CASE REPORT

Metabolic Acidosis Without Clinical Signs of Dehydration in Young Calves

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Summary

Metabolic acidosis without clinical signs of dehydration was diagnosed in four calves between nine and 21 days of age. In each calf either coma or depression with weakness and ataxia was observed. Two calves had slow deep respirations. Treatment with intravenous administration of solutions of sodium bicarbonate was accompanied by a rise in blood pH and a return to normal demeanor, ambulation and appetites, allowing these calves to return to their respective herds.

Key words: Calves, metabolic acidosis, depression, sodium bicarbonate.

Résumé

Acidose métabolique, sans signes cliniques de déshydratation, chez des jeunes veaux

Cet article rapporte le diagnostic d'une acidose métabolique, sans signes cliniques de déshydratation, chez quatre veaux âgés de neuf à 21 jours; ils manifestaient un coma ou une dépression qui s'accompagnaient de faiblesse et d'ataxie; deux d'entre eux affichaient aussi une respiration lente et profonde. L'administration intraveineuse d'une solution de bicarbonate de sodium entraîna une élévation du pH sanguin, ainsi qu'un retour à un maintien, une démarche et un appétit normaux. Le succès du traitement précité permit de renvoyer les veaux dans leurs troupeaux respectifs.

Mots clés: veaux, acidose métabolique, dépression, bicarbonate de sodium.

Introduction

We have been concerned with losses in one to four week old calves which die without a cause of death being determined at necropsy. These calves often have a history of diarrhea and although obviously depressed are usually not dehydrated or severely emaciated.

The following is a description of a syndrome of metabolic acidosis without signs of dehydration which we have detected in some weak to comatose beef calves in their second to fourth week of life.

Case Reports

Methods — Each calf was examined and blood was collected for laboratory tests. Blood was drawn from the jugular vein and placed in a tube containing potassium ethylenediamine tetraacetate (EDTA) for hematology and into a serum tube for electrolyte analysis, protein electrophoresis and urea nitrogen determination; a tube containing potassium oxalate and sodium fluoride for glucose determination; and a heparinized syringe for blood gas analysis.

Hemograms were performed using an automated counter[1] and blood smears were stained with Wright-Giemsa stain[2] prior to microscopic examination. Serum sodium, potassium and chloride were measured using ion specific electrodes.[3] Serum total protein was measured using a Biuret method[4] on an automated analyzer,[5] serum proteins were fractionated by agarose gel electrophoresis[6] and were quantitated on a densitometer.[7] Urea nitrogen was measured on urease treated plasma using a conductivity method.[8] Glucose was measured using a hexokinase method[9] in an automated analyzer. Blood gas analysis was performed on an automated analyzer[10] and the results were

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[1]Coulter Counter, Model S, Coulter Electronics Inc., Haileah, Florida.
[2]H-Pack, Ames Company, Elkhart, Indiana.
[3]Beckman System E4A, Beckman Instruments Inc., Brea, California.
[4]Total Protein, Biuret Method, Worthington Diagnostic Systems Inc., Freehold, New Jersey.
[5]Abbott Bichromatic Analyzer 100, Abbott Laboratories Inc., Mississauga, Ontario.
[6]Paragon Electrophoresis System, Beckman Instruments Inc., Brea, California.
[7]ACD-18 Automatic Computing Densitometer, Gelman Instruments Co., Ann Arbor, Michigan.
[8]BUN Combined Reagent and BUN Analyzer II, Beckman Instruments Inc., Brea, California.
[9]Hexokinase, Diagnostic Chemicals Ltd., Charlottetown, Prince Edward Island.
[10]Corning 178 pH/Blood Gas Analyzer, Corning Medical, Medfield, Massashusetts.

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corrected for calf temperature and hemoglobin concentration.

Ancillary procedures in selected cases included radiography, cerebrospinal fluid analysis, peritoneal fluid cytology and organic (lactate, acetate) acid and acetoacetate determinations. Cerebrospinal fluid was collected from the lumbosacral subarachnoid space (1) and peritoneal fluid was obtained according to the method of Oehme (2). L-lactate concentrations were determined in an automated lactate analyzer[11] from 100 μL aliquots of heparinized blood processed according to the method of Soutter, Sharp and Clark (3). Blood acetate concentrations were determined by monitoring the formation of dihydronicotinamide-adenine dinucleotide (NADH) during the conversion of acetate to oxaloacetate (4). Acetoacetate concentrations were determined by a semiquantitative colorimetric method using nitroprusside powder and serial dilutions of plasma (5).

