansion of the plasma, however administration of the hypertonic saline first may temporarily resuscitate the calf and allow it to suckle.

Care should be taken to remain in the vein when administering hypertonic solutions, as tissue damage will result from perivascular administration.

### 4. Provide additional energy

100ml of 50% dextrose or 40% glucose should be added to each 1L of isotonic solution to prevent hypoglycaemia in cachectic, hypothermic calves. Where hypertonic saline is administered to resuscitate calves, prepare the isotonic bicarbonate solution using 5% glucose.

### 5. Determine ongoing maintenance requirements

Following correction of fluid deficits, calculate the fluid volume required to provide maintenance and replace ongoing losses for the next 24 hours.

In most situations this can be provided by an oral electrolyte solution (see table 11).

- If large volume fluids have been used, the volume required is that for 0% dehydration.
- If hypertonic solutions have been used, the daily volume required (including that administered with the hypertonic solution) is the **total** to correct the deficit and provide for maintenance and loss.

Assuming the base deficit is corrected, any oral electrolyte solution that meets the specifications on page 24 may be used. If intravenous bicarbonate has not been used to correct the base deficit, give a bicarbonate containing oral electrolyte solution.

Mildly alkalisating isotonic IV fluids, acetated Ringers (Normosol R) or lactated Ringers (Hartmann's) can provide for maintenance and ongoing losses in particularly valuable calves, or when a calf is depressed and septicaemic. The rate of administration should incorporate the maintenance requirement (3ml/kg/hr) plus compensate for ongoing losses due to diarrhoea – an additional 40ml/hr (1L/day) to 250ml/hr (6L/day).

Table 13: Correction of dehydration and acidosis using IV fluids

Parameter	Large volume isotonic solutions	Hypertonic fluids
Volume required	estimated % dehydrated x body weight (kg)	4ml/kg BW <b>concurrently with an isotonic oral alkalisng electrolyte solution</b> (1L/10kg BW)
Composition	<ul style="list-style-type: none"> <li>Normal saline with added bicarbonate</li> <li>Isotonic bicarbonate followed by acetated Ringers/lactated Ringers (Hartmann's)</li> </ul>	Hypertonic saline (7.2% saline) preceded by isotonic bicarbonate for severe acidosis
Flow rate	40–80ml/kg/hour	Give over four minutes
Subsequent oral fluids required	Assume IV fluids will correct dehydration and provide for maintenance and continued fluid loss	Total volume must correct dehydration and provide for maintenance and continued fluid loss
Provide energy	Add 100ml 50% dextrose or 40% glucose/litre isotonic solution	Make up isotonic bicarbonate solution with 5% glucose or give IV glucose separately if calf is cachectic or hypothermic

## Administration of intravenous fluids

A catheter can be placed in either the jugular or auricular vein. If the calf is collapsed and comatose and a vein cannot be raised, intraosseous administration may be required.

### Jugular vein

Catheterise with a 14–18g, 2.5–7.5cm catheter. The catheter can be stitched in place or positioned with cyanoacrylate ('superglue' or tissue adhesive). Once the IV line is connected secure with more tape and cyanoacrylate, together with a bandage around the neck. A wide bore extension tube will allow for rapid flow rates and, where necessary, this can be connected to the catheter with an extension tube.

If the calf is collapsed and the vein hard to localise, suspend the calf upside down to distend the jugular veins. The calf's neck should be clipped and prepared prior to inversion and the calf laid flat as soon as the catheter is placed. It may be necessary to cut down on to the vein.

Hypertonic fluids can generally be administered through a 16g 1½ inch needle placed in the jugular vein, however a catheter should be used if the calf is particularly mobile.

