There are few field tests with proven reliability for on-the-farm analysis. This is not to imply that field tests are of no value but reflects a lack of information and data concerning their usefulness. The diphenylamine test for nitrates will detect high concentration of nitrate (2%) in feed but is not sufficiently sensitive for nitrate concentrations (0.5%) that may lead to chronic nitrate intoxication. The reagent is made by adding 0.5 gm of diphenylamine to 20 ml water and concentrated sulfuric acid is added q.s. to 100 ml. This is a stock solution which is mixed with equal parts of 80% sulfuric acid for use. One drop of the reagent is placed on the cut surface of the plant and a color change from green to blue is indicative of high nitrate content (approximately 2%). Corn suspected of containing aflatoxin may be examined with an ultraviolet light. If aflatoxin is present the split corn kernel will have an area of blue fluorescence around the endosperm layer.

Laboratory procedures that may serve adequately for clinical diagnosis may not necessarily be adequate for evidence in a lawsuit. This should be recognized prior to rendering any opinion to the client concerning a basis for suing for damages or recovery of losses.

The future direction of clinical toxicology is dependent on greater specialization of veterinary toxicologists and subsidized support of university and governmental analytical laboratories to serve in a consultative capacity with the private veterinary practitioner. It is impossible to speculate on the impact of chronic intoxication on animal health because of our limited ability for diagnosis. Since we instituted a modest program of lead analysis in our laboratory there has been a significant increase in the number of diagnoses of chronic lead intoxication in both pet animals and cattle. It is unlikely, however, that laboratory toxicologic diagnosis will ever become as routine as the complete blood count.

Practical Fluid and Electrolyte Therapy and its Pathophysiological Basis

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Introduction

All of us who are involved in bovine medicine and therapeutics are aware of the need for fluid therapy in many disease conditions. Intimately associated with dehydration, toxemia, gastro-intestinal disorders, and the need for fluid therapy are derangements of electrolytes and acid-base balance.

Let me first acknowledge that a whole battery of laboratory tests is impractical and probably impossible in practice. The objective of this presentation is to discuss the pathophysiology of acid-base and electrolyte disorders as they occur in bovine disease. With a basic understanding of pathophysiology, fluid therapy can be approached in a rational manner even without the benefit of laboratory tests. If those of us who can use laboratory determinations in cattle succeed in communicating our findings to those who don't, we will have come one stop closer toward making the contribution to veterinary medicine that I feel we owe.

The bovine species is somewhat unique in that both acidosis and alkalosis are common. Acidosis is a common condition in disease in all species. Alkalosis, however, is rather rare in most species but very common in cattle. This is not to imply that acidosis and alkalosis *per se* are diseases of cattle which need to be treated. Rather, acid-base and electrolyte disorders are manifestations of disease which must be taken into account in designing a regimen of supportive therapy.

Many excellent articles have been written on the subject of practical fluid therapy. Included in many of these papers are various formulations of fluids and electrolytes for use in supportive therapy. Along such lines I really have nothing new to add. After all, the standard toward which each of these formulas strives is a standard upon which none of us can improve: that is plasma itself.

Instead, I would like to suggest that, as a starting point, two different kinds of corrective fluids are required: one for supportive therapy in cases of acidosis and the other for supportive therapy in alkalosis. In acidosis, Eltraad L. A.,* or a formula of similar com-

Haver-Lockhart, Shawnee, Kansas

position, should be used for intravenous therapy. In the alkalotic patient, Ringer's solution or a formula of similar composition should be used. Oral fluids, designed for supportive therapy of the acidotic patient and the alkalotic patient can also be used. Fluid therapy in calf scours cases is somewhat more involved. A bicarbonate-containing solution is required for intravenous therapy. Oral therapy in calf scours cases may also be effective if an electrolyte solution which contains glucose is used.

Acid-Base Physiology

Acid-Base Balance

The precise regulation of the pH or hydrogen ion concentration of the body fluids is one of the most crucial physiological functions of the body. The precision of the acid-base regulatory mechanisms of the body is well illustrated by looking at normal values (13).

pH 7.35 to 7.45

$[H^+]$ 44.7 X 10^{-6} mEq/L to 35.5 X 10^{-6} mEq/L

The body has various acid-base regulatory mechanisms to defend against changes in body fluid pH. The precise description of these delicately balanced mechanisms is not our purpose in this discussion. We can summarize, however, by simply stating that the pH of the body fluids is proportional to the concentration of bicarbonate and the partial pressure of CO_2.

$$pH \propto \frac{[HCO_3^-]}{PCO_2}$$

"metabolic" / "respiratory"

We can further simplify by saying that primary alterations of the bicarbonate component of the expression may be termed metabolic, and that primary alterations of the PCO_2 component of the expression may be termed respiratory.

For purposes of this discussion we shall deal only with metabolic disturbances of acid-base balance. From the expression, we can see that any decrease in the bicarbonate concentration would tend to cause a decrease in pH, hence metabolic acidosis. Conversely, any increase in the bicarbonate concentration would tend to cause an increase in pH, hence metabolic alkalosis.

Metabolic acidosis is defined as a decrease in blood pH due to a relative decrease in the blood bicarbonate concentration. Metabolic alkalosis is an increase in blood pH due to a relative increase in bicarbonate concentration.

Metabolic Acidosis

Metabolic acidosis is a serious acid-base disturbance which is encountered commonly in bovine disease. There are many causes of acidosis in disease. Physiologically speaking, however, there are only two ways that metabolic acidosis can occur: either due to a gain of acid by the body or due to a loss of base (Table 1).