The treatment of acidosis in all calves involved the intravenous administration of sodium bicarbonate solutions. Isotonic solutions were prepared by dissolving 47 or 23.5 g of sodium bicarbonate in 3.6 L of either distilled water, or half isotonic (0.425%) saline, to yield a final bicarbonate concentration of 157 mmol/L or 78 mmol/L respectively. The amount of sodium bicarbonate required to correct the acidosis in each calf was calculated using the formula (6):

Amount of bicarbonate required, mmol =

Base deficit, mmol/L x 0.3 x body weight, kg

Each calf was also administered a trimethoprim-sulfadoxine[12] antibiotic (16 mg/kg, IM, SID) during hospitalization.

History and Clinical Findings

Patient 1 — A 50 kg 21 day old Charolais heifer was presented to the Western College of Veterinary Medicine (WCVM) because of weakness and disinclination to nurse. Diarrhea the previous week had responded to oral antibiotics.

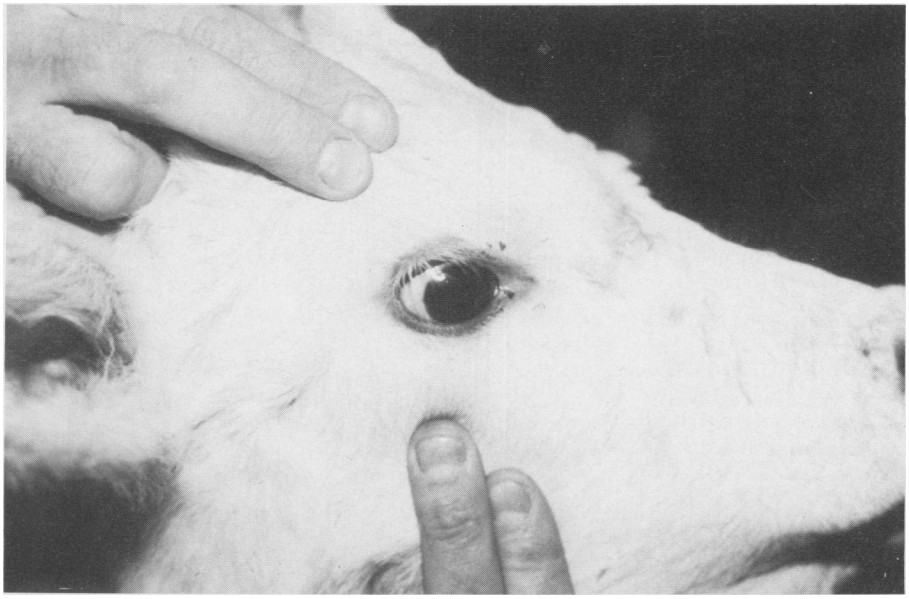

FIGURE 1. Normal eye position in the orbit and the lack of separation of the third eyelid from the orbit is consistent with a normal state of hydration in this 21 day old calf (Patient 1).

The calf's temperature, pulse and respiration were 39.5°C, 140/min and 60/min, respectively. She was very depressed, exhibited rear leg ataxia with knuckling of the fetlocks when stimulated to walk and occasionally assumed a dog-sitting posture and required assistance to stand. A suckle reflex was absent. The menace response was depressed although indirect and direct pupillary light reflexes were intact. Hydration was normal as assessed by a normal position of the eye within the orbit (Figure 1) and normal skin elasticity of the neck and upper eyelid. Increased nontympanic fluid splashing sounds were audible upon succession of the abdomen. The perineum was smeared with dried feces, while soft formed feces were present in the rectum.

Laboratory tests revealed a compensated metabolic acidosis (Table I). Cerebrospinal fluid was collected, the protein content (1.05 g/L) was slightly elevated (normal values are up to 0.40 g/L (10)). Radiographs of the lumbar spine were normal.

Patient 2 — A 47 kg nine day old male Charolais was examined for a complaint of weakness and ataxia of the front and rear legs. He had been delivered by forced extraction from a first calf heifer, but was strong and vigorous at birth. A brief episode of diarrhea had been evident four days prior to admission. This stopped coincident with oral administration of antibiotics.

