# Further Studies on the Clinical Features and Clinicopathological Findings of a Syndrome of Metabolic Acidosis with Minimal Dehydration in Neonatal Calves

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## ABSTRACT

**A syndrome of metabolic acidosis of unknown etiology was diagnosed in twelve beef calves 7 to 31 days old. Principal clinical signs were unconsciousness or depression concomitant with weakness and ataxia. Other signs included weak or absent suckle and menace reflexes, succussable nontympanic fluid sounds in the anterior abdomen, and a slow, deep thoracic and abdominal pattern of respiration. The variation in clinical signs between calves was highly correlated ($r = 0.87$, $P < 0.001$) with their acid-base (base deficit) status. Abnormal laboratory findings included reduced venous blood pH, $pCO_2$ and bicarbonate ion concentration as well as hyperchloremia, elevated blood urea nitrogen, increased anion gap and neutrophilic leukocytosis with a left shift. Sodium bicarbonate solution administered intravenously effectively raised blood pH and improved demeanor, ambulation and appetite. All calves did well following a return to a normal acid-base status.**


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


## RÉSUMÉ

**Les auteurs ont diagnostiqué un syndrome d'acidose métabolique, d'étiologie inconnue, chez 12 veaux de boucherie, âgés de sept à 31 jours. Les principaux signes cliniques se caractérisaient par de l'inconscience ou de la dépression qui s'accompagnaient de faiblesse et d'ataxie. D'autres signes incluaient la faiblesse ou l'absence du réflexe de la tétée et de celui des menaces, des bruits de liquide, non associés au tympanisme et audibles au ballottement de la partie antérieure de l'abdomen, ainsi qu'un lent profil de respiration thoracique et abdominale profonde. La variation des signes cliniques d'un veau à l'autre dépendait beaucoup ($r = 0.87$, $P < 0.001$) de leur degré d'acidose. Les résultats d'épreuves de laboratoire révélèrent une baisse du pH du sang veineux, de la $pCO_2$ et de la concentration en ions bicarbonate, ainsi que de l'hyperchlorémie, une élévation du taux sanguin d'urée, une augmentation du déficit d'anions et une leucocytose neutrophilique, avec virage à gauche. L'administration intraveineuse d'une solution de bicarbonate de soude éleva efficacement le pH sanguin et améliora le comportement, la démarche et l'appétit. Tous les veaux se portaient bien, après un retour à un équilibre acide-base normal.**


**Mots clés:** veaux, acidose métabolique, bicarbonate de soude.

## INTRODUCTION

Metabolic acidosis is important because of its depressant effects on neurological and cardiovascular function (1-3). It is also thought to be one cause of calf death (4,5). In the neonatal calf, metabolic acidosis is usually found as a sequela to diarrhea induced dehydration (6-11). Recently a syndrome of metabolic acidosis has been reported in four neonatal calves which lacked signs of obvious dehydration (12). The primary purposes of the study reported here were to provide additional information regarding the clinical features and clinicopathological findings in calves with metabolic acidosis without clinical signs of dehydration and to evaluate the relationship between acidosis and clinical signs.

In a previous study this syndrome responded to treatment with isotonic sodium bicarbonate (12). However, the large volumes of fluid used raised the question as to whether improvement was due to correction of acidosis or to volume expansion by fluid. A study in dogs with experimentally produced metabolic acidosis has shown that expansion of extracellular volume alone can restore plasma bicarbonate ion concentration to normal via a marked increase in renal hydrogen ion secretion (13). Therefore, an additional objective of this study was to answer this question by comparing the response to therapy with saline versus sodium bicarbonate solutions.

