

# Hypernatremia in neonatal elk calves: 30 cases (1988–1998)

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**Objective**—To characterize hypernatremia in neonatal elk calves, including clinical signs, incidence, physical examination findings, and possible causes.

**Design**—Retrospective case series.

**Animals**—26 neonatal elk calves were examined; 4 calves were evaluated twice, for a total of 30 examinations.

**Procedure**—Medical records were reviewed for signalment, history, physical examination findings, results of diagnostic tests, and response to treatment. Hypernatremia was defined as serum sodium concentration > 153 mEq/L.

**Results**—Hypernatremia was diagnosed in 14 calves and was significantly associated with diarrhea, high WBC count, high anion gap, and high serum concentrations of albumin, chloride, creatinine, and urea. Hypernatremia was not significantly associated with survival, but high serum albumin concentration and rectal temperature were significantly associated with survival of calves. Animals given antibiotics and electrolyte solutions orally prior to evaluation were significantly more likely to die than those untreated. Dehydration was a common reason for evaluation but was not significantly associated with survival.

**Conclusions and Clinical Relevance**—Hypernatremia was significantly associated with diarrhea. Treatment of diarrheic elk calves is often the same as that used in bovine calves with diarrhea; however, bovine calves are commonly hypo- or normonatremic. Our experience suggests that treatment protocols used in bovine calves are unsatisfactory for elk calves. The rate at which serum sodium concentration is reduced should be < 1.7 mEq Na/L/h to avoid development of neurologic signs associated with iatrogenically induced cerebral edema. (*J Am Vet Med Assoc* 2000;216:68–70)

Farming of elk (*Cervus elaphus*) in western Canada and North America has been increasing steadily since 1992. The provinces of Alberta and Saskatchewan have had the greatest increase in numbers of elk and represent 44.9 and 38.6%, respectively, of the total number (34,979) of elk farmed in Canada. The estimated value of these animals is \$390 million, generating approximately \$7.1 million from the annual harvest of antlers.<sup>1</sup>

Presently, the primary market is velvet; therefore,

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there is strong interest in breeding elk. Calves from breeder and producer units represent a substantial financial investment. Neonatal elk have many of the same conditions as bovine calves, namely diarrhea, respiratory tract disease, and parasitism, all of which may be compounded by a lack of colostrum.<sup>2</sup> As a result, veterinarians are examining more elk calves, about which little is described. The purpose of the study reported here was to characterize hypernatremia in neonatal elk calves, including clinical signs, incidence, and physical examination findings, and to propose a cause for this condition.

## Criteria for Selection of Cases

Medical records of all neonatal elk calves < 2 months old admitted to the Western College of Veterinary Medicine (WCVN) between 1988 and 1998 were reviewed. Only those records containing biochemical and hematologic data or blood gas analysis performed at the time of admission (ie, before any treatment was instigated by our clinicians) were included. Of the 26 elk calves included in our study, 4 were evaluated twice and represent 2 cases for the purpose of statistical analyses; therefore, a total of 30 cases were analyzed. Four additional calves with orthopedic problems met the age criterion but were excluded from the study because of lack of appropriate blood analysis.

## Procedure

All calves had a complete physical examination. A subjective analysis of hydration status was ascertained by skin tent time and enophthalmos. Objective measures of hydration status were PCV and total serum protein (TP) concentration. Results of biochemical, hematologic, and blood gas analyses were reviewed and tabulated to include pH, blood gas tensions, serum electrolyte concentrations, hepatic enzyme (sorbitol dehydrogenase,  $\gamma$ -glutamyltransferase, aspartate aminotransferase) activities, total bilirubin concentration, renal function variables (serum urea nitrogen and creatinine concentrations), and blood glucose and albumin concentrations. Clinical indices included rectal temperature, pulse rate, and respiratory rate. Hematologic values of total and differential WBC count, PCV, and TP were recorded. Clinical signs, treatments, final outcome (died or survived), and diagnosis following postmortem examination, if appropriate, were also evaluated.

Calves were allotted into 2 groups on the basis of serum sodium concentrations. Serum sodium concentration in healthy elk calves is  $141 \pm 6$  mEq/L (mean  $\pm$  SD).<sup>2</sup> The hypernatremic group was defined as having serum sodium concentration > 2 SD (ie, 95% of the population) above the mean sodium value (> 153

mEq/L). All data were analyzed by use of a paired *t*-test,  $\chi^2$  analysis, and stepwise logistic regression. Values of *P* < 0.05 were considered significant.