Gain of Acid: There are a number of ways that a gain of acid can occur in bovine diseases and lead to serious metabolic acidosis. First and most obvious is the absorption of acid from the rumen. Perhaps the best disease example of this is in grain overload lactic acidosis in which lactic acid is absorbed in large quantities from the rumen (4).

Table 1

Acid-Base Disorders

Disturbance	Physiological Meaning	Examples	Treatment
Metabolic Acidosis	Gain of acid (not H_2CO_3)	Grain overload Ketoacidosis Tissue anoxia Tissue catabolism	Eltraad L.A. Acidosis formula "5:4:1" solution Asiatic formula
	Loss of base	Diarrhea Loss of saliva	
Metabolic Alkalosis	Gain of base	Iatrogenic	Ringer[s sol'n Isotonic saline
	Loss of acid (not H_2CO_3	Abomasal displacement Intestinal obstruction GI stasis	Alkalosis formula

Secondly, the incomplete oxidation of fat in the bovine species causes excess production of acetate, β-hydroxy butyrate, and acetoacetate. The accumulation of these ketoacids results in acetonemia with which all of us are familiar. I have not had occasion to measure severe acidosis due primarily to ketosis, but ketoacids most assuredly contribute to acidosis in many cases.

A third cause of acid gain in disease is the incomplete oxidation of carbohydrates. This occurs commonly during dehydration from any cause, due to diminished tissue perfusion and oxygenation. The products of anaerobic metabolism are organic acids such as lactate which may accumulate and lead to acidosis.

Still another reason for acid gain is the accumulation of tissue breakdown products such as phosphates and sulfates. Dehydration and starvation can lead to increased tissue catabolism. When renal function is impaired at the same time, these inorganic acids may accumulate in the body fluids.

Finally, diminished kidney function can contribute to acidosis due to all of the above factors. The normal kidney is capable of excreting a large acid load. Dehydration and hypovolemia imply decreased renal blood flow and impaired renal function. Thus, we see that dehydration, which is often the primary reason for initiating fluid therapy in the first place not only causes increased organic and inorganic acid production in the body, but also causes diminished ability of the kidney to excrete the acid load.

Loss of Base: Metabolic acidosis due to the loss of base from the body frequently occurs in bovine disease. The most obvious example of acidosis due to the loss of base is in diarrhea, in which large quantities of bicarbonate are lost in the feces. The pathophysiology of neonatal diarrhea has been well described before this convention and in numerous excellent veterinary

articles. I would like to point out that severe diarrhea does result in a loss of base but also leads to dehydration which in turn causes a gain of acid due to the accumulation of acid metabolites.

Another example of disease which causes a loss of base and acidosis is any condition in which there is excessive loss of saliva. This may be seen with retropharyngeal abscesses, for example, when the animal drools saliva due to inability to swallow.

Distinguishing Between Gain of Acid and Loss of Base: Analysis of serum electrolytes can be used to distinguish between the gain of acid and the loss of base as a cause of acidosis. In the normal animal, the sum of the major anions, bicarbonate and chloride, plus 12 mEq/L of unmeasured anions is equal to the major cations, sodium.

$$[HCO_3^-] + [Cl^-] + 12 = [Na^+]$$

Stated another way, the sodium concentration minus the bicarbonate and chloride concentrations equals the unmeasured anion concentration. The normal concentration of unmeasured anions in the body is 12 mEq/L with a range of 5-15 mEq/L in man (3).

$$[Na^+] - ([HCO_3^-] + [Cl^-]) = 12 \text{ (Normal range 5-15)}$$

What is included in the unmeasured anion fraction of the blood? It is comprised chiefly of acid metabolites: lactates, phosphates, sulfates, ketone bodies, etc. An increase in the concentration of unmeasured anions is simply an indirect measurement of the increased acid products we've been talking about (Table 2). Thus, we see that when acidosis occurs and the concentration of unmeasured anions is increased, that is evidence for acidosis due to the gain of acid. When acidosis occurs and the amount of unmeasured anion remains within the normal range, the acidosis is due to a loss of base.

Table 2

Unmeasured Anions

1. Incomplete oxidation of fats:	Ketone bodies
2. Incomplete oxidation of carbohydrates:	Lactate, oxalate, etc.
3. Tissue breakdown products:	Phosphates, sulfates, etc.

Metabolic Alkalosis

Metabolic alkalosis is an acid-base disturbance which occurs in bovine disease with surprising frequency. Cows with a wide variety of disorders of the upper portion of the gastrointestinal tract often develop severe metabolic alkalosis and characteristic electrolyte changes. Following the same line of reasoning that we pursued for acidosis, we can say that alkalosis can occur either due to a gain of base or due to a loss of acid (Table 1).

Gain of Base: Experimentally metabolic alkalosis can be induced by giving sodium bicarbonate orally or intravenously. Other than overzealous administration of base, there are few examples of alkalosis in disease due to a gain of base. One example may be urea toxicity (if you call that natural disease) in which the pH of the rumen contents rises drastically and may cause alkalosis due to the gain of base. In urea poisoning, however, the absorption of ammonia is probably responsible for the clinical signs observed.

Loss of Acid: For all practical purposes, metabolic alkalosis occurs in cattle as a result of a loss of acid into the gastrointestinal system. This is very common in cattle as a result of sequestration of large volumes of acidic abomasal juice in the abomasum and forestomachs (5).

