Evaluation of Abomasal Outflow Diversion as an Experimental Model of Hypochloremic, Hypokalemic Metabolic Alkalosis in Lactating Cows

Jeffrey L. Ward, Donald F. Smith, Susan L. Fubini and Daneen M. Deuel-Aromando

ABSTRACT

Four adult, lactating dairy cows were subjected to diversion (loss) of gastric contents through a T-shaped cannula placed in the cranial part of the duodenum just distal to the pylorus. Diversion was continued for 10 to 12 hours, at which point the cows were very weak and depressed. The volume of effluent during this period ranged from 37.3 to 46.8 L, with the largest volume being produced during the first four hours. All cows became dehydrated, with mean packed cell volume and total plasma protein concentration increasing 30% and 19.6%, respectively, but with only a slight increase in plasma creatinine concentration. Plasma Cl^- concentrations decreased from a mean of 97.3 mEq/L at the beginning of diversion to a mean of 87.2 mEq/L at eight hours. This was followed by a plateau or slight increase in concentrations over the final hours of diversion. Plasma K^+ concentration followed a similar pattern, decreasing from a mean of 3.9 mEq/L to a mean of 2.94 mEq/L at six hours, followed by increasing values until termination of diversion. No changes in plasma Na^+ concentration were noted, except for a mild decrease in one cow. Plasma calcium concentrations decreased significantly, reaching 6.6 ± 0.6 mEq/L at the end of diversion. Venous pH, plasma HCO_3^- concentration, and plasma base excess concentration increased during the first four to eight hours of diversion, followed by a gradual decline. Although a mild hypochloremic metabolic alkalosis resulted from diversion of abomasal outflow in all cows, substantiated by a mild increase in plasma strong ion difference, the changes observed were not as great as expected. In addition, the cows did not tolerate the diversion process well, becoming rapidly dehydrated, hypocalcemic, and deteriorating in clinical appearance and physical parameters.

RÉSUMÉ

Quatre vaches adultes en lactation ont été utilisées pour étudier l'effet de la perte du contenu gastrique à travers une canule placée dans la partie crâniale du duodénum. La diversion du contenu gastrique à travers la canule s'est poursuivie pour une période de 10 à 12 heures. Après ce temps, les animaux ont commencé à montrer des signes de faiblesse et d'abattement. Le volume recueilli pendant cette période varie de 37,3 L à 46,8 L. La majeure partie du volume produit est recuellie pendant les quatre premières heures. Toutes les vaches sont devenues déshydratées tel qu'indiqué par l'augmentation de l'hématocrite (30%) et des protéines totales (19,6%). Par contre, la concentration plasmatique de la créatinine n'a augmenté que faiblement. La concentration plasmatique des chlorures s'est abaissée d'une valeur moyenne de 97,3 mEq/L au début de l'expérience à une valeur moyenne de 87,2 mEq/L huit heures plus tard. Cette diminution de la concentration plasmatique des chlorures est suivie d'une légère augmentation ou d'une période plateau dans les dernières heures de l'expérimentation. La concentration plasmatique du potassium est diminuée d'une valeur moyenne de 3,9 mEq/L à une valeur moyenne de 2,94 mEq/L six heures plus tard. De la même façon que les chlorures, le potassium plasmatique montre une légère augmentation dans les dernières heures de l'expérimentation. La concentration plasmatique du sodium ne montre pas de changement, excepté une légère diminution chez seulement une vache. La concentration plasmatique du calcium diminue de façon significative pendant l'expérimentation (valeur moyenne à la fin de l'expérimentation: 6,6 ± 0,6 mEq/L). Le pH veineux, la concentration plasmatique en bicarbonate et l'excès de base plasmatique ont augmenté pendant les quatre à huit premières heures de diversion, pour ensuite diminuer graduellement. Malgré le développement d'une alcalose métabolique hypochlorémique, pendant l'expérimentation la différence des ions forts a diminué faiblement. Les vaches ne semblent pas tolérer cette expérimentation et sont devenues rapidement déshydratées, hypocalcémiques, et leur apparence clinique ainsi que les paramètres physiques se sont détériorés. (*Traduit par Dr Gilles Fecteau*)

Department of Clinical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, New York 14853. Present address of Dr. J. L. Ward: Department of Pharmacology, College of Veterinary Medicine, Cornell University, Ithaca, New York 14853.

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INTRODUCTION

Hypochloremic, hypokalemic metabolic alkalosis (HHMA) is a common metabolic disturbance of adult dairy cattle with gastrointestinal tract disorders such as abomasal displacement and volvulus, vagal indigestion, and mechanical or functional stasis of the intestinal tract (1–4). Gastric fluid, with a high hydrogen and chloride ion concentration, is sequestered in the abomasum or is refluxed by "internal vomiting" into the forestomach compartments (3–5).

Previous experimental models of HHMA in cattle and sheep have resulted from surgical obstruction of the intestine, either by placement of circumferential ligatures around the duodenum or by blockage of reentrant cannulae (6–11). However, many of these reported models did not permit repeated trials on the same animals, nor did they allow for collection and analysis of abomasal effluent because the gastric contents were not diverted to the exterior. While obstruction models may mimic naturally occurring conditions more closely than diversion models, the forestomach distention that occurs with obstruction models may cause respiratory compromise and induce respiratory acidosis, which would tend to complicate evaluation of the metabolic consequences.

