ORIGINAL ARTICLES

Rearing and management of diarrhoea in calves to weaning

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Introduction

The production of beef weaners from dairy cows by dairy farmers or by professional rearers is now well established in New Zealand, and most of the increase in beef cattle numbers is occurring through increased retention of dairy-beef calves (Anon 1992a). Attracted to the potential extra income from dairy-beef, some dairy farmers have moved away from supplying their calves for slaughter when the calves are at least 4 days old and between 25 and 45 kg body weight. If high demand continues for dairy-beef calves, professional calf rearers may relax the traditional requirements of a minimum 35 kg at one week of age (Schouten 1989) and full Friesian or Friesian-cross colour (Buxton 1990).

Relaxing the selection criteria when purchasing calves for dairy-beef enterprises risks problems with calf diarrhoea in herds. Professional calf rearers need to institute a selection and management system that will reduce or prevent exposure of calves and maximise their resistance to enteric pathogens.

The first half of this review describes the principles of calf rearing from birth to weaning; the second half discusses the major enteropathogens and the management of neonatal calf diarrhoea.

Selection of Calves for Rearing

For the professional rearer, the source of the calves greatly influences the disease risks to be encountered. Some sources provide calves at higher risk of disease than others depending on, for example, (a) health and feeding management on the farm of origin, especially the provision of adequate, high-quality colostrum shortly after birth; (b) distance and mode of transport to the rearer; (c) whether the calves were purchased directly from the farm or through an agent; and (d) the amount of mixing of stock from different farms. Calves purchased directly from a dairy farm are often healthier than those purchased through sale yards or agents. This difference can be attributed to less exposure to pathogens, less commingling, direct delivery and often better nutritional care (Smith and Lynch 1991). The careful selection of calves that have been colostrum-fed in a clean environment are factors leading to success. A higher price for such calves can be a wise investment, and it also encourages the breeder to supply quality animals.

Intake of Colostrum

Colostrum and its feeding on the farm of origin are important. Efficiency of immunoglobulin absorption in the calf is reduced by 50% between birth and 12 hours of life (Stott *et al* 1979; Smith and Lynch 1991; White 1993). Newborn calves should be fed 10 to 15% of their body weight of colostrum within the first 12 hours of life (Bradley and Niilo 1984; Geene 1986a; Heath 1992a) and preferably within the first 6 hours. A 40 kg calf needs 4 to 5 litres. Calves raised intensively may require force-feeding by using an oesophageal feeder (Besser *et al* 1991; Radostits 1992). Concentration of colostral immunoglobulin is determined by maternal factors of age, parity, breed, nutritional status, length of dry period, premature milking, and udder infection status (Geene 1986b; Mohammed *et al* 1991). Colostral immunoglobulin concentration decreases rapidly with each milking after calving (Porter 1972): 50 to 60% of the original concentration after the first milking and only 30% after the second milking (Stott *et al* 1981). Acquisition of immunity by the calf can be compromised by failure of the calf to ingest or absorb colostrum (McGuire *et al* 1976; Johnston and Stewart 1986; Odde 1988; Aldridge *et al* 1992).

The use of a colostrometer to assess colostral quality before feeding has been advocated (Fleenor and Stott 1980; Heath 1992a), the specific gravity being an indication of immunoglobulin content. However, both temperature and fat content of colostrum influence the results, and colostrometers may overestimate the immunoglobulin concentration of colostrum (Mechor 1991). The poorer-quality colostrum (heifer and second and third-day colostrum) should be fed to calves older than one day of age, either straight or diluted with whole milk or milk replacer. It is a superb source of nutrients and cellular and non-specific immune factors; the latter produce local protection against gastro-intestinal pathogens (Logan *et al* 1977; Woode 1978; Fleenor and Stott 1980; Geene 1986c).

Handling and processing of colostrum also affects its quality. Colostrum should be collected and fed in a sanitary manner to avoid contamination. Excess colostrum is often pooled and stored for future use as a source of immunoglobulin. Only colostrum from the first milking is suitable for storage; the secretions from second and subsequent milkings contain relatively little immunoglobulin (White 1993). Colostrum is relatively labile and can not be kept for more than a few days at 2 to 8°C without significant decrease in immunoglobulin concentration (White 1993). It is very stable when frozen and can maintain its quality for over one year at -20°C (Roy 1990; Heath 1992a). Thawing and warming colostrum by microwave have been commonly believed to denature immunoglobulin, but Jones *et al* (1987) and Haines *et al* (1992) found no difference between fresh and microwave-thawed colostral immunoglobulin composition. Colostrum should not be heated over 56°C for any length of time to prevent breakdown of the constituent proteins. Unused colostrum should not be re-frozen but kept stored at 2 to 8°C and discarded if not used within 48 hours. Commercially drying colostrum has many attractions (Zaremba *et al* 1993; Todd *et al* 1993), but practical constraints have meant this practice is not widespread (Haines *et al* 1990; White 1993).

Colostrum-deprived calves usually exhibit increased disease incidence and mortality (Vermunt 1993). Immunoglobulin screening of incoming neonatal calves is a valuable method for determining susceptibility to some diseases. Successful absorption of immunoglobulins from colostrum is assessed by determining the concentrations of total protein (Reid and Martinez 1975; Naylor and Kronfeld 1977; Braun and Tennant 1983; Van Keulen *et al* 1985; Perino *et al* 1993) or gamma-glutamyl-transferase (GGT) (Thompson and Pauli 1981; Perino *et al* 1993) in serum of calves. Normal values for total serum protein are greater

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than 4.2 g/dL and for GGT greater than 200 IU/L (Perino *et al* 1993). Other tests of passive immunity include the zinc sulphate turbidity test (McEwan *et al* 1970), radioimmunoassay (Naylor and Kronfeld 1977), electrophoresis (Banks and McGuire 1989), glutaraldehyde coagulation (Tennant *et al* 1979; Van Keulen *et al* 1984) and sodium sulphite turbidity test (Pfeiffer and McGuire 1977). Calves can be tested for passive immune status when they are 24 hours to 7 days old, and any of these tests is best performed before calves are purchased.