## Results

The most common clinical sign of hypernatremia in the elk calves of this report was diarrhea (*n* = 14; 46.6%), followed by dehydration (5; 16.6%), signs of depression and anorexia (3; 10.0%), pneumonia (3; 10.0%), convulsions or collapse (2; 6.6%), stiffness (2; 6.6%), and omphalophlebitis (1; 3.3%). Of the 14 elk with diarrhea, 5 were not given antibiotics, 3 had been given 1 antibiotic, and 6 had been given > 1 antibiotic. In addition, 5 had been given fluids or electrolytes orally by the owner, and 1 had been given a vitamin E-selenium injection. The duration of illness prior to evaluation at the WCVI varied between 12 hours and 6 days.

Serum sodium concentrations were measured in 26 calves; 14 (53.8%) were hypernatremic (mean sodium concentration, 166.8 mEq/L) and 12 (46.2%) were normonatremic (mean sodium concentration, 146.0 mEq/L). Hypernatremia was significantly associated with high WBC count, anion gap, and chloride, urea, creatinine, and albumin concentrations (Table 1). Diarrhea was also significantly associated with hypernatremia; it was observed in 9 of 14 calves with hypernatremia and only 5 of 12 calves with normonatremia. The calves with hypernatremia had a mean respiratory rate of 28 breaths/min, mean pulse rate of 92.1 beats per minute (bpm), and mean rectal temperature of 38.23 C, which was not significantly different from the rates and temperatures in normonatremic calves (32 breaths/min, 118.9 bpm, and 39.01 C, respectively; *P* = 0.30, 0.10, and 0.10, respectively).

Of the 26 neonatal elk, 12 (40%) died. Of these, 8 were hypernatremic. A necropsy was performed on all dead calves, and the final necropsy diagnoses included undifferentiated enteritis (*n* = 5; 41.6%), pneumonia (3; 25%), omphalophlebitis (2; 16.6%), acute hepatitis (1; 0.08%), and polyarthritides-polyserositis (1; 0.08%). Emaciation was observed in 2 calves on postmortem examination. Renal disease was not detected in any animal at gross necropsy or on histologic examination, and evidence of acute cerebral edema was observed in only 1 calf.

Neither hypernatremia nor diarrhea was associated with survival. In the hypernatremic group, calves treated with lactated Ringer's solution IV were significantly (*P* = 0.03) more likely to survive than those given other

Table 2—Variables significantly associated with survival in diseased elk calves

Variable	Calves survived (n = 14)	Calves died (n = 12)	<i>P</i> value
Albumin (g/100 ml)	2.96 ± 0.66*	2.0 ± 0.49*	0.005
Rectal temperature (C)	39.1 ± 1.01*	38.1 ± 1.2*	0.025
No. calves receiving antibiotics on farm	8	10	0.033
No. calves receiving fluids orally on farm	5	8	0.035

\*Mean ± SD.

fluid replacement IV, such as 5% dextrose, saline (0.9% NaCl) solution, or nonlactated Ringer's solution. Of the 14 calves with hypernatremia, 7 were given electrolytes orally prior to admission. These calves were significantly (*P* = 0.03) more likely to die (Table 2). Stepwise linear regression revealed that older calves and calves with a higher rectal temperature were more likely to survive (Table 2).

Two calves that received treatment at the WCVI had serial blood gas and electrolyte analyses performed every 4 hours during the first day of treatment. These animals were selected as being indicative of the general referral population. They did not have evidence of neurologic disturbance prior to treatment, and the treatment protocol was followed in order to establish how rapidly serum sodium concentration could be lowered. Reduction in serum sodium concentration at a rate > 1.7 mEq/L/h was associated with neurologic disturbances characterized by recumbency, nystagmus, blood from the nares, and signs of depression. Treatment with 25% mannitol, at a dosage of 0.63 g/kg (0.286 g/lb) of body weight by slow IV administration for a 15-minute period, improved the neurologic signs.

## Discussion

Hypernatremia affected approximately half of the neonatal elk admitted to our clinic. In our experience, hypernatremia is more common in elk calves than in other large animal neonates (eg, bovine calves with diarrhea are more likely to develop hyponatremia).<sup>3</sup>

The typical elk calf with hypernatremia had diarrhea, dehydration, weakness, and signs of depression. Seven of 14 calves with hypernatremia and 6 of 12 calves with normonatremia had been given some form of electrolyte solution orally prior to admission to our clinic.