Techniques have been described for reentrant cannulation of the cranial part of the duodenum of cattle and sheep (12–14). These methods allow duodenal flow to be reestablished, thus permitting multiple experiments using the same animal. Diversion of abomasal outflow, utilizing these techniques, has been performed in multiple studies in sheep to induce HHMA and to evaluate different treatment strategies (15,16). The usefulness of the model to induce HHMA in cattle has not been evaluated. A preliminary trial in a single cow fitted with a T-type cannula and subjected to diversion of abomasal outflow suggested that HHMA could be caused in cattle by using this technique (Smith DF, Cornell University, Ithaca, New York, unpublished observations, 1985).

The purpose of the present study was to evaluate the usefulness of a one-piece reentrant cannula to induce HHMA by diversion of abomasal outflow when placed in the proximal part of the duodenum of lactating dairy cows.

MATERIALS AND METHODS

Six adult, lactating Holstein-Friesian cows, weighing between 560 and 720 kg, and considered to be clinically normal on the basis of physical examination and hematological profile, were used in this study. All cows were 60 to 120 days pregnant and had been lactating 120 to 180 days at the time of the experiment. Complete data were collected for the four cows in which experimental trials were completed. A complete, balanced diet designed for lactating cows, consisting of a total mixed ration (50:50 forage to concentrate ratio), and water were provided *ad libitum* throughout the experimental period. The average daily feed intake provided approximately 3.7 kg crude protein, 3.8 kg acid detergent fiber (ADF), 290 g potassium, 37 g sodium, and 78 g chloride, with a feed cation/anion balance of +65 mEq/kg DM. The experimental protocol was approved by the University Institutional Animal Care and Use Committee.

SURGICAL PROCEDURE

Each cow was fitted with a T-shaped, single-barrel cannula as described previously (14). Cannulae, collars, and solid plug parts were individually machined from Teflon polymer. The barrel had inside and outside diameters of 23 mm and 27 mm, respectively, and a length of 90 mm. The body had inside and outside diameters of 23 mm and 32 mm respectively, and a length of 110 mm. This type of cannula design was chosen because it is inexpensive and easily fabricated, and it is more likely to last for several lactations when compared to other types. In addition, the simple design of the cannula does not require transection of the duodenum during placement, and obstruction of digesta flow is not as likely to occur as with other cannulas (14).

Feed was withheld for 36 hours and water for 24 hours before surgery. With the cow positioned in left lateral recumbency under general inhalation anesthesia, a 30 cm curved paracostal incision was made 4 cm ventral to and parallel with the costal arch from rib 13 to rib 8. The subcutaneous tissues, cutaneous trunci muscle, and the external rectus sheath were incised in the same manner. The transverse abdominal muscle was incised dorsoventrally, in the direction of its muscle fibers, at the level of the tenth intercostal space. This incision was continued through the internal rectus sheath and peritoneum.

Following entry into the abdomen, the pylorus and proximal 20 cm of the cranial part of the duodenum were exteriorized and separated from the incision by moist towels. The site for cannula insertion was centered 10 cm distal to the pylorus. A 5 to 6 cm long enterotomy was made along the right (parietal) surface of the duodenum and the cannula was inserted into the lumen. The enterotomy was closed adjacent to the barrel of the cannula with No. 1 nylon suture (Ethilon, Ethicon, Somerville, New Jersey) in a simple interrupted pattern. The edges of the enterotomy around the barrel were inverted and drawn snugly around the cannula with a purse-string suture of the same material.

A piece of knitted polypropylene surgical mesh (Marlex, CR Bard and Co., Murrey Hill, New Jersey) was positioned around the duodenum to hold the intestinal wall against the cannula body. The mesh was precut to correspond to the length of the cannula body, and was placed around the duodenum deep to major vessels. The mesh was drawn snugly around the intestine and cannula body with imbricating sutures of 2–0 nylon (Ethilon, Ethicon).

In preparation for exteriorization of the cannula barrel, a 2 cm diameter circular piece of skin was excised from the right body wall in the 10th intercostal space at the level of the scapulohumeral joint. The muscle layers and peritoneum were bluntly incised and the cannula barrel was pushed from the peritoneal cavity through the circular incision in the body wall. The outer collar was then applied to hold the cannula in place.

Incisions were closed in the following manner. Peritoneum and internal rectus sheath were closed with No. 2 chromic gut in a simple continuous

pattern. The rectus abdominous muscle was closed with No. 1 chromic gut in a simple interrupted pattern. The external rectus sheath was sutured with No. 2 polyglactin 910 suture (Vicryl, Ethicon) in a simple interrupted pattern. The cutaneous trunci muscle and subcutaneous tissues were closed with 0 chromic gut in a simple continuous pattern. Skin was closed with No. 1 vetafil in a continuous-interlocking pattern.

Cows were hospitalized for three to five days, starting the morning of surgery, and received 44,000 IU procaine penicillin G (Pfi-Pen G, Pfizer, New York, New York) per kg IM, every twelve hours for five days.