### Intraosseous administration

If the calf is comatose and a vein cannot be raised, fluids may be administered intraosseously until perfusion is sufficient for placement of an IV catheter. Shave an area 2.5cm<sup>2</sup> over the proximal humerus or femur. Aseptically prepare the site and insert a 14g 1½ inch needle into the centre of the bone longitudinal to its length. The bone is soft and the needle can be 'drilled' in. The needle will contain a core of bone so it should be removed carefully, observing the position, and a second 14g 1½ inch needle placed in the same hole. Attach a syringe containing 50ml of saline and inject to establish flow, then attach a 1L bag of isotonic fluids and run in as fast as possible. The preferred solution would be 1.3% sodium bicarbonate, although saline or lactated Ringers could be used if bicarbonate is not available. After 1L has been administered it is usually possible to find the jugular, but if not a second litre may be administered. Once the calf regains consciousness, this technique is difficult to maintain as movement makes it hard to keep the needle clean and in place. Calves requiring intraosseous fluid therapy should be treated with broad spectrum antimicrobials.

### *Auricular*

This vein runs on the outer surface of the pinna and can be located by placing a rubber band or similar tourniquet around the base of the ear. Warming the ear will also facilitate localisation. A 22g 1<sup>1</sup>/<sub>2</sub> inch catheter can be secured with a suture or cyanoacrylate, attached to a giving set, with the ear bandaged to the head. It is useful to place a wad of bandage or the inner cardboard tube from the centre of a bandage on the inside of the head when bandaging. The auricular vein is more difficult to maintain over the longer term than the jugular vein.

### *Subcutaneous and intraperitoneal administration*

Administration of fluids subcutaneously is only appropriate for maintenance or mild dehydration as severe peripheral vasoconstriction in calves >8% dehydrated will restrict fluid absorption. Consequently there is no advantage over oral administration. Give no more than 500ml at any one site. Fluids must be sterile, isotonic, warm and should not contain glucose as this may lead to abscessation. Administer high on the neck or thorax with a fast drip rate to allow more even distribution into the subcutaneous space.

Fluids are absorbed more rapidly from the intraperitoneal route compared to subcutaneous administration, but this is not recommended due to the potential for peritonitis. If it has to be used, the fluid must be isotonic and balanced to prevent fluid and electrolyte imbalances.

## **Antimicrobials**

Use of antimicrobial therapy in calves with diarrhoea should be risk based. The underlying aetiology in the majority of NCD outbreaks is likely to be viral or protozoal. There is no place for indiscriminate use of antibiotics in every slightly dehydrated scouring calf, especially those willing to suckle, unless a bacterial origin is known.

Treatment of scouring calves with antibiotics should be considered when:

1. Diagnostic investigation indicates NCD is caused by a bacterial pathogen
2. Calves have or are at high risk of bacteraemia and sepsis

## **Calves at high risk of bacteraemia and sepsis**

Calves with diarrhoea often have small intestinal overgrowth with *E. coli*, regardless of the inciting cause. Approximately 30% of systemically ill calves with FPT are bacteraemic, as are 8% of systemically ill calves that do not have FPT. *E. coli* is the most common bacterial isolate in these cases. Rapid recognition and treatment of sepsis improves the likelihood of a successful outcome.

Prophylactic antibiotics should be considered:

- if calves are sick enough to require repeat fluid therapy (see figure 1), especially in herds with a history of FPT
- if calves are pyrexia, have scleral injection or fibrin clots in the anterior chamber
- if there is blood in the faeces
- if calves are treated with IV fluids

Antibiotics should always be given when non-sterile fluids are used parenterally.