The process by which hydrochloric acid is secreted into the abomasum involves the removal of a chloride ion from the circulation and the return of a bicarbonate ion to the circulation. The overall process is represented by the following reaction: (11)

$$NaCl + H_2CO_3 \longrightarrow \underset{\text{(secreted)}}{HCl} + \underset{\text{(Plasma)}}{NaHCO_3}$$

Any condition in which the secreted HCl is delayed in moving out of the abomasum and into the intestines for reabsorption can lead to severe metabolic alkalosis due to bicarbonate accumulation and hydrochloric acid loss. Hypochloremia of the same magnitude as the increased bicarbonate concentration also occurs during metabolic alkalosis. A schematic representation of the reaction as it occurs in the abomasum is shown (Figure 1).

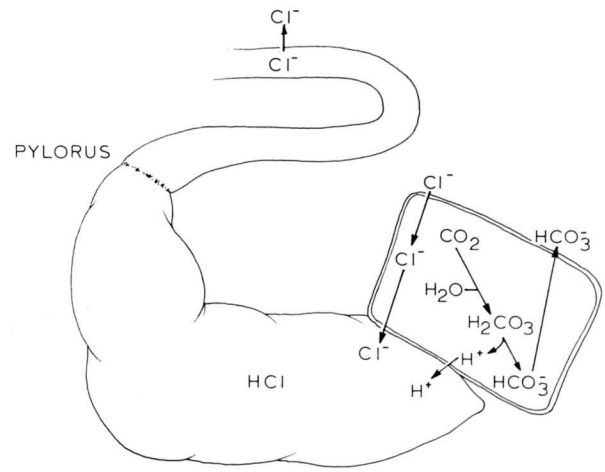


Figure 1: Schematic diagram of abomasum depicting a secreting mucosal cell.

Diseases in which hypochloremic metabolic alkalosis occurs include functional or anatomical intestinal obstruction, abomasal disorders, and also diseases characterized by gastrointestinal stasis. Specific diseases in which we have observed the pattern include intestinal intussusception, abomasal displacement or torsion, abomasal impaction, and indigestion from a variety of causes such as vagus indigestion.

Summary

In summary fluid imbalances in cattle are often characterized by either acidosis or alkalosis.

Acidosis is caused by either a gain of acid or by a loss of base. A gain of acid is seen in 1) absorption of acid from the rumen as in grain overload; 2) ketoacidosis, 3) organic acidosis due to anaerobic metabolism, 4) inorganic acidosis due to tissue breakdown products, and 5) impaired kidney function which aggravated the above conditions. Acidosis due to a loss of base is seen during diarrhea and in conditions in which excessive loss of saliva occurs.

Alkalosis in naturally occuring bovine disease is caused by the loss of acid. Abomasal HCl is lost by being trapped in the upper gastrointestinal tract.

Case Examples

With these physiological concepts in mind, we can now look at the laboratory findings from several examples of bovine disease conditions and discuss fluid therapy.

Acidosis

Loss of Base: Pharyngitis. The first case shows some laboratory data from a feedlot steer with retropharyngeal abscesses (Figure 2). The lesions prevented normal swallowing and caused the animal

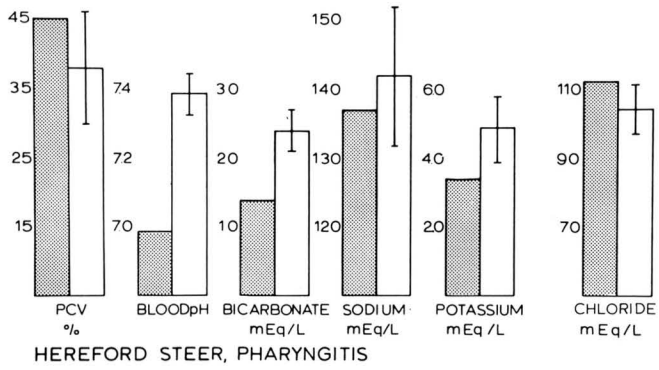


Figure 2: Laboratory data (shaded bars) compared to normal range (unshaded bars).

to drool copious amounts of saliva. Severe dehydration was apparent clinically and is reflected in the elevated packed cell volume. The blood pH is below seven which indicates severe acidosis. The bicarbonate concentration is low, about 14 mEq/L so we can conclude that the acidosis was a metabolic acidosis. (The acidosis in this case also has a respiratory component which is not shown here.) The sodium concentration is within the normal range. The chloride concentration is only slightly elevated. We, therefore, conclude that the dehydration is essentially an isotonic dehydration. The potassium concentration is below normal, which may have partially accounted for muscular weakness observed clinically in the animal.

Next, we can use our quick and easy undetermined anion scheme to see if the laboratory data confirms our clinical impression that the acidosis is due to the loss of base because of a loss of saliva. The sodium concentration is 137 mEq/L. The chloride concentration, 112 mEq/L, and the bicarbonate concentration, 14 mEq/L, total about 126 mEq/L. Subtracting 126 mEq/L of major anions (Cl^- and HCO_3^-) from 137 mEq/L of major cations (Na^+) leaves about 11 mEq/L of undetermined anions. Eleven is within the normal range of 5-15. Thus, the laboratory data supports the clinical suggestion of acidosis due to the loss of base.