Calf Management

Upon introduction, calves should be quarantined in clean pens that are not to be occupied by calves on the home farm for a period of 7 days. This is considered the maximum incubation period for the common enteropathogens (Jerrett 1991). Calves that are depressed and show signs of illness, such as a wet, swollen or painful navel, arthritis or diarrhoea should not be purchased. Animals that have an unthrifty, emaciated appearance should also be rejected. Ideally, calves should weigh 35 kg or more at 4 to 5 days of age (Schouten 1989; Smith and Lynch 1991).

On arrival, calves should be weighed, ear-tagged and evaluated for body temperature, vigour and body condition. These data and the source (in Australia, tail tags bear a code that facilitates this) should be recorded. Calves that have been transported long distances will be tired and dehydrated on arrival (Atkinson 1992) and each will benefit from the provision of 2 to 3 litres of water and electrolytes. Milk feeding should not be started until 12 hours after arrival. During rearing, groups should not exceed 10 to 15 animals (Schouten 1989; Heath 1992a). Only calves of similar age should be penned together. Separation reduces exposure to multiple pathogens, several of which are age specific for the time at which they cause disease (Naylor 1990). Solid partitions of sufficient height should separate pens to prevent direct contact between calves in different pens, thereby reducing exposure to pathogens.

Calves should be housed on slats over concrete or on packed sawdust or woodshavings from timber that has not been chemically treated. The latter two bedding materials must be replenished regularly and used on a thick base of gravel, scoria or pumice to provide good drainage. The minimum floor area must be 1 m^2 per calf (Schouten 1989); 2 to 3 m^2 would be ideal. Calves should be kept in open-fronted pens with northern exposure in New Zealand and Australia. These pens must be dry and free of draught and measure twice as deep as wide (Schouten 1989). There should be adequate roof overhang to ensure pens do not get wet when it rains.

During dry and sunny weather conditions, calves should have access to pasture. Paddocks in which calves are reared should be vacated and spelled between batches of calves to limit the build-up of pathogens, particularly coccidia.

Calves that develop signs of disease should be isolated and treated. 'Navel suckers' and 'urine drinkers' should also be penned on their own.

Rotavirus and oocysts of coccidia and cryptosporidia are highly resistant to environmental conditions and many disinfectants. Rotavirus resists temperatures of 60°C for 1 hour and survives 9 months in faecal material stored at room temperature (Jerrett 1991). The virus is destroyed by contact with 5% lysol or 10% formol-saline for 2 hours but not by contact with sodium hypochlorite or iodophors (Snodgrass and Herring 1977). Sodium hypochlorite, sodium hydroxide and aldehyde-based products are ineffective against oocysts (Current 1983). The use of a steam jet under pressure has been recommended as the best method of disinfection (Aurich *et al* 1990; Angus 1993). However, to destroy oocysts, a temperature above 65°C has to be maintained for at least 30 minutes (Chermette and Boufassa-Ouzrot 1988). Oocysts may also be killed by contact for 18 hours with 5% ammonia, 10% formol-saline (Campbell *et al* 1982), or 3% hydrogen peroxide (Angus 1993). Exposure to sunlight for at least 4 to 8 hours and humidity less than 25% is still the best method to kill both sporulated and unsporulated oocysts (Gregory *et al* 1982; Yvore 1984; Schillhorn van Veen 1986). In general, solid surfaces and utensils should be scrubbed vigorously with hot water and a detergent.

Disbudding calves should be performed before weaning (Anon 1992b, c), ideally, as soon as the horn buds can be felt, which is usually at 2 to 3 weeks of age. Acceptable methods of dehorning include the use of a hot iron, electric or gas calf dehorner (Anon 1992b, c) or surgical instruments such as a scoop (Barnes' dehorner) or a trephine (Robert's disbudder)(Anon 1992c).

Treatment for control of internal and external parasites is generally not necessary until weaning, but if calves have early access to pasture during the milk-feeding period it may be required.

Calf Feeding

Proper feeding of introduced calves is of paramount importance. They can be fed whole milk, stored colostrum or milk replacer. An amount in litres equal to 5% of body weight per feeding should be given twice daily (Crowley *et al* 1989) for at least the first three weeks, then once daily until weaning.

Calves can be fed from machine dispensers or individual buckets (with or without a teat attached), multiple teat feeders (calfaterias) and 200 L drums fitted with a number of teats with non-returning valves. For feeding *ad libitum* through teats connected to a milk source, less teats than calves are required. These systems use more milk than restricted-intake systems (Thickett 1991).

Digestion

When milk passes into the abomasum, rennin and hydrochloric acid coagulate milk casein to form a curd. Clotting occurs within 5 to 6 minutes of ingestion of the milk (Medina *et al* 1983). It allows slow digestion of the casein in the abomasum.

Use of Whole Milk

Professional rearers may opt to collect whole milk from a dairy farm only once every few days. Milk can be preserved for storage during warm weather by adding 2.5 g of organic acids (citric, fumaric, malic and formic) (Fallon and Hart 1980) or 5 mL of 30% by volume of hydrogen peroxide per litre of milk (Dawson *et al* 1982).

The average crossbred calf is fed an average of 5 L of milk daily for 8 weeks, reaching a weaning weight of 80 kg.