## **Selection of the appropriate antibiotic**

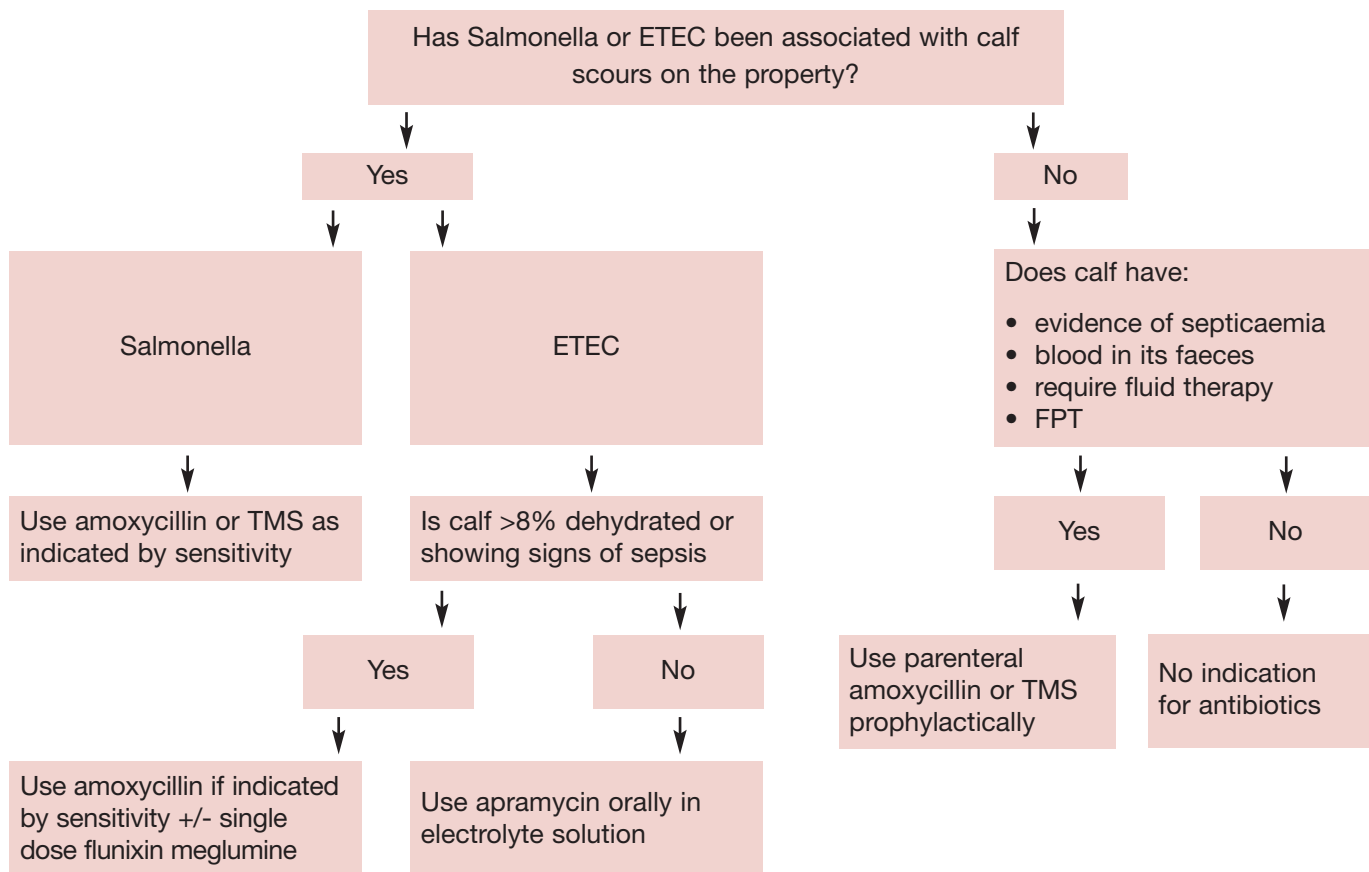
Bacteraemia and sepsis results from infiltration of intestinal bacteria through damaged gastrointestinal mucosa. Prophylactic antimicrobial treatment of calves at high risk of bacteraemia and sepsis should include an antimicrobial agent with gram negative and gram positive spectrum and be targeted against *E. coli*. Faecal bacterial culture and antimicrobial susceptibility testing is not recommended as a basis for selecting antibiotics to treat secondary infections as faecal bacterial populations do not accurately reflect small intestinal or blood bacterial populations. Antimicrobial efficacy is therefore best evaluated by the clinical response of a number of calves to treatment.

Parenteral administration of a broad-spectrum beta-lactam antimicrobial such as amoxicillin or trimethoprim-sulfonamide (TMS) is recommended for neonatal calves with diarrhoea and systemic illness. The bacteriostatic action and prevalence of antimicrobial resistance to tetracyclines and non-potentiated sulphonamides limits their effectiveness in septic neonates. Trimethoprim is rapidly metabolised in ruminating animals and TMS is not recommended in calves older than six weeks.