The features of this case are dehyration, metabolic acidosis due to a loss of base, and mild hypokalemia. Because the dehydration is isotonic, balanced electrolyte solutions should be used for rehydration. Because of the bicarbonate deficit, the solution chosen should be high in bicarbonate or a bicarbonate precursor. Because of the hypokalemia, extra potassium in the solution is also desirable. Looking at the chart, we see that Eltraad L. A. adequately meets those requirements (Table 3). It is an isotonic, balanced electrolyte solution with extra potassium and 55 mEq/L of bicarbonate equivalents, in the form of acetate. As a starting point, Eltraad L. A. or a similar formulation would be an appropriate choice of intravenous fluids.

Table 3

Supportive Fluids (in mEq/L)

	Na^+	K^+	Ca^{++}	Mg^{++}	Cl^-	HCO_3^-	Glucose	Tot.
Plasma	142	5	5	3	105	25		310
Intravenous Fluids:								
Eltraad L. A.	140	10	5	3	103	55*		316
"5:4:1" sol'n	133	13			98	48		292
Ringer's sol'n	147	4	6		157			314
Phys. Saline	154				154			308
Oral Fluids:								
Acidosis Form.	145	19			104	60		328
Alkalosis Form	127	19			146			292
Asiatic Form.	100	10			70	40	120	340

*After Metabolic Conversion

Formulations

Intravenous Fluids		Oral Fluids	
Eltraad L.A. Concentrate (Haver-Lockhart)			100 gm NaCl
		Acidosis Form:	100 gm $NaHCO_3$
	5 gm NaCl		qs 5 gal.
"5:4:1"	4 gm $NaHCO_3$ qs 1 liter		30 gm Kcl
	1 gm KCl	Alkalosis Form:	150 gm NaCl
			qs 5 gal
	35 gm NaCl		30 gm KCl
Ringer's	1.2 gm KCl qs 1 gal.		
	1.3 gm $CaCl_2$		13.9 gm NaCl
		Asiatic Form:	14.4 gm $NaHCO_3$
			qs 1 gal.
			3.0 gm KCl
Saline	35 gm NaCl qs 1 gal.		86.4 gm Glucose

The next question concerns other aspects of supportive therapy such as energy requirements. The addition of dextrose to the solution provides some energy and also enhances the movement of potassium into cells. This is not to imply that the total energy requirement can be met with dextrose. How much

dextrose should be added? Glucose is transferred out of the circulation at an average rate of 60 gm/hr in normal lactating cows (6). The maximum rate of glucose utilization may be somewhat higher than this, but it is unlikely that the administration of more than 100 grams per hour would be of much benefit during intravenous therapy. This means that the addition of 200 cc of 50% dextrose to an "hour's worth" of IV fluids should be a suitable dose. Rapid administration of dextrose will result in diuresis which is, of course, undesirable during rehydration.

Amino acids can also be used to improve the nitrogen balance and enhance protein synthesis in debilitated patients. Unfortunately, the common amino acid-dextrose preparations promoted for use in cattle and horses contain insignificant amounts of amino acids. A 5% amino acid solution, Amigen**, is available as a much richer source of amino acids. The cost of 500 cc of Amigen is around $3 as compared to $.75 for veterinary amino acid-dextrose-vitamin B preparations. Amigen, however, contains about 200 times as much amino acid. As far as dosage is concerned, if extrapolation of human dosage recommendations is valid, then 500 cc of 5% amino acids given over an hour or two with plenty of dextrose should be suitable in cattle.

How much intravenous fluid should be given? This depends upon the condition of the animal and the situation under which fluids are being administered. Techniques for the administration of large quantities of fluids intravenously to the unattended animal have been described. We use PE 240 polyethylene catheters in the jugular vein and a long simplex tube connected to a five gallon plastic water bag suspended over the stall. Think in terms of gallons, not liters. Keep in mind that large quantities of IV fluids will save animals that are in real trouble and buy some time until surgery can be performed or some toxic infection can be controlled.

Once the initial rehydration is well underway and the animal is making some improvement, oral fluids can often be used effectively to supplement the more costly and more bothersome intravenous therapy. A formula which we use orally for the acidotic cow is shown (Table 2). There is nothing special about the formula. It is simply an isotonic solution with balanced electrolytes, extra potassium, and bicarbonate. We give five gallons or more of this solution at a time.

Gain of Acid: Toxemia. The next case gives the laboratory data from a Guernsey cow which was extremely weak and dehydrated, a rather typical toxemic cow with mastitis and metritis (Figure 3).

The packed cell volume is within the normal range. Since the cow was severely dehydrated, the normal packed cell volume immediately suggests anemia. The blood pH is about 7.3, which indicates a degree of acidosis. The bicarbonate concentration is 16 mEq/L, which indicates that the acidosis is metabolic in nature. Her sodium and chloride concentrations are

**Baxter Laboratories, Morton Grove, Illinois.*

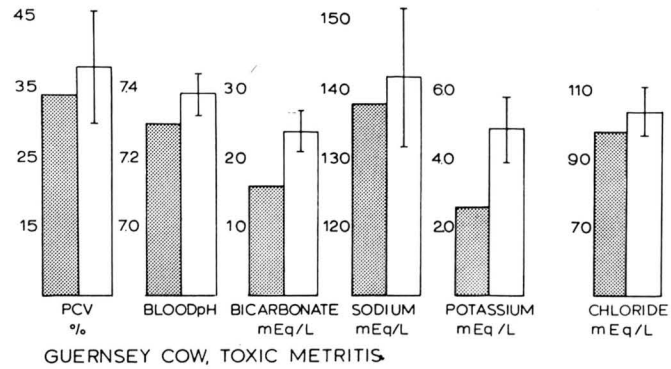


Figure 3: Laboratory data (shaded bars) compared to normal range (unshaded bars).

practically normal. Her potassium concentration is quite low. Subtracting the chloride concentration, 97 mEq/L and the bicarbonate, 16, from the sodium concentration, 138, we find that she has 25 mEq/L of unmeasured anions in the system. Twenty five is definitely above the normal range of unmeasured anions. This may be taken as evidence for acidosis due to a gain of acid metabolites in the blood.