After surgery, all cows were returned to the university-owned teaching and research herd for six to eight months, during which period they were utilized in nutrition and metabolism studies.

EXPERIMENTAL PERIOD

All cows were acclimated for two weeks in metabolism stanchions, permitting collection of urine and feces, as well as measurement of feed and water intake. Cows were milked every twelve hours and milk weights were recorded.

The experimental period consisted of a 24 h baseline period, followed by a diversion period. During the period before diversion, vital signs and feed and water intake were recorded, and base-line (control) measurements of PCV, plasma total protein (TP), venous acid-base, plasma creatinine, and plasma electrolyte concentrations were measured. At 12 h before diversion, a 14 gauge catheter (Abbocath-T, Abbott Hospitals, Inc., N. Chicago, Illinois) was placed and secured in a jugular vein for subsequent blood sampling and intravenous fluid administration. At the beginning of the diversion period (0 hour), the inner plug was removed from the cannula and a stainless steel diversion gate was inserted to cause diversion of abomasal outflow. Effluent was directed through tubing attached to the diversion gate and collected in a 10 L polyethylene reservoir. All cows were milked and the udders stripped prior to the onset of diversion and again at the end of the experiment.

Feed and water intake were measured at 2 h intervals, and fecal and urine production were monitored during the diversion period. All urine collections consisted of free-catch samples, and at the end of each experiment the bladder was catheterized to collect residual urine. At 2 h intervals, abomasal outflow was collected and measured, vital signs were recorded, and 5 mL of venous blood was collected into an evacuated tube containing sodium heparin. Blood samples were placed on ice, and acid-base values, plasma electrolyte concentrations, and PCV and TP were measured within 15 min of collection. Abomasal outflow diversion and sample collection continued until termination of the experiment. The degree of depression and weakness were used as subjective criteria for the discontinuation of diversion, as the predetermined objective criteria (PCV > 50%; $[Cl^-]_{plasma}$ < 65 mEq/L; or $[K^+]_{plasma}$ < 2.0 mEq/L) were not met by any of the cows.

SAMPLE PROCESSING

An automated blood gas analyzer (ABL 30 Acid-base Analyzer, Radiometer, Copenhagen, Denmark) set at 37°C was used to measure pH, PCO_2 and PO_2, and to calculate HCO_3^- and base excess (BE) concentrations. Values were corrected to correspond to the cow's rectal temperature.

A microhematocrit with a capillary tube reader was used to determine PCV; plasma TP concentration was estimated by refractometer. The heparinized blood samples were then centrifuged and plasma was separated. Plasma Na^+ and K^+ concentrations were measured using an automated ion-specific electrode analyzer (NOVA 1 Sodium/Potassium Analyzer, NOVA Biomedical, Waltham, Massachusetts). Chloride concentration was measured using a silver electrode chloride meter (920M Chloride Meter, Corning Medical, Medfield, Massachusetts). Plasma creatinine and total Ca^{++} concentrations were measured using a colorimetric analysis system (Ektachem DT60 Analyzer, Eastman Kodak Co., Rochester, New York). Plasma strong ion difference (SID) was calculated using the equation $[Na^+] + [K^+] - [Cl^-]$; plasma anion gap was calculated using the equation $[Na^+] + [K^+] - [Cl^-] - [HCO_3^-]$.

Aliquots of milk, urine, and abomasal effluent were analyzed for Na^+ and K^+ concentrations using a flame photometer (IL343 Digital Flame Photometer, Instrumentation Laboratory Inc., Lexington, Massachusetts), and Cl^- concentrations were determined with the chloride meter. Estimation of pH was made using pH papers.

STATISTICAL ANALYSES

Beginning at time 0, mean and SEM were calculated for variables at two hour intervals during the diversion period. All data, from both the baseline and diversion periods, are displayed as scatter plots. Differences between prediversion and peak values, as well as between prediversion and end diversion values, were assessed for significance by use of a paired Student's t test ($\alpha = 0.025$), with an experiment-wise significance level of $\alpha = 0.05$. Since a linear trend was seen in the change of PCV, TP, and plasma Cl^- concentration over time, least squares linear regression was computed for these variables.

RESULTS

Data were obtained from four cows in which the experimental trials were completed. Duration of diversion before the end point was reached was 10 to 12 h.

Data for two additional cows are not included for the following reasons. In one cow signs of polyuria, isosthenuria, and elevated serum creatinine concentration led to a diagnosis of chronic, polyuric renal failure. This cow was subsequently euthanized due to chronic weight loss. In the other cow, acute coliform mastitis developed, causing recurrent illness during the study period.

The cannulae were well tolerated by the cows, which showed only minimal irritation due to mild cellulitis and irritation at the site of cannula exit from the body wall. In two cows, the external portion of the cannula barrel became cracked or fragmented from contact with stall partitions. In

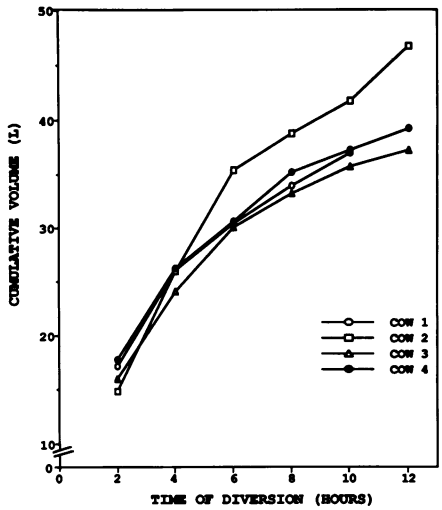

Fig. 1. Cumulative volume of abomasal outflow obtained during diversion trials in four cows.

one of these cows, the broken cannula barrel retracted below the skin, necessitating application of an extension to the barrel.