The calf's temperature, pulse and respiration were 39.7°C, 88/min and 40/min, respectively. He was severely depressed, weak and ataxic in the rear limbs when forced to walk. He lacked a menace response although indirect and direct pupillary light reflexes were intact. A weak suckle reflex was present. Hydration was normal as assessed by a normal position of the eye within the orbit and normal skin elasticity of the neck and upper eyelid. Increased inspiratory and expiratory bronchial sounds were noted anteroventrally in each lung field, however, a cough could not be elicited. Normal pasty feces were present in the rectum while succussion of the abdomen produced increased nontympanic fluid splashing sounds.

[11]Lactate Analyzer 640, Roche, Kontron Scientific Ltd., Mississauga, Ontario.
[12]Trivetrin, Burroughs Wellcome Inc., Kirkland, Quebec.

TABLE I

INITIAL CLINICOPATHOLOGICAL FINDINGS IN FOUR CALVES WITH METABOLIC ACIDOSIS IN THE ABSENCE OF CLINICAL SIGNS OF DEHYDRATION

Parameter	Patient 1	2	3	4	Normal Values (Mean ± S.D.)
Blood gas (Venous)					
pH	7.065	7.146	7.064	6.933	7.38 ± 0.05[b]
Partial pressure of carbon dioxide (Torr)	31.3	36.0	52.8	24.5	55 ± 9[b]
Bicarbonate (mmol/L)	8.7	12.1	14.8	5.3	31 ± 5[b]
Base deficit (mmol/L)	19.6	15.0	15.5	28.5	
Erythrocyte count (x 10^{12}/L)	8.12	5.78	8.18	11.76	7.38 ± 1.14[c]
Hemoglobin (g/L)	116.0	76.0	112.0	158.0	105 ± 19[c]
Packed cell volume (L/L)	0.33	0.24	0.33	0.44	0.33 ± 0.06[c]
Leukocyte count (x 10^9/L)	9.0	9.1	7.5	13.4	8.5 ± 2.0[c]
Total neutrophils (x 10^9/L)	4.0	4.2	4.1	4.0	2.7 ± 1.6[c]
Band neutrophils (x 10^9/L)	0.0	0.0	0.8	2.7	
Lymphocytes (x 10^9/L)	4.9	4.4	3.2	5.0	5.1 ± 1.5[c]
Monocytes (x 10^9/L)	0.0	0.5	0.0	1.6	0.6 ± 0.3[c]
Total protein (g/L)	67.0	62.0	47.0	66.0	51 ± 7.0[e]
Albumin (g/L)	35.0	24.0	24.0	29.0	24 ± 2.0[e]
Gamma globulin (g/L)	10.0	16.0	2.0	11.0	10 ± 6[e]
Sodium (mmol/L)	139.0	135.0	146.0	143.0	148 ± 13[b]
Potassium (mmol/L)	3.7	5.2	4.9	4.1	5.4 ± 0.8[b]
Chloride (mmol/L)	110.0	106.0	111.0	116.0	95 ± 5[b]
Urea nitrogen (mmol/L)	10.7	5.9	ND[a]	12.3	4.6 ± 1.88[d]
Glucose (mmol/L)	3.7	ND	3.0	ND	4.39 ± 0.61[d]

[a]ND = not determined

[b]Reference 7

[c]Reference 8

[d]Reference 27

[e]Reference 9, colostrum fed calves

Laboratory data revealed a metabolic acidosis (Table I).

Patient 3 — A 47 kg 14 day old male Charolais was presented to the WCVM in a state of collapse. He had been dull and inactive for several days prior to presentation and had received oral electrolytes the previous day.

The calf had a rectal temperature of 39°C, a heart rate of 107/min, a respiratory rate of 20/min and was comatose. Capillary refill time (2 sec) was normal and oral cavity mucous membranes were pink and warm. Hydration was normal as assessed by a normal position of the eye within the orbit and normal skin elasticity of the neck and upper eyelid. Slow, deep, thoracic and abdominal respirations with equal emphasis upon inspiration and expiration were noted. Auscultation revealed no abnormal lung sounds. Upon auscultation of the abdomen, the intestines emitted gurgling sounds and succussion provided increased nontympanic fluid splashing sounds. Firm, white feces were contained in the rectum and dried feces were smeared over the perineum.

Laboratory results indicated a compensated metabolic acidosis and hypogammaglobulinemia (Table I).