Studies performed on rats indicate that movement of sodium, water, or both between the blood and CSF in acute hypernatremia is greater in juveniles, and osmoreceptors in juveniles are more sensitive than in adults.<sup>4</sup>

Table 1—Variables significantly associated with hypernatremia in diseased elk calves

Variable* (mean ± SD)	Sodium status of elk		<i>P</i> value	Reference values§
	Hypernatremic† (n)	Normonatremic† (n)		
Chloride (mEq/L)	132.7 ± 17.9 (14)	113.8 ± 7.7 (12)	0.003	103 ± 4
Urea (mg/100 ml)	128.3 ± 68.3 (10)	21.0 ± 16.5 (6)	0.002	21.6 ± 5.9
Creatinine (mg/100 ml)	4.9 ± 3.1 (10)	1.85 ± 1.3 (6)	0.036	1.87 ± 0.34
Anion gap	21.9 ± 11.3 (13)	12.8 ± 4.97 (12)	0.014	NA
Albumin (g/100 ml)	2.8 ± 0.7 (10)	2.0 ± 0.6 (6)	0.030	4.1 ± 0.5
WBC count (X 10 <sup>3</sup> /μl)	17.27 ± 14.27 (11)	4.49 ± 2.38 (8)	0.023	4.57 ± 7.76

\*For conversion to SI units, see *The Merck Veterinary Manual*.<sup>11</sup> † > 153 mEq/L. ‡ ≤ 153 mEq/L. § For reference values, refer to Haigh et al.<sup>2</sup>  
NA = Not available.

The increased sensitivity of osmoreceptors may explain why young human patients and those with lesser degrees of hypernatremia have a higher rate of survival.<sup>5</sup>

Administration of electrolyte solutions designed for bovine calves is a common practice among elk farmers. Of the 26 elk calves admitted to the WCVL, 13 had been given electrolytes orally. If these solutions are reconstituted according to manufacturers' guidelines, they contain sodium concentrations of 120 mEq/L<sup>a</sup> or 105 mEq/L.<sup>b</sup> For diarrheic bovine calves, electrolyte solutions with high sodium contents are necessary, because these calves are often hyponatremic. Therefore, these would not be the solutions of choice for treatment of elk calves.

Hypernatremia is a recognized problem in human geriatric<sup>6,7</sup> and pediatric<sup>8</sup> medicine; it is difficult to treat and is associated with a high rate of mortality.<sup>9</sup> Electrolyte solutions designed for human rehydration contain sodium concentrations of 75<sup>c</sup> to 90<sup>d</sup> mEq/L. For fluid maintenance, however, sodium content is reduced to 45<sup>e</sup> or 50 mEq/L.<sup>10,f</sup> Dilutions (1:2 or 1:4) of the commercially available bovine oral rehydration solutions to reduce sodium content or the use of preparations with low sodium concentrations may be more effective ways of replacing fluid losses associated with diarrhea without the risk of inducing hypernatremia.

The exact cause of hypernatremia in most elk calves was not evident. In 2 calves it was associated with unregulated access to a salt mineral block. However, because hypernatremia was significantly associated with diarrhea, it may be associated with a large free water loss. Serum urea and creatinine concentrations were significantly higher in those animals with hypernatremia (Table 1), and may represent prerenal or renal azotemia. Fluid losses from evaporative respiration may also be important. Because of the association of diarrhea with hypernatremia, we believe that severe water losses are compounded by prerenal insufficiency and respiratory water losses.

In our experience, treatment of hypernatremic elk calves is difficult. Intravenous fluid administration is the mainstay of treatment. However, achieving the accurate balance between speed of rehydration and the decrease in serum sodium concentration is complex. There is a risk of rehydrating the animal too rapidly, which may cause cerebral edema; subsequent diuresis needs to be adequate to control for this. Lactated Ringer's solution has one of the lowest sodium concentrations (130 mEq/L) of the commercially available electrolyte solutions. This, in combination with the significant difference in survival rates, suggests that lactated Ringer's solution would be the solution of choice in the hypernatremic animal. Mannitol was

used to effect diuresis in 2 calves and appeared to be effective in improving neurologic disturbances. Although this number is not significant, it serves to illustrate the need for careful clinical assessment and the potential for causing cerebral edema.

The reduction in survival associated with antibiotic administration by farmers is paradoxical. The association may be secondary to other factors, such as late evaluation by a veterinarian, or that the calves in question were more ill than those animals that had not been given antibiotics.

It may be beneficial to reduce evaporative water losses and to administer low sodium electrolyte solutions in elk calves with hypernatremia. Collection of urine samples would be useful to characterize the degree of renal compromise in these animals. The addition of high molecular weight solutes, such as dextran, in fluids administered IV may allow for faster correction of hypernatremia, without the complication of cerebral edema.

<sup>a</sup>Revibe, Langford, 400 Michener Rd, Guelph, ON, Canada.

<sup>b</sup>Entrolyte LifeGuard, Smith Kline Beecham Animal Health, Mississauga, ON, Canada.

<sup>c</sup>Rehydralyte, Ross Products Inc, Columbus, Ohio.

<sup>d</sup>WHO-UNICEF Oral Rehydration Salts, Omen Drugs Pvt. Ltd, Govindpura, Bhopal, India.

<sup>e</sup>Pedialyte, Ross Products Inc, Mont, QC, Canada.

<sup>f</sup>Infalyte, Mead Johnson Nutritional Group, Evansville, Ind.

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