CLINICAL SIGNS OF DISEASE

All cows had normal vital signs, attitude, feed and water intake, and production of feces, urine, and milk during the prediversion period. Milk production ranged from 18 to 32 kg daily. All cows became completely anorectic within 4 h of the start of diversion. Water intake during diversion ranged from 23.5 to 64.7 L (mean 35.1 ± 9.9 L), with little intake measured after 8 h. As diversion proceeded, the cows became dehydrated as evaluated by skin turgor and degree of enophthalmus. The cows tolerated the cannula well; however, movement of the animals tended to dislodge the diversion apparatus, which necessitated close, continuous monitoring. Depression and marked weakness were apparent within 10–12 h, with three of the four cows becoming recumbent and having difficulty rising. Diversion was stopped at that point, the cannula was returned to its normal configuration to allow for normal duodenal flow of ingesta, and all cows were treated with 20–40 L of 0.9% NaCl and 400–600 mEq of KCl via the jugular catheter.

Rectal temperature was constant (38° to 39°C) throughout the diversion period. Heart rate remained unchanged, with a range of 68 to 80 beats per minute both prediversion and at end diversion. Respiratory rates ranged from 32 to 40 breaths per minute throughout the experiment.

Three of the four cows failed to pass feces during the diversion period. Total urine production for the diversion period was 5.1 to 8.8 L (mean 7.1 ± 0.8 L). Total urinary excretion of electrolytes during diversion is presented in Table I.

Only two cows had measurable milk production during the diversion period. These cows produced 0.4 L and 2.1 L respectively, representing negligible fluid and electrolyte losses.

ABOMASAL OUTFLOW

Abomasal outflow was greatest during the first 2 h of diversion and decreased as the end-point was approached (Fig. 1). Outflow from the cannula occurred in 100 to 150 mL "spurts" every 30 to 40 seconds. As diversion progressed, the time between these "spurts" increased. In addition, the amount of particulate matter in the effluent decreased dramatically with time. For the final 4 h of diversion, the effluent was a homogenous, dark green, viscous fluid. Total abomasal outflow ranged from 37.3 to 46.8 L (mean 40.3 ± 2.2 L).

TABLE I. Total electrolyte content of urine and abomasal effluent

Source	Cl^- (mEq)	Na^+ (mEq)	K^+ (mEq)
Urine	49 ± 11	626 ± 85	653 ± 24
Effluent	4576 ± 309	1858 ± 70	586 ± 32
Total	4625 ± 320	2484 ± 155	1239 ± 57

Values represent total electrolytes collected (MEAN ± SEM) during 10 to 12 h of abomasal outflow diversion in four cows

TABLE II. Laboratory parameters during abomasal outflow diversion

Variable	Prediversion	Peak Level[a]	End-diversion
PCV (%)	30.5 ± 0.9	41.5 ± 2.1[b]	41.5 ± 2.1[b]
TP (g/dL)	9.4 ± 0.3	11.2 ± 0.3[b]	11.2 ± 0.3[b]
Creatinine (mg/dL)	0.98 ± 0.03	1.40 ± 0.15[b]	1.20 ± 0.14
Ca^{++} (mg/dL)	9.4 ± 0.5	6.6 ± 0.6[b]	6.6 ± 0.6[b]
Cl^- (mEq/L)	97.3 ± 0.8	86.3 ± 1.4[b]	87.9 ± 1.6[b]
Na^+ (mEq/L)	142.3 ± 0.8	139.4 ± 1.3	139.7 ± 1.3
K^+ (mEq/L)	3.9 ± 0.1	2.9 ± 0.1[b]	3.0 ± 0.2
SID (mEq/L)	44.9 ± 0.6	54.9 ± 0.7[b]	51.8 ± 0.8[b]
pH	7.35 ± 0.01	7.45 ± 0.01[b]	7.42 ± 0.01[b]
HCO_3^- (mEq/L)	22.2 ± 0.9	31.5 ± 0.29[b]	27.2 ± 1.9
Base Excess (mEq/L)	−2.2 ± 1.0	+6.9 ± 0.4[b]	+3.2 ± 1.6

Values are MEAN ± SEM for four cows prior to diversion of abomasal outflow, at peak levels, and at the end of 10 to 12 h of diversion

[a]Peak levels represent either maximum or minimum values attained during diversion

[b]Indicates significantly different than prediversion values ($p < 0.025$)

The pH of the abomasal effluent ranged from 3 to 4 initially, and then increased to a maximum of 5 to 7 at the termination of the trials. The total electrolyte composition of diverted abomasal effluent is summarized in Table I.