## MATERIALS AND METHODS

### POPULATION STUDIED

Calves in this study were selected from calves ≤ 31 days of age exam-

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ined between March 31, 1983 and April 1, 1984 at the Western College of Veterinary Medicine (WCVM). A venous blood gases profile was obtained when clinical signs of either unconsciousness or depression concomitant with weakness and ataxia were observed in calves with apparent normal hydration and the *absence* of other obvious illnesses (e.g. diarrhea, omphalitis, arthritis, septicemia, pneumonia). Hydration was judged to be clinically near normal if the eye and third eyelid occupied a normal position within the orbit. Criteria for the diagnosis of metabolic acidosis were venous pH $< 7.250$ and bicarbonate ion [$HCO_3^-$] concentration $< 20$ mmol/L.

Twelve calves were selected for study. Historically, all calves were presented to the WCVM because of anorexia and a striking depression or comatose behavior. Eight calves were considered by their respective owners to have experienced variable degrees of diarrhea. Whereas a mild diarrhea was self-limiting after 24 to 48 hours duration in two calves, four calves had received oral fluid and electrolytes of variable volume (0.5-1.0 L) and frequency of administration (single treatment to three times daily) over a highly variable duration of treatment (one day to intermittently over seven days). Two additional calves had received only oral and/or parenteral antibiotics.

CLINICAL STUDIES

The physical condition of each calf was assessed upon admission into the WCVM (zero hours) and four hours later and assigned a numerical score (Table I). The battery of clinical signs chosen for score assignment reflected neurological (suckle reflex, ability to stand, menace reflex, tactile response) and cardiovascular (warmth of extremities and oral cavity) function. The scoring system was developed to determine the relationship of clinical signs to severity of acidosis and to monitor each calf's clinical response to treatment within each fluid group.

*Hematological Studies* — A complete blood count consisting of packed cell volume (PCV), hemoglobin concentration, red blood cell indices, and white blood cell count with differential leukocyte count was performed at zero hours by methods previously described (12). Additional PCV determinations were made after four hours and 24 hours of intravenous fluid administration.

*Biochemical Determinations* — Serum sodium, potassium, chloride, calcium, phosphorus, magnesium, blood urea nitrogen (BUN), blood glucose and total serum protein (including albumin, globulin and electrophoretic pattern) were determined at zero hours.

Chemical methods employed for the determination of calcium, phosphorus and magnesium were based on a colorimetric reaction. Calcium, phosphorus and magnesium were complexed with the dyes O-Cresol-Phthalein (A-Gent Calcium, Abbott Laboratories, Irving, Texas), calmagite (Inorganic Phosphorus, D.K. 1120-15, Fisher Scientific Ltd, Ottawa, Ontario), and Molybdenum Blue (Magnesium, D.K., 1100-12, Fisher Scientific Ltd, Ottawa, Ontario) respectively, and measured with a spectrophotometer (Biochromatic Analyzer 100, Abbott Laboratories, Irving, Texas). All other biochemical results were determined by methods previously described (12).

*Acid-Base Studies* — Venous blood pH, bicarbonate ion concentration, partial pressure of carbon dioxide ($pCO_2$), and base deficit were determined at zero hours and after four hours and 24 hours of intravenous fluid administration. An automated blood gas analyzer was used as previously described (12).

*Organic Acid and Ketoacid Studies* — Lactate, acetate, and ketoacid (acetoacetate) concentrations were determined on blood samples obtained from the jugular vein of each calf at entry (zero hours). Methods employed to measure each acid have been previously described (12).

*Anion Gap Concentration* — Anion gaps were calculated as the difference between cations (sodium + potassium) and anions (chloride + bicarbonate).