Using waste milk containing antibiotics as a feed for calves appeals to many dairy farmers, but the antibiotics may lead to the presence of tissue residues. If antibiotic-containing milk is left to ferment naturally, a reduction of the antibiotic concentrations occurs through degradation (Keys *et al* 1979). However, such milk has poor palatability and rejection rates are high (Wray *et al* 1990). Feeding unfermented, antibiotic-containing milk to calves results in poor growth rates but does not select for microbial resistance in the calves' enteric flora (Wray *et al* 1990).

Use of Stored Colostrum

A surplus of colostrum may be available during the concentrated calving season common to the New Zealand and Victorian seasonal dairy industries. Colostrum can be stored at ambient temperature for periods ranging from 4 days in New South Wales to 2 months in New Zealand (Donnelly *et al* 1980). Bacteria ferment the lactose to lactic acid, which preserves the colostrum. Colostrum preserved in this manner has similar nutritional value to

fresh whole milk (Donnelly *et al* 1980) and may be fed at similar rates.

Fermented colostrum can not be expected to give a newborn calf adequate passive immunity (White 1993). This is due to the dilution of antibodies from the second and later milkings in the colostral pool (Besser *et al* 1983; Aldridge *et al* 1992) and to some destruction of the antibodies during fermentation (Maidment 1982). Feeding of fermented colostrum to calves up to 3 weeks of age provides a source of lactoglobulins in the intestinal tract and reduces the incidence of neonatal diarrhoea due to rotavirus and coronavirus (Geene 1986c; Radostits 1992).

Colostrum should be stored in a stainless steel vat or plastic container and out of direct sunlight. If the outside temperature is high, the fermented colostrum spoils. The addition of a preservative, such as propionic or acetic acid at 1% of the volume, will prevent spoilage. The colostrum needs to be stirred daily to prevent the build-up of a surface crust. Fresh colostrum can be added to the 'pool', which must be mixed thoroughly. Milk that contains antibiotics should not be added. Colostrum should not be stored for longer than 4 to 5 weeks; there is a 50% decrease in its protein concentration after 28 days (White 1993).

Use of Milk Powder/ Milk Replacer

A high-quality milk replacer should contain 20-22% crude protein, 10-25% dietary fat, 0.25-0.50% fibre, and less than 12% minerals or ash (Medina *et al* 1983; Smith and Lynch 1991). The presence of fibre and a high mineral content indicates the use of non-milk ingredients (Medina *et al* 1983), in other words, some or all of the protein is of non-milk origin. Whole milk and colostrum are rich in vitamins A, D and some B vitamins (Smith and Lynch 1991; White 1993). Vitamins A, D, E, and B complex and antioxidants are included in most milk replacers to prevent rancidity (Roy 1980), often in excess of the calf's daily requirements (Thickett 1991).

The clotting behaviour of reconstituted milk can be predicted by using an in-vitro rennet test. In this test, 5 mL of rennet are mixed with 500 mL of reconstituted milk at a temperature of about 40°C. After 15 min, milk replacers containing a high proportion of (skim) milk powder form a good curd.

Many milk powders rely on protein derived from whey powder and non-milk sources, principally processed soyabeans and fish protein hydrolysates. These products do not form a curd and leave the abomasum rapidly after feeding (Cruywagen *et al* 1990), but nevertheless are adequately digested in the small intestine (Steenkramer 1982; Petit *et al* 1987; Thickett 1991). From a physiological or calf health point of view, the lack of clotting of a milk replacer is of no concern. Based on average rates of gain, the nutritive value of the vegetable-based powders is lower than that of the milk-based powders (Wilson *et al* 1991). This may be due to the soyameal, which can contain several anti-nutritional substances such as anti-trypsin factor, haemagglutinins and antigens (Stobo 1983; Tomkins and Jaster 1991). These powders should be fed only to calves older than 3 to 4 weeks, because young calves can not digest vegetable proteins as efficiently as milk proteins (Zafrira *et al* 1972; Roy *et al* 1977; Moran *et al* 1988). Alternating between 'real milk' and 'non-milk' powders during the milk-feeding period may lead to digestive or nutritional scours because of imbalances in the composition of protein and energy (Heath 1992a).

Some milk powders contain coccidiostats, such as decoquinate, or ionophores, such as monensin or lasalocid. When medication with coccidiostats is administered, the animals are less able to develop immunity against coccidiosis (Kirkpatrick and Farrell 1984). Therefore, calves may still be susceptible to coccidiosis after weaning. However, ionophores do not completely suppress parasite development, allowing a proportion of parasites to develop (Taylor 1992). This incomplete control of coccidia development stimulates production of immunity in the host (Jeffers 1989). Ionophores kill coccidia in the earliest asexual stages of its life cycle (Tyler *et al* 1992). Consequently, ionophores must be included in the ration before or shortly after the ingestion of infective oocysts if the agents are to have prophylactic value. Ionophores are of limited value in the treatment of clinical coccidiosis (Schillhorn van Veen 1986). Inclusion of ionophores in milk powders fed to calves before weaning increases feed intake and improves growth of young calves (Sinks *et al* 1992; Heath 1992b). This probably occurs through modification of the environment of the developing rumen and increasing ruminal fermentation (Elliott 1993) as well as control of coccidia (Sinks *et al* 1992). Monensin and lasalocid are not effective in the prevention of cryptosporidiosis (Moon 1982; Lindsay and Blagburn 1991; Snodgrass 1993).

Low concentrations of antibacterial agents, such as zinc bacitracin, are added to most milk replacers to improve growth rates of calves by modifying the microbial environment in the rumen (Medina *et al* 1983). These concentrations are about 10% of the therapeutic dose against bacterial enteropathogens and are unlikely to have any disease preventive benefits (Medina *et al* 1983).

The recommendations of the manufacturer for reconstitution and mixing should be followed to avoid digestive scours and poor weight gains in calves. Most products use a rate of about 125 g of powder per litre of water.