Patient 4 — A 52 kg 14 day old male Charolais was presented for a complaint of anorexia of several days duration. Although there was no previous history of illness, the calf had been orphaned one week prior to admission and transferred to a surrogate dam. Treatment with oral electrolytes had not improved feed intake.

The calf was comatose with a temperature, pulse and respiration of 34°C, 80/min and 32/min, respectively. The heart sounds were regular and strong although the mucous membranes of the oral cavity and palpebral conjunctiva were pale and the capillary refill time (4 sec) was prolonged. Hydration was normal as assessed by the position of the eye within the orbit and skin elasticity of the neck and upper eyelid. Deep thoracic and abdominal respiratory movements with equal emphasis upon inspiration and expiration were noted without any abnormal lung sounds on auscultation. There were normal pasty feces within the rectum and increased nontympanic fluid splashing sounds on succussion of the abdomen.

Laboratory results showed a profound compensated metabolic acidosis and neutrophilic leukocytosis with a left shift (Table I). A sample of peritoneal fluid was normal on cytological examination. Plasma L-lactate, acetate and acetoacetate were measured and found to be (reference values in parenthesis), 0.85 mmol/L (< 1.18 mmol/L), 0.037 mmol/L (< 0.306 mmol/L) and < 20 mg/L (< 20 mg/L), respectively.

Treatment

The initial management of each calf was directed towards improving acid-base status. In most cases the calculated bicarbonate deficit was replaced during a four to eight hour period, an additional blood gas analysis was then performed to monitor blood pH, bicarbonate concentration and base deficit. Additional bicarbonate was adminis-

TABLE II

PACKED CELL VOLUME (PCV), VENOUS BLOOD GAS DETERMINATIONS, AMOUNT OF BICARBONATE AND VOLUME OF FLUID ADMINISTERED DURING ISOTONIC SODIUM BICARBONATE FLUID REPLACEMENT THERAPY IN FOUR CALVES WITH METABOLIC ACIDOSIS

Calf	Time h	PCV L.L	pH	Partial Pressure of Carbon Dioxide Torr	Bicarbonate mmol/L	Base Deficit mmol/L	Cumulative Amount of Bicarbonate Administered mmol/L	Cumulative Volume of Fluid Administered L
1	Entry	0.33	7.065	31.3	8.7	19.6	-0-	-0-
	8	ND[a]	7.218	34.6	13.7	12.0	282	1.8[b]
	24	0.31	7.319	46.7	20.2	8.5	564	3.6
2	Entry	0.24	7.146	36.0	12.1	15.0	-0-	-0-
	24	0.22	7.392	42.3	25.0	1.6	282	3.6[c]
3	Entry	0.33	7.064	52.8	14.8	15.5	-0-	-0-
	8	ND	7.239	54.3	23.2	5.0	392	2.5[b]
	24	0.29	7.308	66.6	32.9	-5.1	392	2.5
4	Entry	0.44	6.933	24.5	5.3	28.5	-0-	-0-
	4	0.41	7.278	29.2	13.7	11.7	440	2.8[b]
	8	0.36	7.378	52.0	29.7	-5.4	627	4.0
	24	0.35	7.341	41.2	21.8	2.5	627	4.0

[a]ND = not determined

[b]Isotonic sodium bicarbonate

[c]Half isotonic (0.425%) saline and sodium bicarbonate

tered if necessary. Sodium bicarbonate therapy was terminated when blood gas values for bicarbonate reached the normal range (20-40 mmol/L). The total amount of bicarbonate and volume of fluid delivered to each calf is summarized in Table II.

Each calf responded dramatically to this specific therapy and the clinical improvement noted closely paralleled the improvement in each calf's acid-base status.

Calf 1 was much brighter at the end of 24 hours therapy. The calf was able to stand unassisted although still slightly ataxic when allowed to walk. The suckle reflex returned to normal and she nursed oral electrolytes and milk vigorously during the next 24 hours. A completely normal gait was apparent 48 hours after admission at which time she was discharged.

Calf 2 was much brighter and less ataxic after one day of hospitalization. The suckle reflex was still weak. However, he was clinically normal by day 2 of hospitalization and was discharged.