FLUID COMPARISONS

Twelve calves were randomly assigned to each of two fluid groups (six calves/group), weighed, fitted with a 14 ga intravenous catheter

**TABLE I. Numerical Scoring System to Quantify Clinical Signs in Calves with Metabolic Acidosis (expressed as a depression score)**

| Variable | Method of Assessment | Score[a] | Interpretation |
|---|---|---|---|
| Suckle reflex | Index finger in mouth | 0 | Strong organized suckle |
| | | 1 | Weak coordinated suckle |
| | | 2 | Disorganized chewing |
| | | 3 | absent |
| Menace reflex | Rapid hand movement toward eye | 0 | Strong instantaneous reflex |
| | | 1 | Slow delayed reflex |
| | | 2 | absent |
| Tactile response | Skin pinched over lumbar area | 0 | Skin twitch, head movement toward flank |
| | | 1 | Skin twitch, no head movement toward flank |
| | | 2 | No skin twitch, no head movement toward flank |
| Ability to stand | Manually prod ribcage with pen | 0 | Ability to stand |
| | | 2 | Inability to stand |
| Warmth of oral cavity | Fingers in contact with Mucosa of hard/soft palate | 0 | Normal mucosa warmth |
| | | 1 | Cool mucosa |
| | | 2 | Cold mucosa |
| Warmth of extremities | Hand clasped around fetlock | 0 | Normal skin warmth |
| | | 1 | Cool skin |
| | | 2 | Cold skin |

[a]The score for each variable is added to yield a minimum score of zero in healthy calves and a maximum possible score of 13 in severely affected calves

(Surflo, Fuji Terumo, Tokyo, Japan) and infused with either isotonic (150 mmol/L) sodium chloride (saline control) or isotonic (150 mmol/L) sodium bicarbonate solution. The initial quantity of fluid administered to each calf was calculated from the following formula:

Volume of fluid, L = body weight, kg x 0.3 x base deficit, mmol/L ÷ 150, where 0.3 represents a constant for the volume of distribution of bicarbonate in extracellular fluid and 150 represents the millimolar concentration of sodium chloride and sodium bicarbonate in each fluid. Therefore, the initial volume of fluid delivered intravenously to each calf was dependent upon body weight and severity of acidosis.

Immediately following a four hour period of fluid administration each calf's acid-base status and PCV were evaluated. A numerical score for clinical signs (depression score) was also determined; the evaluator was unaware of the treatment administered.

The experiment was stopped at four hours and any calves that remained acidotic, i.e. base deficit > 12 mmol/L, were administered sodium bicarbonate in an amount calculated to correct the remaining base deficit. On the other hand, when base deficit was < 12 mmol/L, calves were placed on a lactated Ringer's solution at maintenance levels until hour 24 postadmission.

## CONTROL POPULATION

Twelve clinically normal calves 26.3 ± 18.5 (mean ± 1 SD) days of age from two farms were used. There were four purebred Charolais calves, four cross-bred Charolais calves and four cross-bred Simmental calves. The calves were held with minimal restraint in a standing position while blood was collected aseptically from a jugular vein. Samples were collected into serum tubes for sodium, potassium, chloride, calcium and magnesium determinations. Samples were collected anaerobically for evaluation of blood gases and the blood gas values were corrected for calf temperature and hemoglobin concentration. Assays were performed as described above.

## STATISTICAL ANALYSIS

A TI-59 (Texas Instruments, Inc., Dallas, Texas) programmable electronic calculator and statistical package were used to perform correlation analyses according to the method of Hay's (14). Blood hematology and chemistry values obtained upon admission were compared to the control population or reference values for normal calves using Student's t-test and the sample means and standard deviations. Means were compared between treatments using a one-tailed Student's t-test. Paired t-