The ideal milk replacer needs to be palatable, easily reconstituted (preferably in cold water), remain in suspension when mixed with water, converted efficiently into body tissues and not cause scouring or any other health problem. After palatability, convenience and feeding value, the next most important feature of a milk replacer is its cost relative to whole milk. Based on the 1993 value of $ 5.20 (NZ$ 6.50) per kg of butterfat, milk replacer at $ 2.90 (NZ$ 3.60) per kg of powder is at least one and one-half times more expensive than whole milk (4% fat). The ease of handling and superior quality expectations are probably important factors when farmers make decisions on which 'milk' product to use. Vegetable-based products generally cost 10% less than milk-based powders (Wilson *et al* 1991).

Use of Forage and Dry Feed Supplementation Before Weaning

Allowing a calf access to pasture or high-quality hay from one week of age encourages early development of the rumen. A high-quality, palatable starter ration of coarsely ground meal or pellets should be introduced when calves are one week old, starting with 0.3 kg per calf per day and finishing with about 1.5 to 2 kg per day with weekly increments of 0.2 kg. One kg of pellets provides the same energy as 3.3 litres of whole milk.

The meal should be replenished regularly so it does not get stale. Contamination of the concentrate ration with urine and faeces should be prevented by mounting the feeding troughs against a wall or gate at 50 cm above ground level.

Fresh water must be available from the beginning of the rearing period. Initially, calves may drink very little, but their water intake increases by 5 litres for every kilo of dry feed intake. The iron content of the water should be examined; high iron intake causes scouring (Miller *et al* 1988). The maximum tolerable concentration of dietary iron is 1000 ppm for cattle (National Research Council 1980).

The growth rate of calves should be monitored by regular weighing during the entire 2 months before weaning. Calves that grow poorly during this period need individual attention to match the growth of their herd mates (Donovan and Braun 1987). Sub-optimum growth during the milk-feeding period can be the result of: (a) inadequate quantity of milk, milk replacer, or calf

meal, (b) poor-quality milk replacer, (c) poor-quality starter ration, (d) premature weaning, or (e) diseases of the gastro-intestinal or respiratory tract.

Weaning

The objective should be an average gain of 0.7 to 0.75 kg per day over the entire milk-feeding period. To be weaned at 8 weeks, a calf should be double its birth weight and able to deal with high quality forages. Most calves (bull beef, dairy beef and dairy heifer replacements) are weaned at 8 weeks of age or about 80 kg of body weight. Alternatively, calves may be weaned at 6 weeks of age or 70 kg of body weight.

Calves that are consuming more than 1 kg of starter ration daily and have reached target weaning weights are generally ready for weaning (Smith and Lynch 1991; Thickett 1991). The milk should not be diluted to prepare calves for weaning; it is better to feed less frequently or smaller amounts. This encourages calves to eat pasture and dry feed. The feeding of meal should continue at a rate of at least 0.5 kg per calf per day for 3 to 4 weeks after weaning in order to reduce the growth setback often associated with weaning.

Calf Diarrhoea

Calf diarrhoea predominates during the first 30 to 50 days of life. Clinical differentiation of the causes of neonatal diarrhoea is difficult, because there are many potential pathogens that cause similar clinical signs. Often more than one causative agent is present.

Non-infectious or dietary diarrhoea in calves is mild and transient and is often blamed wrongly as an important cause of diarrhoea (Jerrett 1985). Dietary scours may exacerbate diarrhoea caused by infectious agents. Nutritional scours can result from feeding diluted milk that does not form a stable clot in the calf's abomasum, overfeeding, underfeeding during cold and inclement weather, irregular feeding practices, the incorrect mixing rate or composition of milk replacer, feeding of milk replacer containing denatured proteins, and a change of milk quality in nursing calves or calves on whole milk (Vermunt 1993).

Causes, Epidemiology and Diagnosis

The main infectious causes of neonatal calf diarrhoea are pathogens that are endemic in most calf rearing establishments: rotavirus, coronavirus, enterotoxigenic *Escherichia coli*, *Salmonella* species, and cryptosporidia (Acres *et al* 1977; Tzipori 1981; Jerrett 1982, 1985; Snodgrass *et al* 1986). Coccidiosis is generally not a problem in calves less than one month old.

Cryptosporidia and *S typhimurium* are zoonotic pathogens. People are therefore at risk of contracting disease when in direct or indirect contact with infected calves, so strict hygiene should be practised.

Rotavirus — Rotavirus infection occurs early in a calf's life, usually from 2 days to 3 weeks of age (Horner 1984; Jerrett 1985). Virus replication occurs in the surface epithelial cells of predominantly the lower small intestine. Infected cells die, slough and are replaced by immature cells. These changes result in stunting and fusion of adjacent villi. There is decreased absorption and leakage of fluid due to intestinal damage. Faeces are often mucoid and yellow.

Rotavirus is shed in large numbers in the faeces of infected animals (as many as 10^{10} virus particles/g faeces) (Radostits 1992), and virus particles are often detectable by direct electron microscopic examination of a clarified faecal suspension (Snodgrass *et al* 1976). Other methods for diagnosis of rotavirus infections in calves include direct electron microscopy and fluorescent antibody technique on intestinal sections, enzyme-linked immunosorbent assay (ELISA) and dot immunobinding assay tests on faeces, or immuno- and counter immuno-electro-osmophoresis, complement fixation and latex slide agglutination tests (Chauhan and Singh 1992; Garcia-Sanchez *et al* 1993).

Rotavirus is endemic on almost all cattle farms (Snodgrass *et al* 1986) and is excreted intermittently by a significant proportion of the normal calf and adult cow populations (Radostits 1992). These carrier animals are probably the main source of infection for new batches of calves.