The clinical and laboratory features of this cow which represents a case of toxemia are dehydration, metabolic acidosis due to the gain of acid, and hypokalemia. Looking again at the chart, we see that Eltraad L. A. would be an appropriate starting point for intravenous therapy (Table 3). Glucose should be added to the fluids as well. We use 100 to 200 cc of 50% dextrose per gallon of IV fluids for reasons explained earlier. We also use the 5% amino acid solution. After rehydration is well underway, we divide 500 cc of Amigen between two gallons or more of intravenous fluids.

Loss of Base Plus Gain of Acid: Calf Scours. The next case shows the laboratory data from a number of field cases of calf scours which were studied, Tennant et al., 1972 (Figure 4). Shaded bars show the mean of the measured values ± 1 standard deviation, compared to the normal range of values (12).

The packed cell volume is markedly elevated with a mean of about 45%. This reflects severe dehydration which is also apparent clinically in calf scours cases.

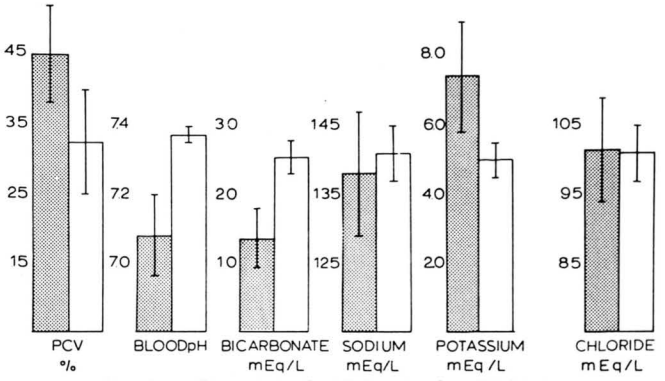
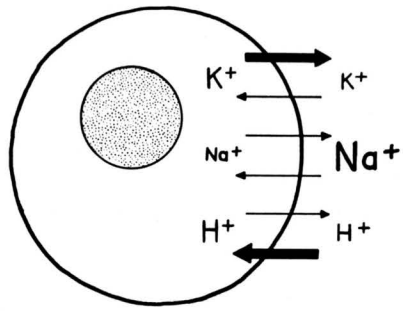


Figure 4: Laboratory data (shaded bars ± *1 standard deviation) compared to normal range (unshaded bars).*

The blood pH is much lower than normal. The mean bicarbonate concentration is about 14 mEq/L, well below the normal range. The conclusion is made that the calves are in metabolic acidosis. Both the serum sodium and the serum chloride concentrations showed quite large variations in this study but their mean concentrations were close to normal. Finally, we see that the serum potassium concentration is markedly elevated, a common observation in bovine neonatal diarrhea.

The pathophysiology of hyperkalemia in neonatal calf diarrhea has been described. One of the regulatory mechanisms which the body employs to guard against the changes in hydrogen ion concentration in extracellular fluid is the exchange of extracellular hydrogen ions for intracellular sodium and potassium ions (10). Potassium, the major intracellular cation, moves out of the cells as hydrogen ions move in (Figure 5) (7). At the same time, renal blood flow is also compromised so that the kidney is unable to excrete potassium at the normal rate. Hyperkalemia is the result of movement of potassium from the cells to the extracellular fluids and decreased renal tubular excretion of potassium.



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Proc. A. A. B. P. 1971

Figure 5: Schematic diagram depicting the potassium movement out of the cell during acidosis.

Was the acidosis in these cases caused by a loss of base or by a gain of acid? A loss of base must certainly have occurred during the initial diarrheal phase. Using the undetermined anion scheme, we subtract the mean chloride concentration, 100, and the bicarbonate, 14, from the sodium, 138, and see that there are 24 mEq/L of undetermined anions. The undetermined anion fraction is well above normal, allowing the conclusion that the acidosis in these calves is also caused by a gain of acid. The gain of acid in calves with scours is expected due to severe dehydration, prerenal azotemia, peripheral anaerobic metabolism, and tissue catabolism.

The fluid, electrolyte, and acid-base picture in typical cases of calf scours is dehydration, metabolic acidoisis, and hyperkalemia. The solution chosen for intravenous therapy in these cases should, therefore, be designed to rehydrate the animal, correct the bicarbonate deficit, and enhance the movement of potassium back into the cells. Looking at the chart, it would appear that Eltraad L. A. comes very close to meeting those requirements (Table 3). During severe dehydration typical of calf scours, however, lactic acidosis is present due to anaerobic metabolism. The addition of more organic acid, acetate in the case of Eltraad L. A., may actually worsen the acidosis.

Because of these points, Eltraad L. A., lactated Ringer's, and other similar solutions are inadequate for the initial stages of intravenous fluid therapy. A balanced electrolyte solution containing bicarbonate itself as well as dextrose is required. A quick and easy formula which we have used with success is the "5:4:1" solution, prepared by simply adding five grams of sodium chloride, four grams of sodium bicarbonate, and one gram of potassium chloride to one liter of water (Table 3). To this we add 40 cc of 50% dextrose. This provides a balanced electrolyte solution, high in bicarbonate, which contains dextrose to enhance the transport of potassium into the cell.