CLINICOPATHOLOGICAL PARAMETERS

Table II summarizes the measured laboratory values at the beginning of diversion, at maximal deviation from baseline levels, and at the end of diversion. Figures 2 and 3 depict the changes in PCV, TP, plasma electrolyte concentrations, and blood gas parameters during diversion for all cows.

The PCV and plasma TP concentration increased in linear fashion throughout the diversion period ($PCV = 24.25 + h$; $r = 0.61$; $p < 0.01$), representing mean increases of 30% and 19.6% respectively. Plasma Cl^- concentration decreased in a linear fashion for the first 6 h of diversion ($p\ [Cl^-] = 106.5 - 1.59\ h$; $r = -0.89$; $p < 0.01$), but after this period more variation appeared among the cows and concentrations began to stabilize until the end of diversion. Plasma Na^+ concentrations were highly variable among cows, with no decrease noted except in cow 2. Plasma K^+ concentrations responded similarly to Cl^-,

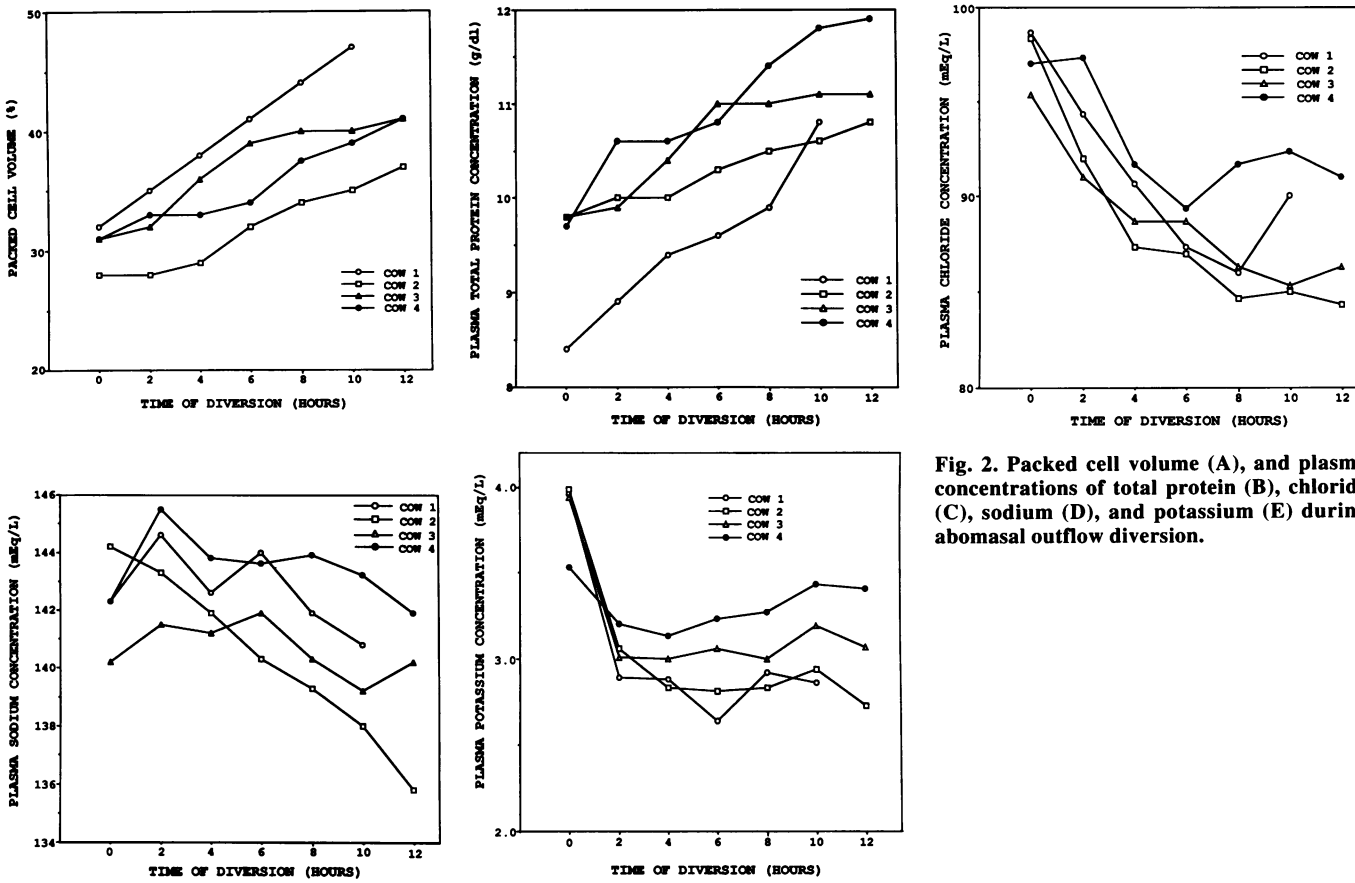

Fig. 2. Packed cell volume (A), and plasma concentrations of total protein (B), chloride (C), sodium (D), and potassium (E) during abomasal outflow diversion.

with values decreasing for the first 4 to 6 h of diversion and then increasing slightly. Although the plasma anion gap showed no significant change, plasma SID demonstrated significant increases ($p < 0.01$) at peak levels and at the end of diversion (normal SID range: 42–48 mEq/L)

Venous pH, plasma HCO_3^- concentration, and BE concentration increased for the first 4 to 8 h of diversion, followed by a gradual decline until the end of the trials. The peak values for each of these parameters were: pH 7.43 to 7.47, (mean 7.45 ± 0.01); HCO_3^- concentration 30.8 to 32.2 mEq/L (mean 31.3 ± 0.3); and BE concentration + 6.1 to + 7.8 mEq/L (mean +6.9 ± 0.4). No significant changes were observed in pCO_2.