Coronavirus — Coronavirus is regarded as an important pathogen in the northern hemisphere causing infection of both the upper respiratory and lower intestinal tracts in young calves (Moon *et al* 1978; Clark 1993). Enteric infections typically occur in calves between 1 and 2 weeks of age (Langpap *et al* 1979; Reynolds *et al* 1986). Although infection with coronavirus occurs in Australia (Jerrett 1991), it is of little importance in neonatal calf diarrhoea.

In the enteric form, clinical signs in affected calves are indistinguishable from those of rotavirus infection (Clark 1993). Virus replication in the epithelial cells starts in the proximal small intestine and spreads throughout the small and large intestines (Mebus *et al* 1975). Diagnosis is usually by electron microscopy on faeces (Stair *et al* 1972). Other detection methods include ELISA test and immuno-electron microscopy techniques.

The virus is shed intermittently by normal cows and shedding peaks during the winter months and at parturition (Collins *et al* 1987). Calves develop an age resistance against the viral diarrhoeas at about 3 weeks of age (Radostits 1992).

E coli — Neonatal *E coli* enterotoxicosis (mainly K99 pilus antigen (Isaacson *et al* 1978)) occurs in calves less than 6 days old and mostly when only 1 to 2 days old (Bulgin *et al* 1982; Jerrett 1985; Snodgrass *et al* 1986). After that age, calves develop a physiological resistance to the organism (Acres 1985). Bacteria of these fimbriated, enterotoxigenic *E coli* (ETEC) strains adhere to the mucosal surface of the small intestine, colonise the gut, and elaborate thermostable enterotoxin resulting in extremely watery diarrhoea through increased secretion from enterocytes (Belton 1984). The K99 pilus antigen has an almost complete correlation with enterotoxin production (Sherwood *et al* 1983). Affected calves rapidly become dehydrated. The ETEC cause minimal injury to the intestinal epithelium.

Diagnosis is by ELISA test on faeces or the presence of predominant or heavy pure growths of *E coli* in the duodenum or anterior ileum, a site where it is usually not present in healthy calves. Antigen typing of K99 by slide agglutination test is done routinely by few laboratories. Certain *E coli* strains are regarded as enterotoxic and have an ability to adhere to surface enterocytes and damage them. These attaching and effacing *E coli* (AEEC) are non-K99 bearing pathogens and are diagnosed by histopathology of fixed intestinal tissue.

Infected animals are the main reservoir for *E coli*, and their faeces are the major source of environment contamination with bacteria (Radostits 1992). Passage of the *E coli* through animals causes a 'multiplier effect'. Diarrhoeic calves are the most prolific multipliers, because they often pass large quantities of liquid faeces containing billions of ETEC per gram of faeces (Radostits 1992). Recovered calves may shed bacteria for several months and such carrier animals are one of the main causes of natural outbreaks when introduced to an uninfected herd.

Salmonella species — Salmonellosis due to *S typhimurium* infection is mainly a problem in calves of 1 to 7 weeks of age (Williams 1980). *S typhimurium* bacteria secrete an enterotoxin and invade and damage the intestinal mucosa; the diarrhoea is mainly hypersecretive. Faeces are watery, contain fibrin and may smell foul. Calves are often severely depressed and have fever. Most deaths in *Salmonella* infections are associated with septicaemia (Jerrett 1985).

Diagnosis is by means of faecal sampling and, after enrichment, culture on special media such as brilliant green agar (Jones *et al* 1983; Lance *et al* 1992). Any growth of *Salmonella* species is significant (Heath 1992b).

S typhimurium has a wide host range, but most infections in calves are derived from other cattle (Hughes *et al* 1971). Infection is often introduced to calf rearing units by infected but clinically normal calves purchased at saleyards (Wray and Sojka 1977; McLaren and Wray 1991). Occasional calves are asymptomatic carriers of *Salmonella* species (Hinton *et al* 1983; Lance *et al* 1992). *Salmonella* species can persist in calf rearing units for up to 2 years (McLaren and Wray 1991).

S dublin is of minor importance as a cause of calf diarrhoea in New Zealand and Australia.

Cryptosporidia — The enteric form of cryptosporidiosis in calves is due to *C parvum* infestation (Holland *et al* 1992; Corwin 1992) and occurs mainly during the first three weeks of life (O'Donoghue 1985). These protozoa multiply in the same kind of cells that rotavirus and coronavirus infect, but usually also in the large intestine. Cryptosporidia develop at the cell surface rather than within the host cell cytoplasm (Kirkpatrick 1988). Damage to the mucosal cells of the intestinal tract causes nutrients to be incompletely absorbed. Many affected calves have a slight fever (Anderson and Bulgin 1991), but appetites are often good (Corwin 1992). Faeces are generally profuse, watery, non-haemorrhagic and coloured greenish-yellow. The sporulated oocysts shed in the faeces are immediately infective (Anderson 1981).

Diagnosis is by means of a modified Ziehl-Neelsen carbol-fuchsin stain of a faecal smear or a coverslip flotation method using a sucrose solution (Casemore *et al* 1985). The latter is more sensitive. The sporulated oocysts can be overlooked easily because they are very small. Histopathology of samples of intestine from an affected calf shows villous atrophy. Lesions occur from the beginning of the ileum through to the distal colon; the distal ileum is most consistently infected (Kirkpatrick and Farrell 1984; Corwin 1992).

Cryptosporidia are found in normal healthy calves (McSporran 1992; Radostits 1992), but can cause overwhelming disease in calves that are immunosuppressed or developing an immune response (Corwin 1992).