After the first liter or two are given rapidly, intravenous therapy may be continued with Eltraad L. A. spiked with dextrose and amino acids. The question may be raised, "Why give Eltraad L. A. at all, why not continue with the 5:4:1 solution?" The reason is that without careful monitoring of the acid-base status of the calf, treatment with bicarbonate is subject to rather dangerous errors. Once the peripheral blood volume has been partially restored, the body can make its own bicarbonate by oxidizing organic acids such as lactate or acetate. One can not help but be impressed by the efficiency with which the physiological processes restore these calves to normal balance.

Oral Therapy. Perhaps an even more exciting concept in fluid therapy for calf scours is oral therapy. There can be little doubt that enterotoxins from pathogenic *E. coli* often play a significant role in calf scours. These enterotoxins cause hypersecretion of fluid by the intestinal mucosa of the small intestine.

Vibrionic cholera is an enterotoxic diarrheal disease of man which bears a striking resemblance to colibacillosis in pigs and calves. The composition of the stool of cholera victims is shown below.

Na^+	140 mEq/L
K^+	10
Cl^-	110
HCO_3^-	40

This resembles the composition of feces from diarrheic calves. Intravenous administration of fluid of similar composition in man restores and maintains the extracellular fluid volume. The same fluid given orally, however, is not absorbed but rather is added to the stool. If 120 mM/L of glucose is given with electrolytes orally, all of the fluid drunk is absorbed (2). This finding makes practical application of the fact that glucose in the lumen of the intestine enhances the absorption of sodium, even during enterotoxic diarrhea.

The solution which is now widely used for oral treatment of human cholera patients is shown (Table 3, labeled Asiatic solution) (1). We have been using this solution in calf scours cases for nearly a year and have been impressed with the results. If the calf is taken off milk and put on the Asiatic solution before he becomes comatose, intravenous therapy is often unnecessary. After initial rehydration is accomplished intravenously in comatose calves, fluid balance can usually be maintained with the Asiatic solution given orally.

From a practical point of view, packets of ingredients can be weighed out in advance and simply dissolved in a gallon of water or dispensed with the herdsman. Many calves will readily drink the solution from a nursing bottle. Others have to be tubed and given two quarts or more twice or three times a day.

Alkalosis: Loss of Acid

We turn now to some cases of metabolic alkalosis which we will discuss only briefly. As we said, alkalosis in ruminants is caused by the loss of hydrochloric acid into the abomasum and forestomachs. This occurs in abomasal disorders, intestinal obstructions, and quite often in cases of gastrointestinal stasis.

Abomasal Disorder: Impaction. The first case gives the laboratory data from a case of abomasal impaction in an Angus bull (Figure 6). This animal was dehydrated, as reflected in the packed cell volume of nearly 50%. His blood pH is over 7.6 while the bicarbonate concentration is over 55 mEq/L. By definition,

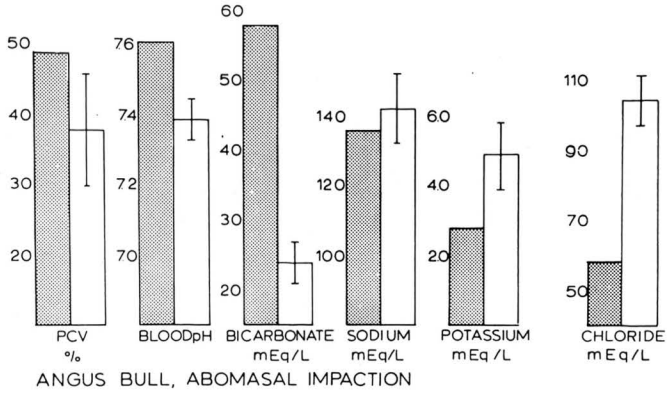


Figure 6: Laboratory data (shaded bars) compared to normal range (unshaded bars).

this animal is in severe metabolic alkalosis. The sodium concentration is normal and the potassium concentration is quite low. The chloride concentration is extremely low, less than 60 mEq/L. It is apparent that the lowered chloride concentration is of about the same magnitude as the increased bicarbonate concentration.

An explanation for the hypokalemia in alkalosis is essentially the opposite as the explanation for hyperkalemia in acidosis. Hydrogen ion diffuses out of the cell to relieve the extracellular, alkalosis, and potassium diffuses into the cell. Increased intracellular potassium has been measured in cows during experimentally-induced metabolic alkalosis (9).

This case shows the very common picture of hypochloremic, hypokalemic metabolic alkalosis. The requirements for intravenous fluid therapy in such cases are balanced electrolyte solutions which are rich in chloride and contain some potassium. In my opinion, there is no need for giving strong acidifying agents such as ammonium chloride. Correcting the chloride deficit alone will correct the alkalosis without the danger of overshoot into acidosis.

Looking at the chart we see that Ringer's solution (not lactated Ringer's) would be the fluid of choice for the supportive treatment of the alkalotic patient (Table 3). Ringer's solution is a balanced electrolyte solution which is rich in chloride and contains physiologic concentrations of potassium. Isotonic saline can also be used because of its high concentration of chloride. Saline, however, does not contain other electrolytes which may be indicated as well.

As a starting point, Ringer's solution should be given intravenously to the alkalotic patient in need of IV fluids supportively. Glucose, amino acids, vitamins, etc., can be added to Ringer's solution as indicated.