Plasma creatinine concentration showed a slight increase, but not significant ($p > 0.05$), at peak samples and at the end of diversion. Plasma calcium concentration showed a substantial decrease during the first 6 h of diversion, and at peak deviation from baseline values and at the end of diversion all cows were significantly hypocalcemic ($p < 0.01$).

DISCUSSION

Many previous models of HHMA in ruminants involved surgical obstruction of the intestine, either by placement of circumferential ligatures around the duodenum, or by intraluminal obstruction achieved via a duodenal cannula (7–11). In 1990, a method for causing HHMA in sheep by diversion of abomasal outflow was described (16). This technique had several advantages, including minimal disruption of the neurovascular supply to the duodenum during surgical placement of the cannula, easy collection of gastric fluid for volume determination and analysis, and the ability to perform repeated studies on the same animals. It must be recognized, however, that the obstruction models may more accurately mimic some of the conditions that cause HHMA (e.g. abomasal volvulus), especially with regards to gastric distention and possible water and electrolyte absorption from sequestered gastric fluids (16).

Changes in clinicopathological parameters during diversion were not uniformly consistent with results of abomasal outflow diversion in sheep (15–17). All cows developed dehydration as indicated by increased PCV and plasma TP concentration, as well as deterioration in physical parameters and clinical appearance. Only a slight increase in plasma creatinine concentration during diversion, with no significant elevation at the end of the experiment, suggests that the degree of dehydration was not severe. These changes were also documented in sheep, however they often did not become severe until diversion had progressed for 72 h or more (16). Dehydration was attributed to fluid loss, including abomasal effluent and urine, and decreased food and water intake.

Clinical depression and weakness in these cows may have been caused, at least in part, by hypocalcemia. This phenomenon has not been found to occur in sheep undergoing abomasal outflow diversion (Fubini SL, Smith DF, Cornell University, Ithaca, New York, unpublished observations, 1988), and its occurrence in these cows is not easily explained. As

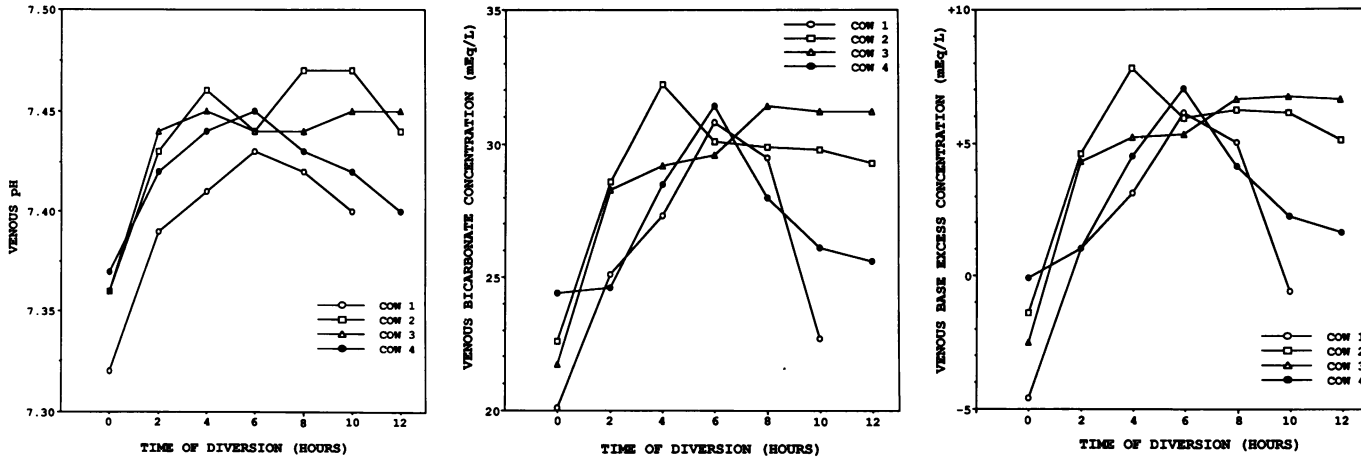

Fig. 3. Venous blood values of pH (A), bicarbonate concentration (B), and base excess concentration (C) during abomasal outflow diversion.

almost no Ca^{++} secretion occurs across the abomasal or forestomach mucosa, most of the calcium presumed to be lost by diversion should have originated from the feed and not the plasma. Cows excrete very little urinary calcium, and even though a mature cow may lose 7 to 8 g of endogenous calcium daily due to insensible intestinal losses (excretion and mucosal cell sloughage) (18), these endogenous losses cannot account for the rapid drop in plasma concentrations. Considering the stage of lactation of these cows, and their low level of milk production, simple cessation of calcium intake (i.e. anorexia) should not by itself cause hypocalcemia. The possibility therefore must be considered that some metabolic or physiological derangement is triggering a shift of calcium from the plasma, or that some other route of substantial loss has been overlooked. Although the exact mechanism is unclear, the incidence of clinical hypocalcemia has been reported to be higher in cows fed a ration with a high cation/anion ratio (19). Since these cows were on such a diet (+65 mEq/kg DM), the changes resulting from this imbalance may have disrupted normal calcium homeostatic mechanisms.