Coccidia — Intestinal coccidiosis in calves is caused by *Eimeria* species and involves the lower ileum, caecum and large intestine (Schillhorn van Veen 1986). Only sporulated oocysts are infective and sporulation times are influenced by temperature; *E zuernii* sporulates after 10 days at 12°C and 3 days at 20°C (Ernst *et al* 1984). The diarrhoeic faeces are often dark and haemorrhagic with threads of intestinal lining; tenesmus is also common. Intestinal damage and clinical signs can precede the shedding of oocysts in the faeces. Infection with coccidia is not always associated with overt signs of disease; many infections are clinically inapparent (Schillhorn van Veen 1986), but often result in reduced weight gain (Foreyt *et al* 1986; Hoblet *et al* 1989).

Diagnosis is by faecal examination for oocysts or histopathology of fixed gut samples (Stockdale 1977). Histopathology is a more specific test for diagnosing coccidiosis. *E bovis* and *E zuernii* are the most pathogenic of the cattle species (Schillhorn van Veen 1986) and can cause a very heavy coccidial infestation with severe catarrhal enteritis and death.

Assuming a 3-week prepatent period and manifestation of clinical signs in 3- to 6-weeks-old calves (Hoblet *et al* 1989), it is clear that infection occurs within the first few weeks of life. Calves ingest oocysts from contaminated pens, fittings and from infected faeces. Infection with coccidia reduces the immune function of calves and may cause increased morbidity and mortality from respiratory disease (Roth *et al* 1989).

Other, less frequently detected pathogens in faeces from diarrhoeic, neonatal calves are *Yersinia pseudotuberculosis* types I, II and III, *Y enterocolitica*, *Campylobacter jejuni*, *Clostridium perfringens* types B and C, and enteric viruses such as astrovirus, parvovirus, adenovirus, togavirus, the bovine viral diarrhoea virus, Breda virus and calicivirus-like agent. More research is required to define their pathogenicity and significance.

Pathophysiology

The common feature of neonatal calf diarrhoea is dehydration, which results whether the cause of the diarrhoea is infectious or nutritional. Rehydration by oral and/or parenteral means is the basis of treating calf diarrhoea.

In young calves, death caused by diarrhoea is mainly attributed to fluid losses and electrolyte imbalances and less to energy deficits (Groutides and Michell 1990) resulting from reduced intake, malabsorption and increased excretion of water, minerals and nutrients. Total faecal volume in diarrhoeic calves may be up to 40 times normal (Lewis and Philips 1972).

Dehydration is initially due to plasma loss, but later due to fluid loss from extra-vascular compartments (Roussel 1983).

Metabolic acidosis arises from an increased loss of bicarbonate in the faeces, an increased production of lactic acid in poorly perfused tissues and a reduction in renal excretion of hydrogen ions (H^+), due to decreased blood volume associated with dehydration (Booth and Naylor 1987).

Hyponatraemia is the result of the significant losses of sodium in the faeces (Fisher and de La Fuente 1972). Sodium absorption occurs independently or in an active co-transport mechanism with glucose or amino acid (glycine, alanine) at the mucosal cell surface (Nalin *et al* 1970; Patra *et al* 1989).

Hyperkalaemia occurs despite a significant loss of potassium ions (K^+) in the faeces, which result in whole body deficits of potassium. The condition is the result of acidosis and compromised renal function (Michell 1993), which causes a shift in potassium from the intracellular to the extracelluar fluid. H^+ ions diffuse into the intracellular fluid compartment and displace K^+ ions from the cells into the extracellular fluid compartment to maintain electro-neutrality.

Hypoglycaemia in scouring calves should be treated with glucose in replacement fluids. Glucose is also necessary to overcome the flux of water and electrolytes into the gut. It enhances Na^+ ion absorption from the intestines when given orally and shifts K^+ ions into the intracellular fluids when administered intravenously (Roussel 1983). Insulin release stimulated by oral glucose assists in moving potassium back into the cells (Fisher and de La Fuente 1972).

Fluid Therapy in Neonatal Calf Diarrhoea

Selecting an Electrolyte Therapy

Fluids and electrolytes lost in diarrhoeic faeces should be replaced and the normal electrolyte balance re-established. This is best achieved by fluid replacement therapy, which may be accompanied by withholding milk (Bywater 1980; Booth and Naylor 1987; Mulville 1991). There are various opinions regarding the best concentration of electrolytes, buffers, and energy sources in oral rehydration solutions. Optimum oral rehydration therapy uses a solution that contains sodium, chloride and potassium together with a base and energy source (Michell 1988, 1989).

An electrolyte formula should contain an alkalising agent such as bicarbonate, citrate, acetate, gluconate or lactate (Naylor *et al* 1990; Michell *et al* 1992; Michell 1993); the last three are bicarbonate ion (HCO_3-) precursors. The presence of glycine in

oral rehydration solutions is less important (Jerrett 1985; Michell 1993), even though it is very palatable to calves.

Oral bicarbonate alleviates metabolic acidosis, but causes a rapid and sustained increase of the abomasal pH (Simmons and Bywater 1991). This may delay or decrease the rate of curd formation and prolong the diarrhoea, and reduce secretion of gastric acid allowing pathogenic bacteria to reach the small intestine (Bywater 1980; Heath *et al* 1989).

Acetate and citrate are both potential alkalis, and when absorbed are metabolised within the liver and other tissues to CO_2 and water (Naylor and Forsyth 1986), combining with H^+ in the process. Replacement of bicarbonate with acetate or citrate gives oral rehydration solutions an alkalising activity without altering the gastric pH. Oral rehydration solutions with a lower pH are more palatable to calves and less prone to bacterial growth during storage (Simmons 1984). Oral citrate, however, binds with calcium in the milk and may result in a prolonged clotting time (Naylor 1991).