**TABLE II. Signalment and Clinical Signs in Calves with a Distinct Metabolic Acidosis**

| Signalment | | | | | Clinical Signs | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Animal | Breed | Sex | Age (days) | Wt (kg) | Demeanor | Ambulation | Menace Response | Lumbar Skin Twitch | Suckle Reflex | Gut[b] Sounds | Other |
| 1 | Charolais | M | 15 | 60 | D[a] | A | N | N | DC | P | |
| 2 | Charolais | M | 21 | 60 | D | A | W | W | SC | P | Deep thoracic and abdominal respiration |
| 3 | Simmental | F | 10 | 51 | D | R | W | N | SC | M | Deep thoracic and abdominal respiration |
| 4 | Charolais | F | 11 | 43 | C | R | Ab | Ab | Ab | P | Hypothermia; slow deep thoracic and abdominal respiration |
| 5 | Charolais | M | 16 | 49 | D | A | N | N | DC | P | Cool extremities; slow deep thoracic and abdominal respiration |
| 6 | Charolais | F | 11 | 42 | D | R | N | N | DC | P | Hypothermia; slow deep thoracic and abdominal respiration |
| 7 | Charolais | F | 7 | 47 | D | A | Ab | N | DC | P | |
| 8 | Charolais | F | 14 | 49 | D | R | Ab | N | Ab | P | Cool extremities; slow deep thoracic and abdominal respiration |
| 9 | SimmentalX | M | 14 | 44 | D | A | W | N | DC | Ab | |
| 10 | CharolaisX | F | 14 | 52 | D | R | Ab | W | DC | P | |
| 11 | Simmental | M | 31 | 62 | D | A | N | N | SC | M | |
| 12 | SimmentalX | M | 14 | 58 | D | R | Ab | N | DC | P | Slow deep thoracic and abdominal respiration |

[a]D = Depressed, A = Ataxia, N = Normal, DC = Disorganized Chewing, P = Prominent, SC = Slow Coordinated, R = Recumbent, M = Moderate, C = Comatose, Ab = Absent, W = Weak

[b]Assessed by succusion of abdomen

tests were used to compare changes from zero to four hours within treatment groups.

## RESULTS

### CLINICAL OBSERVATIONS

All calves studied were either of Simmental or Charolais parentage and the majority were entering their second week of life (Table II). There was no sex predilection. All calves were in moderate to good physical condition and defecated pasty or mucoid formed feces. Hydration appeared clinically normal since the calves had a normal position of the eye and third eyelid within the orbit and "tenting" of the skin over the upper eyelid took one to five seconds to return to normal.

Eleven calves (92%) were depressed and one calf presented unconscious. Six of the twelve calves (50%) displayed varying degrees of fore and/or rear leg ataxia and weakness while the remaining six calves presented with such profound weakness that they preferred to remain recumbent despite efforts to assist them to stand. Although all calves had intact direct and indirect pupillary light reflexes, 5 out of 12 calves (42%) completely lacked a menace reflex and another three calves (25%) responded poorly with a slow delayed closure of eyelids.

The response of calves to a skin pinch applied over the lumbar area was more variable. The majority of calves had a normal response, i.e. skin twitch and movement of head and neck toward flank. However, two calves responded with only a skin twitch and one calf failed to exhibit a response. All calves possessed abnormal suckle reflexes. Whereas two calves did not exhibit a response, three calves had a coordinated but slow suckle reflex. Chewing behavior in response to a finger in the mouth was seen in 7 out of 12 (58%) calves.

When the abdomen was succussed, fluid sounds were auscultated in 11 (92%) calves. A slow, deep thoracic and abdominal breathing pattern was present in seven (58%) calves. Hypothermia ($\leq 37°C$) occurred in two calves.

### HEMATOLOGICAL AND BIOCHEMICAL DETERMINATIONS

The hematological and biochemical findings are presented in Table III. Mean venous blood pH, $pCO_2$, $HCO_3^-$ concentrations and base deficits in the