Calf 3 was slightly improved after eight hours of sodium bicarbonate fluid replacement. A weak menace response and muscle tremors were present. He remained recumbent, lacked a suckle reflex and a central response to skin stimulation. However, by 24 hours he could stand unassisted, but was still weak and ataxic. Menace and tactile responses were normal at this time but placing a finger in the calf's mouth only resulted in a weak chewing response instead of a sucking reflex. Appetite improved during the course of the day and the calf eventually nursed 1 L whole milk. He was maintained on 1 L whole milk Q.I.D. through day 2 of hospitalization. On day 3 of hospitalization the calf developed pneumonia which was successfully treated with chloramphenicol[13] (25 mg/kg, IV, Q.I.D.). He was discharged on hospitalization day 7.

Calf 4 had improved eight hours after initiating bicarbonate therapy; he was able to stand unassisted but was still ataxic. At this time temperature had risen to 40.2°C, menace and tactile responses to external stimuli were normal, but only a weak chewing reflex was elicited when a finger was placed in the calf's mouth. At the end of 24 hours therapy, the calf was clinically normal. The calf was maintained on 1 L whole milk and 1 L oral electrolytes Q.I.D. until discharge on hospitalization day 3.

A follow-up telephone conversation with the owners was made approximately two weeks after discharge of each calf. All animals were well.

Discussion

Metabolic acidosis in diarrheic calves is thought to develop from the combination of large fecal bicarbonate ion losses (11,12) and plasma lactic acid accumulation (11,13) secondary to anaerobic glycolysis induced by dehydration and poor tissue perfusion. In addition, decreased renal excretion of hydrogen ions in dehydrated calves (11,14) and respiratory mechanisms (15) have been implicated in the development of acidosis.

Although one calf developed pneumonia during hospitalization none of the calves had evidence of respiratory disease at presentation. This, together with the metabolic nature of acidosis (large base deficits) and the response to sodium bicarbonate therapy suggests that acidosis was not due to respiratory problems. Organic and keto-acid production was determined in only one calf. However, the normal plasma levels of lactate, acetate and acetoacetate found may indicate that these anions are not important in the development of acidosis. Fecal bicarbonate ion loss secondary to small intestinal enteropathy may be an

[13]Rogar-Mycine '200', Rogar/STB, London, Ontario.

alternate explanation for the metabolic acidosis (16). All calves had a consistently low blood bicarbonate concentration and increased fluid splashing sounds on succussion of the abdomen. Two of the calves had a history of diarrhea and a third calf had evidence of fecal staining of the perineum suggesting a past episode of diarrhea.

Only metabolic acidosis is likely to be responsible for the depression and ataxia seen in these calves. Hypoglycemia has been reported in diarrheic calves which were weak, lethargic or comatose (17-20). The hypoglycemia, often with hyperlactatemia, usually occurred in the terminal stages of the disease. Hypoglycemia was considered not to be an important feature in the calves reported here since all calves responded to therapy with bicarbonate-containing fluids alone. Hyperkalemia, a common finding in calves with diarrheic acidosis (11,21-23) can produce muscle weakness. However, serum potassium levels were found to be normal in each calf at entry.

In the calves described in this report the principal clinical signs noted were referable to the central nervous system. Calves 1 and 2 had varying degrees of depression, weakness and ataxia whereas calves 3 and 4 presented in a comatose state. Initial depression followed by disorientation and, finally, coma if blood pH drops severely, are the expected clinical signs of metabolic acidosis (24). In addition, calves 3 and 4 had a characteristic slow, deep thoracic and abdominal breathing pattern (Kussmaul respiration) which is similar to that seen in people with metabolic acidosis (25).

The treatment of each calf with isotonic solutions of sodium bicarbonate or sodium bicarbonate and saline gave good results. These calves regained normal demeanor, ambulation and appetites following treatment and were returned to their herds. The total quantity of bicarbonate in mmol needed to return the blood pH to near normal levels was quite variable, even among calves of similar weights and degrees of acidosis. In some cases the amount was considerably higher (up to double the amount) than anticipated using a volume of distribution for bicarbonate of 0.3 of body weight to calculate bicarbonate requirements. This is consistent with recommendations for dogs and cats which indicate that the volume of distribution of bicarbonate is 0.6 of body weight (26). Therefore, when the practitioner suspects this syndrome of metabolic acidosis in calves a more accurate estimate of the total dosage of sodium bicarbonate to use can be calculated using this modified formula:

Amount of bicarbonate required, mmol =
base deficit, mmol/L x 0.6 x body weight, kg.