Oral fluids can be given by bottle, bucket, oesophageal feeder or stomach tube and should be at body temperature and based on an isotonic, equimolar mixture of sodium and glucose (Michell *et al* 1992). Hypertonic solutions have been used successfully. However, such solutions can damage normal mucosa (Kameda *et al* 1986). Electrolyte solutions should contain the maximum amount of glucose possible while maintaining isotonicity (Bywater 1977). Hypertonic solutions in the intestine can cause hypernatraemia by promoting withdrawal of water from the extra-cellular fluid (Michell 1993). In cases of villous atrophy as occurs in diarrhoea secondary to rotavirus, coronavirus or cryptosporidia infection, glucose overload may lead to aggravation of the diarrhoea by increased osmosis. Hypertonic solutions should be used with caution. Energy input can be increased by limited milk feeding in addition to oral rehydration solutions.

Formulae for effective oral electrolyte solutions are:

(1)

sodium chloride	20 g
sodium bicarbonate	20 g
potassium chloride	6 g
glucose	200 g
water	4 L

(2)

sodium chloride	25 g
potassium citrate	4.5 g
potassium chloride	3 g
glucose	130 g
water	4 L

Correcting Fluid Loss

The requirement of the animal for replenishment of losses is in addition to the daily maintenance requirements, plus any anticipated further losses. Maintenance fluid requirements on a body weight basis are greater in calves than in older cattle (Mulville 1991).

Calves that are dehydrated less than 8% may be treated with oral therapy only, which is adequate if given early in the disease and continued until full recovery (Heath *et al* 1989). The degree of dehydration is assessed by skin turgor of the eyelids and the degree of enophthalmos (Table 1). Measurement of total serum protein as an index of dehydration is unreliable in newborn calves; it varies with the amount of colostral immunoglobulin absorbed (Roussel 1983).

TABLE 1
Guide to estimate the degree of dehydration and the corresponding base deficit in diarrhoeic calves*

Clinical signs	Dehydration %	Approximate deficit base (mEq)†
History of diarrhoea with mininal clinical signs	5	5
Slight enophthalmos, skin turgor decreased, continues to suckle	7	10
Enophthalmos, skin turgor decreased, mucous membranes tacky, depressed	9	15
Severe enophthalmos, skin tents and does not return within 5 sec, unable to rise, possibly comatose	12	20

* Adapted from Roussel AJ (1983)
† Per litre of extra-cellular fluid

The volume of fluid replacement required is calculated as follows:

Weight of the calf x % of dehydration, for example, 40 kg x 5% = 2 litres of fluid plus allowance for maintenance requirements (minimum of 100 mL/kg/day), for example, 40 kg x 100 mL/kg = 4 litres of fluid. Total amount adds up to 6 litres or 15% of body weight, for example, 40 kg calf x 15% = 6 litres. Ideally, this volume should be given over three, but at least two feedings.

The amount of sodium bicarbonate ($NaHCO_3$) required is calculated as follows:

Total base deficit (in milliequivalents) = base deficit (Table 1) x 0.5 x body weight in kg. For example, 40 kg calf, 7% dehydrated: 10 x 0.5 x 40 = 200 mEq. Divide total base deficit by 12 to convert milliequivalents to grams of $NaHCO_3$: 200 / 12 = 16.5 g of $NaHCO_3$ is needed to correct the acidosis.

Oral fluid therapy results in a positive net gain in body fluids as well as a net increase in faecal volume (Roussel 1983). This should not be confused with treatment failure.

In cases of severe fluid loss, it may be necessary to give 2 litres of fluids 3 to 4 times daily. Diarrhoeic calves should be given oral electrolytes until their diarrhoea subsides. The glucose content in oral rehydration solutions is optimised for Na^+ absorption, not caloric yield (Michell 1993). Even in healthy neonatal calves, hypertonic (oral) replacement fluids fail to provide sufficient energy to sustain body weight (Bradley and Niilo 1984; Fettman *et al* 1986). If milk is removed from the diet in case of diarrhoea, it should be reintroduced after 36 to 48 hours and electrolyte solutions should be fed in addition to milk (Heath *et al* 1989; Michell *et al* 1992) with little risk of exacerbating diarrhoea (Naylor *et al* 1990). Milk and electrolyte feeding should be separated by at least 4 to 6 hours to allow milk to clot for digestion (Heath *et al* 1989).

With early detection and appropriate management, most cases of dehydration associated with calf diarrhoea can be satisfactorily treated by the oral route. For those isolated cases where dehydration is 8% or greater, the intravenous route of therapy is recommended (Roussel 1983). An intravenous catheter with a minimum length of 12.5 cm and a 1.2-1.5 mm internal diameter should be used. The use of hypodermic needles risks excessively rapid administration, inadequate volume replacement, perivascular administration, or a combination of these effects.

Most calves respond favourably to intravenous fluid therapy within 24 to 48 hours. For good response, the aim should be to correct one-third of the total deficit intravenously and the remainder by another route (orally, subcutaneously or intraperitoneally).

The flow rate of intravenous fluids should not exceed 30 to 40 mL/kg body weight per hour (Roussel 1983). A 40 kg calf that is 10% dehydrated should be rehydrated over a period of 3 to 4 hours. The base deficit can be corrected more rapidly, but replacement fluids for maintenance should be administered at a slow rate. Potassium-boosted solutions (for example, Darrow's solution) should be avoided for the initial rehydration of severely acidotic and shocked calves, which require fast infusion rates (Michell 1993).

Lactated Ringer's solution is the best treatment for calves with severe dehydration and metabolic acidosis (Michell 1993). This solution contains a small amount only of potassium and is unlikely to exacerbate hyperkalaemia accompanied by acidosis. Alternatively, a physiological (0.9%) saline solution, with up to 5 g $NaHCO_3$/L, 50 g glucose/L and up to 1.5 g KCl/L added should be used (Roussel 1983).