**TABLE III. Initial Hematology and Biochemistry Values in Calves with a Distinct Metabolic Acidosis**

| | | Acidotic Calves | | | Reference Values | | | |
|---|---|---|---|---|---|---|---|---|
| Variable | Units | Mean | SD | N | Mean | SD | N | p[a] |
| Blood gas (venous) | | | | | | | | |
| pH | | 7.087 | 0.098 | 12 | 7.341[b] | 0.042 | 12 | < 0.001 |
| $pCO_2$ | TORR | 34.37 | 6.86 | 12 | 57.2[b] | 4.38 | 12 | < 0.001 |
| $HCO_3^-$ | mmol/L | 10.43 | 2.6 | 12 | 30.3[b] | 3.8 | 12 | < 0.001 |
| Base deficit | mmol/L | -18.8 | 4.7 | 12 | -4.9[b] | 4.0 | 12 | < 0.001 |
| Erythrocytes | $x10^{12}$/L | 8.48 | 1.8 | 12 | 7.38[c] | 1.14[c] | 64 | 0.007 |
| Hemoglobin | g/L | 111.5 | 8.48 | 12 | 105.0[c] | 19.0[c] | 64 | NS |
| Hematocrit | L/L | 0.34 | 0.08 | 12 | 0.33[c] | 0.06[b] | 17 | NS |
| Leukocytes | $x10^9$/L | 12.98 | 3.79 | 12 | 8.5[c] | 2.0[c] | 17 | < 0.001 |
| Neutrophils | | | | | | | | |
| Segmented | $x10^9$/L | 6.7 | 3.2 | 12 | 2.7[c] | 1.6[b] | 17 | < 0.001 |
| Band | $x10^9$/L | 0.78 | 0.97 | 12 | NA | NA | | NS |
| Lymphocytes | $x10^9$/L | 4.4 | 1.1 | 12 | 5.1[c] | 1.5 | 17 | 0.181 |
| Monocytes | $x10^9$/L | 0.58 | 0.38 | 12 | 0.6[c] | 0.3 | 17 | NS |
| Serum total protein | g/L | 62.17 | 7.85 | 12 | 64.5[d] | 13.4 | 9 | NS |
| Albumin | g/L | 29.58 | 3.4 | 12 | 27.0[d] | 5.5 | 9 | NS |
| Gammaglobulin | g/L | 10.9 | 5.15 | 12 | 12.4[d] | 3.9 | 9 | NS |
| Fibrinogen | g/L | 6.1 | 2.31 | 12 | | NA | | NA |
| Sodium | mmol/L | 140.0 | 5.5 | 12 | 141.4[b] | 2.6 | 12 | NS |
| Potassium | mmol/L | 5.2 | 0.7 | 12 | 5.3[b] | 0.4 | 12 | NS |
| Chloride | mmol/L | 110.7 | 6.8 | 12 | 103.5[b] | 3.2 | 12 | < 0.003 |
| Calcium | mmol/L | 2.7 | 0.35 | 12 | 2.6[b] | 0.19 | 12 | NS |
| Phosphorus | mmol/L | 3.0 | 0.76 | 12 | 2.9[b] | 0.15 | 12 | NS |
| Magnesium | mmol/L | 0.92 | 0.13 | 12 | 0.88[b] | 0.14 | 12 | NS |
| Anion gap | mmol/L | 24.0 | 5.5 | 12 | 12.8[b] | 2.4 | 12 | < 0.001 |
| Urea nitrogen | mmol/L | 11.2 | 4.4 | 12 | 4.27[b] | 1.6 | 12 | < 0.001 |
| Glucose | mmol/L | 4.6 | 0.90 | 12 | 4.39[d] | 0.61 | 90 | NS |
| Lactate | mmol/L | 0.58 | 0.27 | 12 | 0.87[e] | 0.32 | 46 | 0.006 |
| Acetate | mmol/L | 0.10 | 0.099 | 12 | 0.21[e] | 0.21 | 26 | 0.103 |
| Acetoacetate | mg/dL | ND | | | | | | |

[a]Probability that means for acidotic and reference calves are different using a two tailed Student's t-test
[b]Values for control calves
[c]Reference 15. Erythrocyte values are for 14 day old calves, leukocyte values for 15 to 21 day old calves
[d]Reference 16
[e]For Holstein calves, assays performed in author's laboratory
NS = Not significant, $p > 0.2$
NA = Not available
ND = Not detectable, < 2 mg/dL

twelve calves were significantly ($p < 0.001$) lower than reference values for calves of similar ages. Acidotic calves also had a significant elevation in anion gaps ($p < 0.001$) and serum chloride ($p < 0.003$) concentrations. The PCV and hemoglobin concentrations were similar to reference values. As a group these calves had a neutrophilic leukocytosis with a left shift which was compatible with a mild inflammatory reaction.