Plasma Cl^- concentrations decreased in a linear fashion for 6 h and then appeared to plateau. This is in contrast to the sheep model where this decrease continued for 60 to 120 h. In addition, no terminal plateau or increase in plasma Cl^- concentration was apparent in sheep (16), and the principal cause of hypochloremia was concluded to be loss of abomasal secretions rich in HCl (1,20), compounded by urinary loss and decreased intake during diversion. Although a balance analysis was not performed in this study, the effluent and urinary losses observed for the measured electrolytes supports these two routes as major contributors to the decreased plasma concentrations. Although a substantial amount of the electrolytes in the effluent were components of the ingested feed, and were not "lost" from the cow *per se*, a certain amount was certainly derived from gastric secretions and saliva, and may therefore have contributed to the decreases in plasma concentrations. Partitioning of these sources (i.e. plasma-derived vs. feed-derived) is not possible without more complete balance studies. However, it does appear that ongoing diversion of these electrolyte rich fluids in cows does not result in the same rapid, dramatic metabolic changes (i.e. plasma electrolyte losses) as can be demonstrated in sheep.

Because the ruminant forestomach compartments have the capacity to absorb Na^+ and Cl^- (21), the reflux and sequestration of abomasal secretions that occurs in an obstruction/ligation model or in naturally-occurring obstructions may make these ions, to some degree, accessible to the plasma by this route. While the extent of forestomach strong ion absorption has not been studied under conditions of hypochloremia, hyponatremia, or metabolic alkalosis, we must regard the influence of these routes of absorption on plasma electrolyte concentrations as important.

The reason for the changes in plasma Cl^- concentration in these cows near the end of the experiment was not readily apparent. It may have resulted from a decreased rate of HCl secretion due to a reduction in the volume of digesta being introduced from the omasum (20). Accompanied by a contraction of the extracellular fluid space, which is the principal distribution site for Cl^-, this decreased secretion may have contributed to an increase in plasma Cl^- concentration despite a total body chloride deficit.

The decrease in plasma K^+ concentration for the first 4 to 6 h of diversion may be attributed to anorexia, urinary loss, intracellular movement of potassium due to developing alkalosis (22,23) and the loss of secreted K^+ in the abomasal effluent. The pattern of change in plasma K^+ concentration after this initial period, when levels appeared stable despite continuing losses, may have been the result of a shift of intracellular potassium into the extracellular fluid with the development of a superimposed metabolic acidosis (22), or leakage from muscle tissues due to cell necrosis and rhabdomyolysis associated with poor peripheral perfusion and recumbency.

Although hyponatremia was not a consistent finding in several ruminant experimental models of HHMA, it has been observed in clinical studies (4) and in the abomasal outflow diversion model in sheep (15–17). A decrease in plasma Na^+ concentration was not observed in this study, with the

exception of cow 2. Large intracellular stores of sodium, ECF volume contraction, and the relatively short duration of diversion all possibly contributed to the failure to induce hyponatremia.

Despite the loss of a large amount of HCl in the abomasal effluent, comprising both dietary acid and abomasal secretions, severe metabolic alkalosis was not induced in the cows. This is in contrast to the rapid development of severe alkalosis observed in obstruction models (1,6,9,11) and in the sheep diversion model (15–17). Although an increasing trend was seen in pH for the first 4 to 6 h of diversion, and plasma BE and HCO_3^- concentrations showed statistically significant elevations during this same time period, these parameters began to plateau as the end-point of diversion was approached. The maximum venous pH of 7.47 and plasma BE concentration of +7.8 mEq/L are well below those values seen in sheep (15,16). A terminal decline was observed in sheep, but only after an extended diversion period. This was attributed to the development of superimposed metabolic acidosis and was associated with an increase in plasma lactate concentration (16). Although plasma lactate was not measured in this study, the calculated plasma anion gap failed to increase, which makes a significant lactic acidosis unlikely. In addition, the significant increase in the calculated plasma SID is consistent with a mild metabolic alkalosis (24).

The failure to cause significant and sustained HHMA in lactating cows by diversion of abomasal outflow thus appeared to be attributable, at least in part, to the rapid onset of anorexia, decreased water intake, dehydration, hypocalcemia, and pronounced depression and weakness at an early stage of diversion. The apparent ileus which developed, as evidenced by the lack of fecal output and the dramatic reduction in particulate matter moving from the ruminoreticulum into the abomasum, may have markedly reduced spontaneous secretion by the abomasum. As a result, since no further excess losses of H^+, Cl^- and H_2O were occurring, the cows failed to develop progressive HHMA. In contrast, the model in sheep consistently caused HHMA and was better tolerated by the animals (15,16). The fact that the cows were lactating did not appear to be a major reason for this difference, because of the small amount of milk produced; however, underlying physiological differences present during lactation may be important. Other factors that must be considered include ration differences and species differences in water and electrolyte homeostatic mechanisms.