Furthermore, due to the general inaccessability of a blood gas apparatus for field use an arbitrary base deficit value of 15 mmol/L may be appropriate to use in this formula.

All animals had decreased packed cell volumes (PCV) following 24 hours of therapy. The PCV decreased by approximately 9% in calf 4, and 2 to 4% in the remaining calves. All calves were noticed to urinate sometime during the period of fluid administration. It is somewhat difficult to ascertain whether the drop in PCV represents overhydration of normal animals or correction of actual dehydration which escaped accurate physical detection. However, the elevated blood urea nitrogen (BUN) concentration in calf 4 at entry may be a reflection of slight dehydration induced prerenal uremia.

All calves affected with metabolic acidosis in this report were Charolais. However, speculating on a unique susceptibility of calves of this breed to acidosis at this time would be premature. This report describes a small number of calves and Charolais are popular in our practice area.

Previous reports of metabolic acidosis in calves have associated acidosis with obvious dehydration (11,15,21,22). Our report highlights a new syndrome of acidosis without obvious clinical signs of dehydration. This should help explain some cases of depressed, weak and failing calves with a recent history of diarrhea. In the past acidosis may have been underdiagnosed because of the difficulties of doing blood gas analysis in the field and the lack of specific necropsy findings. Increased awareness of the presence of acidosis in clinically well hydrated calves, combined with its response to large volumes of isotonic bicarbonate, should improve therapy in the neonatal calf.

References

1. MAYHEW IG. Cerebrospinal fluid. In: Howard JL, ed. Current veterinary therapy. Food animal practice. Philadelphia: WB Saunders Co., 1981: 1078-1080.
2. OEHME FW. Cytologic examination of the peritoneal fluid in the diagnosis of cattle disease. J Am Vet Med Assoc 1969; 155: 1923-1927.
3. SOUTTER WP, SHARP F, CLARK M. Bedside estimate of whole blood lactate. Br J Anaesth 1978; 50: 445-450.
4. ACETIC ACID. Methods of enzymatic food analysis. Mannheim, W. Germany: Boehringer Mannheim GmbH Biochemica, 1980.
5. KRONFELD DS. Plasma non-esterified fatty acid concentrations in the dairy cow: Responses to nutritional and hormonal stimuli, and significance in ketosis. Vet Rec 1965; 77: 30-35.
6. MALLEMGAARD K, ASTRUP P. The quantitative determination of surplus amounts of acid-base in the human body. Scand J Clin Lab Invest 1960; 12: 187-199.
7. BUTLER DG, WILLOUGHBY RA, McSHERRY BJ. Studies on diarrhea in neonatal calves III. Acid-base and serum electrolyte values in normal calves from birth to ten days of age. Can J Comp Med 1971; 35: 36-39.
8. TENNANT B, HARROLD D, REINA-GUERRA M, KENDRICK JW, LABEN RC. Hematology of the neonatal calf: Erythrocyte and leukocyte values of normal calves. Cornell Vet 1974; 64: 516-532.
9. TENNANT B, HARROLD D, REINA-GUERRA M, LABEN RC. Neonatal alterations in serum gamma globulin levels of Jersey and Holstein-Friesian calves. Am J Vet Res 1969; 30: 345-354.
10. De LAHUNTA A. Veterinary neuroanatomy and clinical neurology. Philadelphia: WB Saunders, 1977: 48.
11. TENNANT B, HARROLD D, REINA-GUERRA M. Physiologic and metabolic factors in the pathogenesis of neonatal enteric infections in calves. J Am Vet Med Assoc 1972; 161: 993-1007.
12. LEWIS LD, PHILLIPS RW. Water and electrolyte losses in neonatal calves with acute diarrhea. A complete balance study. Cornell Vet 1972; 62: 596-607.
13. LEWIS LD, PHILLIPS RW, ELLIOTT CD. Changes in plasma glucose and lactate concentrations and enzyme activities in the neonatal calf with diarrhea. Am J Vet Res 1975; 36: 413-416.
14. DALTON RG, PHILLIPS GD. Renal function in neonatal calves: response to acidosis. Br Vet J 1969; 125: 367-378.
15. PHILLIPS RW, KNOX KL. Diarrheic acidosis in calves. J Comp Lab Med 1969; 3: 1-3.
16. BYWATER RJ, LOGAN EF. The site and characteristics of intestinal water and electrolyte loss in escherichia coli-induced diarrhoea in calves. J Comp Path 1974; 84: 599-610.
17. TENNANT B, HARROLD D, REINA-GUERRA M. Hypoglycemia in neonatal calves associated