Serum urea nitrogen concentrations were significantly ($p < 0.001$) higher than reported values for healthy calves of similar age. Plasma glucose was normal. Whole blood lactate, acetate and acetoacetate concentrations were not elevated.

### CLINICAL SIGNS SCORES

A significant correlation was found between the zero hour score of clinical signs (depression score) and base deficit values in the twelve calves ($r = 0.87$, $P < 0.05$, Fig. 1). Variation in acid-base status accounted for 76% of the variability in neurological and cardiovascular signs.

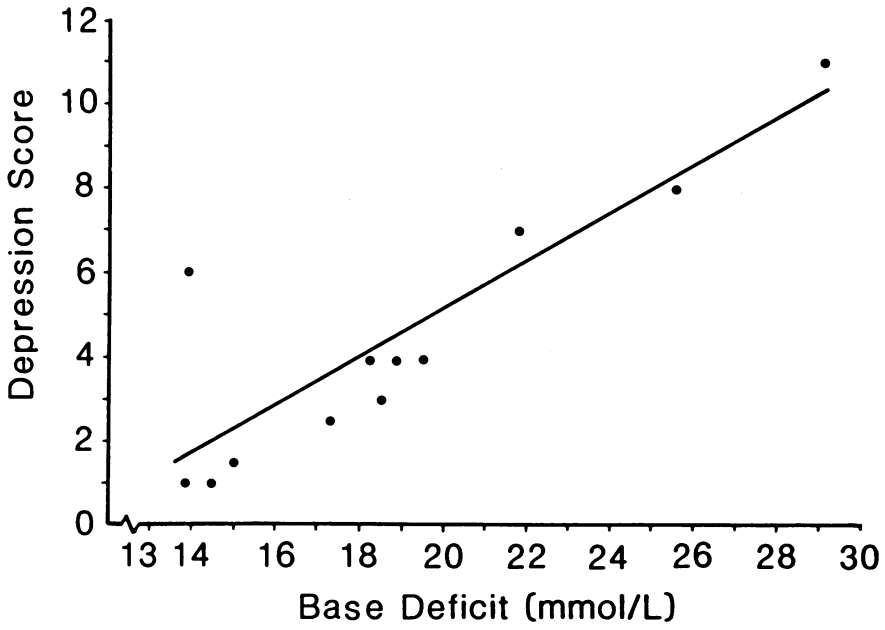


**Fig. 1. Relationship between zero hour depression score and base deficit in 12 calves with a distinct metabolic acidosis. The solid line represents the linear regression line ($r = 0.87$, $p < 0.05$).**

### FLUID COMPARISONS

The zero hour values for weight, PCV, depression score and blood gases values are shown in Table IV. At the beginning of the trial there was a tendency for calves assigned to bicarbonate therapy to be of lighter weight and more acidotic than calves in the saline group. Following four hours of treatment (Table V) with saline there were no significant changes in blood gas values although depression scores had tended to improve. In contrast, calves that received bicarbonate showed significant improvements in blood gases values (for all analyses, $p < 0.001$) and depression score ($p < 0.047$). Improved ability to stand and stronger menace and tactile reflexes were responsible for the lower depression scores in these calves. Both groups of calves had a similar net reduction in PCV following therapy (Table V).

At the conclusion of 24 hours of bicarbonate and lactated Ringer's therapy most calves were clinically normal. The residual depression scores of $0.6 \pm 1.7$ (mean $\pm$ SD) were primarily a reflection of abnormal suckle reflex. All calves survived and were subsequently released from hospitalization.