In addition to intravenous electrolyte solutions, plasma (800 mL per 40 kg calf) or whole blood (1 to 1.5 litre per 40 kg calf) transfusions can be used as an aid in treating diarrhoea in calves (Amstutz 1978). With this type of treatment, there is the potential risk of transfusion reactions and the transmission of infectious agents, such as enzootic bovine leucosis virus, bovine immunodeficiency virus and bovine viral diarrhoea virus. If possible, donor cattle from farms known to be free of these diseases should be selected.

Other Treatments

The emphasis in the treatment of calf diarrhoea is on the correction of dehydration and acidosis, so that the animal is fit enough to mount its own recovery. By comparison, other treatments have only minor impact on the outcome of the disease.

Antibacterial Agents

The oral administration of antibiotics does not alter the course of diarrhoea (Lewis and Philips 1980). The use of parenteral antibiotics in calf diarrhoea should be restricted to cases of salmonellosis, where septicaemia is common (Jerrett 1985). Systemic antibiotics reduce mortality and faecal shedding of the organism (Osborne 1978). The choice of antibiotics should be based on the antimicrobial sensitivity of the *Salmonella* species. Ampicillin, neomycin and tetracycline, which can cause diarrhoea in healthy calves (Rolin *et al* 1986), should be avoided. The use of sulphonamides potentially threatens export markets for veal and beef.

Calves should be considered neonates up to 4 weeks of age. The therapeutic dose rate and frequency of administration of drugs, such as antibiotics, needs adjusting for this age group. The ability of young, diarrhoeic calves to metabolise these compounds may be impaired.

Probiotics

Probiotics supplement the intestinal microflora with viable, beneficial bacteria and thus create conditions unfavourable for the growth of enteropathogens, thereby reducing the occurrence of scours (Montes and Pugh 1993). The mechanism of action is complex and beyond the scope of this paper. Probiotics are not an alternative to fluids and antibiotics in the treatment of neonatal diarrhoea, but rather a complementary therapy for restoring balance to the intestinal flora.

The probiotics that are currently available contain strains of lactic acid-producing organisms, mainly *Lactobacillus* and *Streptococcus* species. The minimum effective dose is 10^8 to 10^{10} colony-forming units per calf per day (Porcuban 1990). Probiotics in gel or paste form are more effective than freeze-dried, because the bacteria suspended in vegetable oil are better able to survive the low pH of the stomach (Porcuban 1990).

There is little or no benefit when probiotics are given to healthy calves or those with adequate immunocompetence and well-established gut microflora (Fox 1988).

Other Symptomatic Treatments

Supplementing oral rehydration solutions for neonatal calf diarrhoea with soluble dietary fibre, such as psyllium mucilloid and pectin, improves the assimilation of oral glucose, mainly through alterations in gastrointestinal transit (Fettman 1992).

Diarrhoea as such has a protective function in reducing bacterial adhesion and evicting enteropathogens (Michell 1993). Symptomatic treatment, especially when directed towards gut motility, may be counterproductive. Reduced motility causes pooling of fluids in the intestines, without a significant increase in absorption (Jerrett 1985). Oral administration of intestinal protectants (kaolin and bentonite) is useful in reducing the severity of diarrhoea.

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Facing the future

"... If the art and science of veterinary medicine are to progress with their full potential, it is clear that no one school can provide the breadth and depth to adequately train the veterinarians for the 21st century. Similarly, it is likely that the concept that we must train each of our students to be equally competent, or equally incompetent, in all the traditional areas of veterinary practice, will soon be a thing of the past.

"This concept can only stifle our progress, and ensure that our profession misses the opportunities open to it. We must both permit and encourage choice in terms of elective opportunities for our students so that chosen areas can be studied in greater depth.

"The deliberations of the Pew Foundation in North America, and of the Working Party on Veterinary Education of our own RCVS have made it clear that the way forward lies in the schools developing their own complementary areas of emphasis — each staffed to an adequate degree. The students may then spend time studying their chosen electives at other than their host institution. We will seek to collaborate with others, and particularly with our colleagues in Glasgow, to provide the educational opportunities that match the potential of our students and the needs of the profession..."

Halliwell REW (1993) *Br Vet J* **149**:313-315

Professor Halliwell is Dean of the Royal (Dick) School of Veterinary Studies, Edinburgh

Storing bibliographic data

There are more than 20 commercial computer programs for creating bibliographic data bases. Blumenthal EZ and Gilad R (1993) *New Engl J Med* **329**:283-284 reviewed 5 programs for use on DOS-operated personal computers. (There are also software packages for use on Macintosh, VAX/VMX, UNIX, and other operating systems.)

The five programs have much in common. Choice should depend, according to the reviewers, on price, user-friendliness, and other features that they tabulated in detail. They found two of the programs — Papyrus (US$99) and Reference Manager (US$499) — most suitable for the task of dealing with down-loaded MEDLINE references.

They point out that the right way to choose the product that best suits one's needs is by trying out the demonstration versions of the software.

Has the profession a history?

"I wonder if there is any profession that has made such strides in advancement as the veterinary profession, *in spite of its members.*

"When the Australian Veterinary Association decided to have a history of the profession there was a flourish of trumpets and collaborators were appointed; the profession visualised the value of such an undertaking and then forgot all about it.

"Unless the individual members of the profession are prepared to give the subject some attention and forward information, the writing of the history must be abandoned, and the only publication likely to be made will be my obituary, written by myself.

"That, by the way, is not a bad line to work on. Let every member write what his own notice should be, and, having done that, write one for any friend or enemy, whether dead or alive. From such information it may be possible for the collaborators to collaborate and eventually write the history ..."

WAN Robertson (1936) *Aust Vet J* **12**:165