Sulfadimidine, sulfadiazine, streptomycin sulfate, dihydrostreptomycin sulfate, neomycin sulfate, and apramycin are labelled for oral administration for the treatment and prevention of calf scours. Orally administered apramycin has proven to be efficacious in field studies involving daily medication of calves at risk of diarrhoea, or in the early stages of the disease. The results of field and experimental trials with other oral antimicrobials have been equivocal. If calves are severely dehydrated, the absorption of oral antimicrobials is likely to be compromised and parenteral administration is recommended.

Treatment of a confirmed NCD-causing bacterial pathogen should be based on the results of sensitivity testing. Parenteral treatment is recommended for Salmonella. Amoxycillin and TMS have been shown to be efficacious against sensitive strains. ETEC is non-invasive and hence oral therapy is preferred, with apramycin being the drug of choice for sensitive isolates. This may be administered in oral electrolyte solutions. If the calf is sick enough to require IV fluids give parenteral antibiotics.

Figure 2: Decision tree for the appropriate use of antibiotics



### Treatment of protozoal infections

There are currently no effective therapeutic options for treatment of cryptosporidiosis in Australia.

Coccidiosis is uncommon in calves less than six weeks of age. Prophylactic options for beef calves are restricted to coccidiostat medicated pellets (monensin, lasalocid, amprolium or decoquinate) or water treatments (amprolium or sulfonamides). Therapeutic options include amprolium or sulfonamides such as sulfadimidine.

### Other treatments

A recent study on the use of flunixin meglumine in calves with NCD had equivocal results. However, flunixin does afford protection against endotoxaemia and short-term administration potentially has benefits in severely affected septicaemic calves. Vitamin B injections may be given to ensure adequate levels while nutritional intake is low. In selenium deficient areas, selenium injections should be given to calves with diarrhoea if the dams have not been supplemented. Vitamin A may have benefits in calves with cryptosporidia.



## Probiotics

A number of probiotic products are registered for the prevention and treatment of calf scours. Probiotics are reported to act by competition with pathogens for intestinal receptor sites, immune system stimulation, secretion of anti-microbial substances or competition with pathogens for intraluminal nutrients

The number of controlled clinical trials evaluating probiotic formulations in calves is limited, and where a significant difference has been shown, the probiotics have been fed prophylactically to hand reared calves, as opposed to being used as part of a treatment regime for calves already affected with diarrhoea.

## What do you do if calves are not responding to treatment?

Calves should be reassessed after initial fluid administration and therapy adjusted accordingly. An improvement in a calf's attitude indicates a favourable response, although diarrhoea is likely to persist. If no improvement in a calf's mentation is observed over 24 hours the following steps should be taken:

1. Perform a thorough physical examination. Look for congenital anomalies such as ventral septal defects or atresia ani; check for umbilical or joint infections, scleral injection or hypopyon that may indicate septicaemia. Look for strabismus, anisocoria and facial twitching that may indicate meningitis or other neurological derangement, abnormal sodium levels or hypoglycaemia.
2. Assess the current degree of dehydration and recalculate the fluid required. If the calf is still dehydrated it is likely that losses are higher than estimated and the daily requirement should be adjusted accordingly. IV therapy may be required.
3. Assume the calf has a bacteraemia and initiate appropriate antibiotic therapy (see page 29).
4. Review the possibility of acidosis: unless the calf is fully rehydrated and has been treated for severe acidosis, it may still be a problem. Calves that are >5% dehydrated should be treated for severe acidosis (base deficit 10–20mmol/L depending on age of calf) (see table 9).

5. If the calf is 5% dehydrated, but has not been treated for acidosis, treat for a 10mmol/L base deficit (see table 9).
6. If the calf is hydrated and has been adequately treated for acidosis, measure blood glucose and serum sodium concentrations. Measuring serum protein, albumin, fibrinogen and performing a complete blood count is useful to support a tentative diagnosis of bacteraemia. Biochemistry should always be considered where there is poor response to therapy at a herd level, to improve treatment regimes and rule out other potential diagnoses.