**TABLE IV. Comparison of Weight, Clinical Signs Score, PCV and Blood Gases Values at Zero Hours in Calves Assigned to Saline or Bicarbonate Treatment Groups**

| Variable | Treatment Group | | Significance[a] |
|---|---|---|---|
| | Saline (Control) | Bicarbonate | P |
| Weight, kg | 55.4 ± 2.17[b] | 47.5 ± 2.72 | 0.046 |
| Clinical signs score | 3.0 ± 0.74 | 5.8 ± 1.45 | 0.112 |
| PCV, L/L | 0.30 ± 0.026 | 0.37 ± 0.03 | 0.104 |
| pH | 7.140 ± 0.023 | 7.034 ± 0.048 | 0.054 |
| Bicarbonate, mmol/L | 11.5 ± 0.51 | 9.4 ± 1.33 | 0.172 |
| Base deficit, mmol/L | 16.2 ± 0.94 | 21.5 ± 2.15 | 0.050 |

[a]Probability that difference between means is due to chance; two tailed Student's t-test
[b]Values are means ± 1 standard error. There are six calves in each treatment group

**TABLE V. Comparison of Changes in Clinical Signs Score, PCV and Blood Gases Values Between Zero and Four Hours in Calves Assigned to Saline or Bicarbonate Treatment Groups**

| Variable | Treatment Group | | Significance[a] |
|---|---|---|---|
| | Saline (Control) | Bicarbonate | P |
| Depression score | -0.50 ± 0.26[b] | -2.33 ± 0.96 | 0.047 |
| PCV, L/L | -0.032 ± 0.013 | -0.027 ± 0.014 | > 0.2 |
| pH | -0.006 ± 0.009 | 0.217 ± 0.027 | < 0.001 |
| Bicarbonate, mmol/L | 0.0 ± 0.29 | 9.5 ± 1.04 | < 0.001 |
| Base deficit, mmol/L | -0.3 ± 0.50 | -13.9 ± 1.36 | < 0.001 |

[a]Probability that difference between means is due to chance; one tailed Student's t-test
[b]Values are computed by subtracting the zero hour values from the four hour values. Results are expressed as means ± 1 standard error. There are six calves in each treatment group

## DISCUSSION

The test calves had a severe metabolic acidosis. The high correlation between the severity of acidosis and variation in clinical signs of the calves as well as the response to bicarbonate therapy suggests this was of major clinical significance. Changes in severity of metabolic acidosis (base

deficit) accounted for 76% of the variation in the clinical signs score of the calves. Clinical signs referable to deranged neurological function would be expected to occur initially in animals with a pure metabolic acidosis since the major effect of acidosis on the body is depression of the central nervous system (3). Accordingly, examination of individual calves in the present study was prompted by owner concern over the striking depression to comatose behavior exhibited by each. Furthermore, it should be emphasized that no other concurrent primary disease process (e.g. bronchopneumonia) was identified in any calf which could impact on clinical signs. Also, a follow-up telephone conversation with each owner approximately two to four weeks after release of a calf indicated that all did well as a result of specific alkalinizing therapy.

Treatment with isotonic sodium bicarbonate was more effective than saline (control fluid) in improving demeanor and ameliorating the acidosis. Saline therapy alone resulted in no significant changes in the status of acidosis and only slight improvements in clinical condition (depression score). These results indicate that correction of acidosis, not simply providing extracellular fluid, was the key to successful therapy.

Bicarbonate requirements to treat the acidosis were calculated assuming a constant of 0.3 for the volume of distribution of bicarbonate ion in extracellular fluid (17). However, there was only a partial correction of acidosis; base deficit improved by only 13.9 mmol/L rather than the expected amount of 21.5 mmol/L. The mean calf weight was 47.5 kg so the volume of distribution of bicarbonate in these acidotic calves was 0.44. This result, together with other studies (18) suggests that values of 0.5 would be more appropriate than 0.3 when calculating bicarbonate requirements in calves.