REFERENCES

1. **GINGERICH DA, MURDICK PW.** Paradoxic aciduria in bovine metabolic alkalosis. J Am Vet Med Assoc 1975; 166: 227–231.
2. **McGUIRK SM, BUTLER DG.** Metabolic alkalosis with paradoxic aciduria in cattle. J Am Vet Med Assoc 1980; 177: 551–554.
3. **SCHOTMAN AJH.** The acid-base balance in clinically healthy and diseased cattle. Neth J Vet Sci 1971; 4: 5–24.
4. **SMITH DF.** Right-side torsion of the abomasum in dairy cows: classification of severity and evaluation of outcome. J Am Vet Med Assoc 1978; 173: 108–111.
5. **WHITLOCK RH, TASKER JB, TENNANT BC.** Hypochloremic metabolic alkalosis and hypokalemia in cattle with upper gastrointestinal obstruction. Am J Dig Dis 1975; 20: 595–596.
6. **AVERY TB, NAGARAJA TG, FREY RA.** Blood, urine, and ruminal fluid changes associated with metabolic alkalosis induced by duodenal obstruction. Am J Vet Res 1986; 47: 890–896.
7. **CORKER E, DZIUK HE.** Obstructive ligation of the digestive tract in sheep. Am J Vet Res 1968; 29: 1429–1439.
8. **GINGERICH DA, MURDICK PW.** Experimentally induced intestinal obstruction in sheep: paradoxic aciduria in metabolic alkalosis. Am J Vet Res 1975; 36: 663–668.
9. **HAMMOND PB, DZIUK HE, USENICK EA, STEVENS CE.** Experimental intestinal obstruction in calves. J Comp Pathol 1964; 74: 210–222.
10. **PAPADOPOULOS P, RAPTOPOULOS D, DESSIRIS A, TSIMOPOULOS G.** Experimental intestinal obstruction in cattle, Part I: Changes in the clinical picture. Zentralbl Veterinaermed [A] 1985; 32: 265–275.
11. **PAPADOPOULOS P, RAPTOPOULOS D, DESSIRIS A, TSIMOPOULOS G.** Experimental intestinal obstruction in cattle, Part II: Changes in the blood, urine and rumen content chemistry. Zentralbl Veterinaermed [A] 1985; 32: 276–288.
12. **IVAN M.** A new method for re-entrant cannulation of the duodenum in sheep. Can J Anim Sci 1977; 57: 225–227.
13. **IVAN M, JOHNSTON DW.** Reentrant cannulation of the small intestine in sheep: cannula and surgical method. J Anim Sci 1981; 52: 849–856.
14. **ROBINSON PH, SMITH DF, SNIFFEN CJ.** Development of a one-piece reentrant cannula for the proximal duodenum of dairy cows. J Dairy Sci 1985; 68: 986–995.
15. **FUBINI SL, SMITH DF, GROHN YT, LEVINE SA, DEUEL DM.** Replacement of chloride deficit by use of 1.8% NaCl to correct experimentally induced hypochloremic metabolic alkalosis in sheep. Am J Vet Res 1991; 52: 1898–1902.
16. **SMITH DF, LUNN DP, ROBINSON GM, McGUIRK SM, NORDHEIM EV, MacWILLIAMS PS.** Experimental model of hypochloremic metabolic alkalosis caused by diversion of abomasal outflow in sheep. Am J Vet Res 1990; 51: 1715–1722.
17. **LUNN DP, McGUIRK SM, SMITH DF, MacWILLIAMS PS.** Renal net acid and electrolyte excretion in an experimental model of hypochloremic metabolic alkalosis in sheep. Am J Vet Res 1990; 51: 1723–1731.
18. **HANSARD SL, COMAR CL, PLUMLEE MP.** The effects of age upon calcium utilization and maintenance requirements in the bovine. J Anim Sci 1954; 13: 25–36.
19. **BLOCK E.** Manipulating dietary anions and cations for prepartum dairy cows to reduce incidence of milk fever. J Dairy Sci 1984; 67: 2939–2948.
20. **HILL KG.** Continuous gastric secretion in the ruminant. Q J Exp Physiol 1955; 40: 32–39.
21. **GABLE G, MARTENS H.** Transport of Na^+ and Cl^- across the forestomach epithelium: mechanisms and interactions with short chain fatty acids. In: Tsuda T, Sasaki Y, Kawashima R, eds. Physiologic Aspects of Digestion and Metabolism in Ruminants. New York: Academic Press, 1991: 129–147.
22. **ADROGUE HJ, MADIAS NE.** Changes in plasma potassium concentration during acute acid-base disturbances. Am J Med 1981; 71: 456–467.
23. **SVENDSEN P.** Evidence of potassium shift from the extracellular to the intracellular fluid space during metabolic alkalosis in cattle. Nord Vet Med 1969; 21: 660–663.
24. **EIKER SW.** An introduction to strong ion difference. Vet Clin North Am Food Anim Pract 1990; 6: 45–49.