Hypoglycaemia is a common sequelae to withdrawal of milk for more than 48 hours, especially in cold weather. It may also be secondary to endotoxaemia. Affected calves are weak or recumbent, but appear to be normally hydrated, or minimally dehydrated. They are often emaciated, and can occasionally have neurological signs including petit mal or grand mal seizures, opisthotonus and coma. Moribund hypothermic calves should be given IV glucose prior to warming, as warming will increase glucose demands and may lead to seizures and death. Addition of glucose to rehydration fluids (up to 5%) is usually sufficient to maintain blood glucose. Cachectic calves with chronic scours are also prone to hypoglycaemia. Energy malnutrition contributes to their debilitated state and it is important to rapidly restore energy intake to ensure resolution.

Hyponatraemia and hypernatraemia are less common findings, but may result from improper mixing of oral electrolyte solutions. Hypernatraemia may result from the use of high sodium milk replacer or limited access to fresh water, and is often farm specific.

Poor response to treatment on a farm level should prompt further diagnostic investigation as it may reflect a specific management-induced problem, such as hypernatraemia, or pathogens that have not been initially identified.

## Supportive and ongoing care

Calves that are unable to suckle should be placed in a warm sheltered or cool shaded environment, depending on the climate. They should be evaluated for secondary problems such as hypoglycaemia and

hypothermia. Hypothermia occurs due to poor hydration, poor nutrition or poor adaptation, and can occur in all climates. Calves should have access to water at all times. Calf jackets should be considered for severely debilitated calves or in cold weather. Covered containers of hot water will also provide warmth. These calves will require ongoing treatment with electrolytes for 24 hours or longer.

Return calves to their dam when they are able to follow and nurse. Aim to return calves to their dams within 36 hours and give two to four litres of electrolytes immediately before release to avoid a sudden intake of a large volume of milk. Where calves are left with or returned to their dams, ensure easy access to water and appropriate shelter.

#### *Providing ongoing energy requirements*

Calves that are willing and able to suckle should be encouraged to do so, and electrolyte therapy adjusted accordingly. Encourage calves that have not received milk for over 24 hours to suckle. Assist them to stand and rub vigorously along the back and over the chest and neck. This simulates maternal caring responses and stimulates appetite. Unless collapsed, beef calves should not be separated from their dam for more than 36 hours. Calves too weak to suckle from their dam but willing to take a bottle should be fed 1L of milk/20kg twice a day between electrolyte feeds.

*Table 14: Maintenance energy requirement of calves*

<b>Weight of calf (kg)</b>	<b>MJ/day</b>	<b>Milk required (L) to supply maintenance requirements</b>
20	5.7	2.1
30	7.7	2.6
40	9.5	3.4
50	11.3	4.0
60	12.9	4.6
70	14.5	5.1
80	16.0	5.6

### **Record keeping**

Appropriate record keeping is needed to monitor the outbreak and to ascertain the effectiveness of treatment protocols, as well as to ensure that mandatory withholding periods are observed. The

If calves are unwilling to suckle after 36 hours, assess the treatment protocol. Ensure energy maintenance requirements are being met by electrolyte (and milk) intake (see table 14). Whole milk will provide approximately 2.8MJ/L. A high-energy electrolyte solution should be used if the calf will not suckle. Calves that do not receive enough energy for maintenance become cachectic and are at risk of hypoglycaemia. Energy malnutrition compromises host immunity and may lead to death in chronically affected calves. Calves should be rubbed and encouraged to stand every 12 hours, and their suckle reflex reassessed. Milk should be fed as soon as they are willing to suck.

Treatment of calves that are not willing to suck is problematical in a commercial situation, as it is difficult to provide sufficient energy using electrolyte solutions, and administration of large volumes of milk by oesophageal feeder will make calves uncomfortable and further inhibit willingness to suck. If calves are in extremely poor condition or not suckling within three to five days, it will be necessary to locate the calf and its dam where the calf can be fed with frequent small volumes of milk (initially ≤500ml) between electrolyte administrations.

Valuable calves may be provided with additional nutritional support through administration of glucose, amino acid and lipid solutions (total parenteral nutrition).

use of individual ear tags and a farm or treatment diary should be encouraged. Where this is not done, raddle, spray paint or a neck band should be used to identify treated calves.

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