Intestinal Obstruction: Intussusception. The next case shows similar laboratory data from a Jersey bull with a small intestinal intussusception (Figure 7).

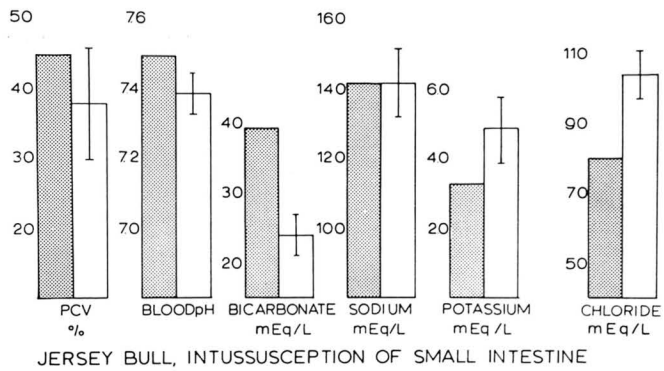


Figure 7: Laboratory data (shaded bars) compared to normal range (unshaded bars).

This data shows essentially the same picture as the previous case: dehydration and hypochloremic, hypokalemic, metabolic alkalosis. Once again, Ringer's solution is clearly the fluid of choice for intravenous supportive therapy in cases of hypochloremic alkalosis (Table 3).

G. I. Stasis: Hypocalcemia. Finally the data from a Guernsey cow with rumen atony secondary to hypocalcemia shows the same picture as the previous two cases (Figure 8). This case was chosen to illustrate metabolic alkalosis in the absence of obstruction or abomasal disorder, simply due to gastrointestinal stasis.

Large quantities of oral fluids can also be used to aid in the supportive therapy of the alkalotic patient. Oral fluids are of little if any benefit prior to relieving an obstruction or prior to the restoration of gastrointestinal motility. The formula listed has been used

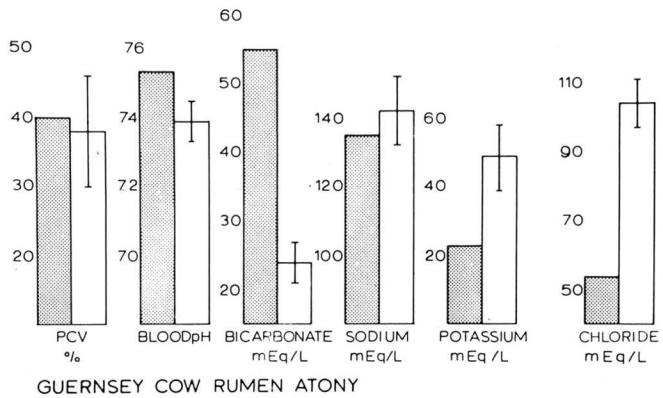


Figure 8: Laboratory data (shaded bars) compared to normal range (unshaded bars).

in hundreds of cases such as post operative abomasal displacement cases (Table 3). There is nothing magic about the formula. It is simply a salt solution which is isotonic and contains higher proportions of chloride and potassium.

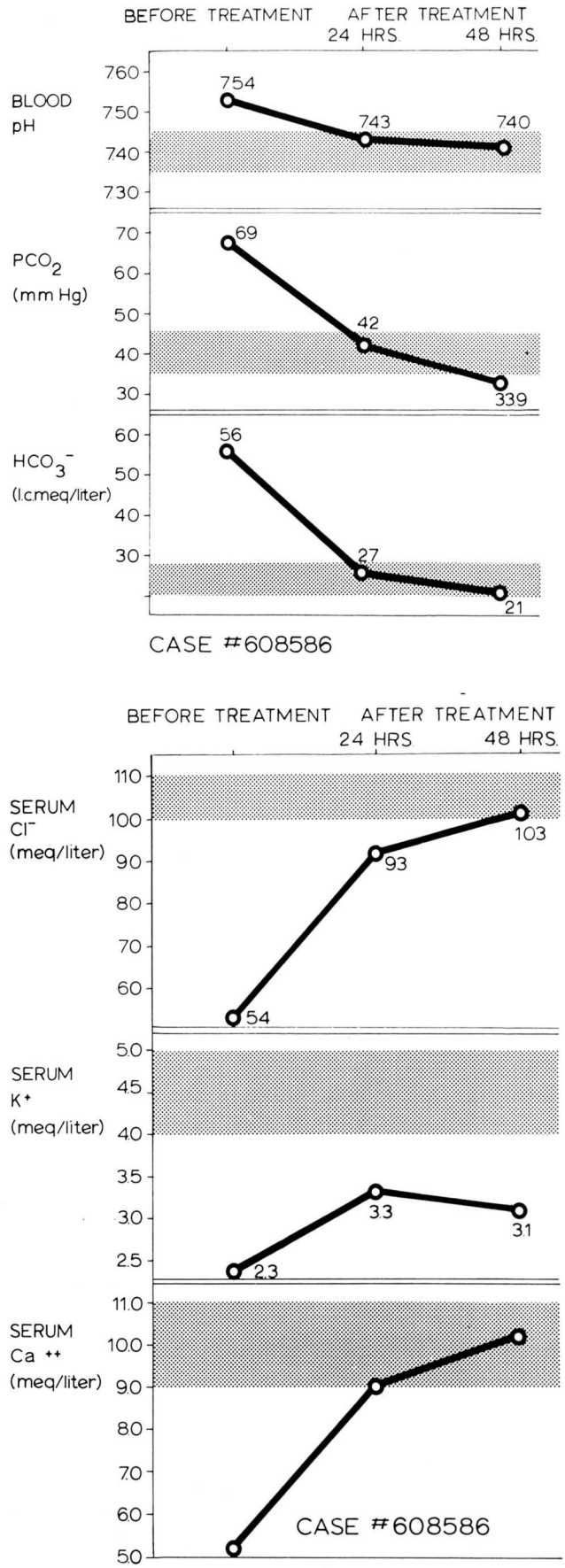
All of the cases presented in this series were treated supportively as discussed and also specifically according to the diagnosis. Most, not all, responded to the treatment. An example of the laboratory changes during and after treatment is shown (Figures 9 and 10). The data are from the hypocalcemic cow.