with acute diarrhea. Cornell Vet 1968; 58: 136-146.
18. PHILLIPS RW, CASE GL. Altered metabolism, acute shock and therapeutic response in a calf with severe coronavirus-induced diarrhea. Am J Vet Res 1980; 41: 1039-1044.
19. DEMIGNE C, REMESY C. Evolution of the postnatal metabolism in the healthy or diarrhoeic calf. Ann Rech Vet 1979; 10: 23-31.
20. McSHERRY BJ, GRINYER I. Disturbances in acid-base balance and electrolyte in calf diarrhea and their treatment. A report of eighteen cases. Am J Vet Res 1954: 15: 535-541.
21. FISHER EW. Hydrogen ion and electrolyte disturbances in neonatal calf diarrhea. Ann NY Acad Sci 1971; 176: 223-230.
22. FISHER EW. Death in neonatal calf diarrhoea. Br Vet J 1965; 121: 132-138.
23. DEMIGNE C, CHARTIER F, REMESY C. Evidence of different types of acidosis associated with diarrhea in the neonatal calf. Ann Rech Vet 1980; 11: 267-272.
24. GUYTON AC. Textbook of medical physiology, 6th ed. Philadelphia: WB Saunders, 1981: 448-462.
25. GOLDBERGER E. A primer of water, electrolyte and acid-base syndromes, 4th ed. Philadelphia: Lea & Febiger, 1970: 129-141.
26. FINCO DR. General guidelines for fluid therapy. J Am Anim Hosp Assoc 1972; 8: 166-177.
27. REECE WO. Acid-base balance and selected hematologic, electrolyte, and blood chemical variables in calves: milk-fed vs conventionally fed. Am J Vet Res 1980; 41: 109-113.

ABSTRACTS

ROSS HM, HUNTER AR, MASSON AG, NETTLETON PF. **Fatal infection of neonatal calves by infectious bovine rhinotracheitis virus.** *Veterinary Record.* 1983; 113: 217-218. (VI Centre, North of Scotland Coll. Agric., Drummondhill, Stratherrick Rd., Inverness, UK).

Infectious bovine rhinotracheitis (IBR) in seven calves born to replacement dairy heifers is described. Calves were healthy at birth but when 3-4 days old developed bilateral serous ocular and nasal discharges, excessive salivation and pyrexia. Five calves died unexpectedly after 2-3 days' illness; the last two calves born recovered slowly over two weeks. Calves in direct and indirect contact were not clinically affected, PM lesions were bilateral conjunctivitis, hyperaemia of the anterior nares and oral cavity, and acute pharyngitis with tenacious mucopurulent exudate and extensive oedema. There was oesophageal congestion with multiple focal lesions of the mucosa and marked distension of the reticulorumen with milk, while abomasal contents were scant. There was acute bronchopneumonia. IBR virus was cultured from a nasal swab, retropharyngeal lymph node, oesophagus, abomasum and rectum of the freshest carcass and from nasal but not ocular or rectal swabs of survivors. Only one of the dams had detectable antibodies to IBR, but paired sera from a calf in an adjacent pen showed significant seroconversion.

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BANKS KL, ADAMS DS, McGUIRE TC, CARLSON J. **Experimental infection of sheep by caprine arthritis-encephalitis virus and goats by progressive pneumonia virus.** *American Journal of Veterinary Research.* 1983; 44: 2307-2311. (Dep. Vet. Microbiol. Path., State Univ., Pullman, Washington 99164-7040, USA).

Upon inoculation with caprine arthritis-encephalitis virus (CAEV) lambs developed a non-suppurative arthritis and antibody to CAEV, and the virus was isolated up to 4 months later. Exposure of 3 lambs to CAEV-infected adult goats did not lead to demonstrable infection after 18 months. Young goats inoculated with PPV replicated the virus and developed arthritis and antiviral antibody. These results demonstrate that these distinct lentiviruses may infect and cause diseases in species other than their accustomed host. Present techniques may not be effective in differentiating which lentivirus is responsible for infection of sheep and goats. The results also indicate that mixing sheep and goats may adversely influence attempts to eradicate lentiviruses from these species.

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