The calves had a normal position of the eye and third eyelid within the orbit as well as normal PCV and serum total proteins. These findings also suggest that marked dehydration was not present. However, as a group the BUN was increased and during fluid therapy there was a drop in PCV of 0.03L/L; this compares with a fall of 0.16 L/L seen during treatment of clinically dehydrated diarrheic calves (18). Overall the data suggest a mild degree of hypovolemia existed in these calves which escaped critical physical detection. It was the author's opinion that the contribution of dehydration to the generation of acidosis in the calves was minimal based on the lack of calf response to expansion of extracellular fluid volume alone. Anorexia, common to all calves, would have contributed to any dehydration since all neonatal calves rely heavily on a liquid diet.

The present study helps define the cause of the acidosis in these calves. Blood pH and bicarbonate concentrations were low, base deficit was high and the partial pressure of carbon dioxide somewhat low. These findings are indicative of a severe metabolic acidosis with some respiratory compensation. Metabolic acidosis can arise secondary to accumulation of organic or inorganic acids, or to loss of bicarbonate or retention of hydrogen ions (3). The calves had an increased anion gap indicating that the accumulation of organic or inorganic acids was partly responsible for the acidosis (19,20). However, the nature of this acid remains to be determined. Lactic acid and ketoacids are common causes of acidosis due to organic ion accumulation (20) but L-lactate, acetate and acetoacetate concentrations were normal.

Gastrointestinal loss of bicarbonate and subsequent acidosis is normally associated with diarrhea and dehydration in calves (9,21). It is possible that gastrointestinal bicarbonate ion loss contributed to the acidosis in the calves because the decrease in serum bicarbonate was greater than the increase in the anion gap. History indicated that a number of calves had had episodes of diarrhea. The excess fluid sounds on absuccussion of the abdomen indicate normal gastrointestinal function. The mild inflammatory changes in the hemograms might reflect intestinal inflammation.

The hypocarbia indicates a respiratory compensation to the acidosis. Seven out of twelve (58%) calves had exaggerated respiratory excursions and this may reflect respiratory compensation. In people, a slow, deep ventilation (Kussmaul respiration) is usually evident in acute metabolic acidosis (2,3).

Some calves in this study had received an oral fluid and electrolyte solution. It has recently been suggested that lack of alkalinizing agents in some oral fluid and electrolyte solutions might result in correction of dehydration but not acidosis (22). At the time of the study, the oral electrolyte preparation in common use in our practice area contained no alkalinizing agent so use of this type of preparation may have contributed to the syndrome. The neurological and cardiovascular affects of the uncorrected acidosis could perpetuate the syndrome by depressing fluid intake and renal perfusion and thus prevent renal excretion of hydrogen ion and correction of the problem. However, the fact that eight calves in the present study did not receive an oral fluid and electrolyte preparation detracts from an explanation that this particular syndrome is merely a reflection of inappropriate acid-base adjustment during expansion of extracellular fluid volume.

In summary, this paper describes a syndrome of severe metabolic acidosis. The syndrome is unusual in that it is not associated with obvious signs of dehydration or diarrhea. Affected calves are depressed and this correlates with the degree of acidosis. The syndrome responds well to therapy with isotonic sodium bicarbonate but poorly to saline therapy. Bicarbonate requirements can be calculated using the formula:

Bicarbonate required, mmol = body weight, kg x 0.5 x base deficit, mmol/L.

In situations where blood gas analysis is not available an approximation of bicarbonate requirements can be obtained by gauging the severity of clinical signs (Fig. 1). For example, a 50 kg calf which was ataxic and had a depression score of 3 would require 2.6 L of 1.3% sodium bicarbonate solution. A severely affected comatose calf with a score of 10 would require 4.5 L of 1.3% bicarbonate solution.

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