Conclusion

Throughout this discussion, we have not talked about correcting acid-base disturbances *per se*. Rather I am simply suggesting that by paying attention to the acid-base and electrolyte status of the animal, it is possible to make a more appropriate selection of supportive fluids.

The acid-base and electrolyte status of the animal can often be estimated by physical examination if a bit of physiological reasoning is employed. Is the animal gaining acid, losing base, or losing acid? Is the cow losing base because of a shingle-splitting diarrhea? Is she gaining acid due to severe dehydration and toxemia? If either of these are occurring, the animal is probably in acidosis. Supportive therapy should therefore include fluids designed for the acidotic patient, Eltraad L. A. intravenously or bicarbonate-containing oral fluids. Is the cow weak and dehydrated due to an abomasal torsion or impaction? If so, chances are she is in hypochloremic metabolic alkalosis and supportive therapy should include Ringer's solution intravenously or an appropriate elecrolyte solution orally. Is it a case of calf scours? It is almost certain that the comatose calf will be acidotic. Therapy should include a bicarbonate-containing intravenous solution such as the 5:4:1 solution. The glucose-electrolyte solution (Asiatic solution) should also receive special consideration in calf scours cases.

The question often comes up, what should you use if you just can't tell about the acid-base and electrolyte status from physical examination alone. If you can't tell whether she's acidotic or alkalotic by



Figures 9 & 10: Laboratory data from Guernsey cow with hypocalcemia before and after treatment with calcium and supportive fluids.

physiological reasoning, it probably doesn't matter what fluid you use. All of the solutions discussed here are sufficiently balanced to be safe to use in almost any condition (though not necessarily the most rational). At The Ohio State University Veterinary Hospital, we find more cattle in alkalosis than in acidosis. Other workers have also observed hypochloremic alkalosis in a wide variety of disease conditions (8). I would, therefore, recommend Ringer's solution for supportive therapy in cases where there is some doubt as to the acid-base status.

All of the solutions I have discussed are easily obtainable. At our hospital, we weigh out packets of electrolytes which we can use to make all sorts of solutions. The Asiatic formula for calf scours can be prepared to be dispensed to clients with calf scours problems.

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Panel Discussion

Harold Amstutz, *D.V.M., Chairman*
W. Lafayette, Indiana

Question: What are the responsibilities of the veterinarian when he uses a drug not approved for food producing animals? What are his responsibilities when he makes up his own solutions?

Answer: The veterinarian who uses a product that is not approved for food animals assumes full responsibility for causing drug residues that might go on to the consumer. I guess the real problem here is that he is going to get away without getting caught. I think what bothers me as a practitioner is if a consumer, rightly or wrongly, did pin that back to me that I gave this animal chloramphenicol and this person got aplastic anemia and died. You can obviously see the impact of that kind of situation. I think the answer to this question is yes, the veterinarian who does this has that responsibility. What can he do if he has to do it in an emergency case when nothing else is going to work? The point I would make here is that he is going to have to become more familiar with the metabolic kinetics of the drugs that he chooses. I think if you don't do that and you give a drug that has not been approved, and you have no earthly idea about the withdrawal time or distribution in tissue, then you better not use it. If you have an idea or you can find some metabolism data somewhere, you've kept yourself up on this, and you know that the product is fairly rapidly metabolized, then I think you could probably build in a safety factor. Suppose in five days the drug is safely metabolized in some species of animal, then you caution the farmer and say that this animal must not go to slaughter for 30 days, or 40 days, etc. Some way, build in a safety factor to protect you. Again, I don't think the FDA can sanction this, obviously we don't. I think we realize the facts of life. You're going to use it. All I'm saying is that I would certainly know something about the metabolism kinetics of that drug before I did it. Be sure that the man whose animal you're using that drug on is sufficiently cautioned not to ship that animal to slaughter. As far as solutions that you prepare, I think the same thing basically applies although basic elements and so forth-electrolytes-they're not considered drugs. Unless a producer comes in with a claim, they're supplemental therapy; they're not considered drugs so you really don't have that problem as such. I think that is a fair answer.

Question: What is the advantage of glycene in some preparations? Are chloramphenicol capsules absorbed from the rumen in baby calves?

Answer: First of all I would like to confess that I really haven't analysed a bunch of different products and in the light of this, I would really like to do this. That is, analyze electrolytes and all the electrolyte products that are sold. Just let me say that the same mechanism by which sodium is absorbed along with glucose is also operative with amino acids. In other words, there is a sodium-glucose carrier mechanism that will transport sodium and glucose together, one for one. There is also an amino acid-glucose transport system that takes an amino acid on one arm and a sodium on the other, and that works also. Glycene is simply an amino acid which, I understand, exploits this same mechanism. I probably should mention the use of amino acids also in this oral solution, but since the basic research from which this came didn't use