Volume 34 | Issue 2 Article 7

1972

Fluid and Acid-Base Balance in Cattle: A Guide for Supportive Fluid Therapy

K. W. Prasse
Iowa State University

J. W. Sexton
Iowa State University

Fluid and Acid-Base Balance in Cattle: A Guide for Supportive Fluid Therapy

by K. W. Prasse,* D.V.M., Ph.D., and
J. W. Sexton,† D.V.M., M.S.

Introduction

This paper is intended for the bovine practitioner. Fluid and acid-base balance is a complex subject and application of basic principles can be difficult in the busy rural practice. However, while we recognize that the veteriarian's primary role is to assist the animal in elimination of the primary disease, the reward of success will often be enhanced through use of supportive fluid therapy.

At Iowa State University we are developing more sophistication in our approach to supportive fluid therapy in the bovine species. This sophistication comes through careful monitoring of serum electrolyte levels and blood acid-base analysis. The instrumentation necessary for this approach is still outside the realm of practicality for the bovine practitioner. Therefore a simple and practical approach for supportive fluid therapy will be presented.

* Dr. Prasse is an associate professor in the Department of Veterinary Pathology, Iowa State University.
† Dr. Sexton is an associate professor in the Department of Veterinary Clinical Sciences, Iowa State University.

Water Requirement and Dehydration

The total body water content is approximately 60% of the body weight. Daily water intake is about 10% of the body weight (greater for young animals). This intake is equivalent to 40 liters (10–11 gallons) in a 1000 lb. cow.[4] Daily water loss equals daily intake and occurs through the skin, lung, kidney and gastrointestinal tract.[6]

Dehydration occurs following reduced water intake or increased water loss. The most common cause of reduced intake in cattle is complete anorexia. Examples of common clinical conditions resulting in complete anorexia and subsequent dehydration are toxic mastitis and toxic metritis.

Increased water loss occurs most frequently with diarrhea, especially in calves. Several other conditions result in profound dehydration by water loss, but the route of loss is not as obvious. These conditions include acute rumen overload, acute abomasal or intestinal atony, torsion, displacement or obstruction. In these conditions water is osmotically sequestered in the gastrointestinal tract by the large volume of putrifactive ingesta. Other forms of dehydration caused by increased water loss are acute massive hemorrhage and polyuric syndromes, neither of which are common in cattle.

Clinical Evaluation for Dehydration

The first consideration in evaluation of the clinical case is simply knowledge of the condition in question. Is reduced water intake or increased water loss anticipated such as in the diseases described above? However, further evaluation for the degree of dehydration can be made by consideration of body weight loss, clinical signs and a few simple laboratory procedures.

Body weight loss is a sensitive index to the degree of rapidly advancing dehydration, but may be difficult to assess in the clinical situation. Rules of thumb which have been advanced are as follows:[2,4]

1. 8–10% weight loss in a 1000 lb. cow is approximately equivalent to a 40 liter (10–11 gallon) water deficit.
2. 5–10% weight loss in a 150 lb. calf is approximately equivalent to a 2–4 liter (½–1 gallon) water deficit.
3. 10–20% weight loss in a 150 lb. calf is approximately equivalent to a 4–8 liter (1–2 gallon) water deficit.

Clinical signs associated with the state of dehydration are dryness and wrinkling of skin, loss of skin elasticity, sunken eyeballs and muscular weakness. The most important fact about these findings is that once the clinical signs are observed dehydration is well advanced. The need for fluid therapy will often precede the development of these clinical signs.

Three simple laboratory procedures are useful for more critical detection and daily monitoring of the state of hydration in the animal. The first of these is hematocrit. The normal range of hematocrit values in cattle is 24–46%.[5] Individual animals, under normal conditions, will have a very constant hemotocrit value somewhere with-in the accepted normal range. Values above the maximum normal are an obvious indication of severe dehydration. However, concomitant dehydration and anemia have opposite effects on hematocrit and a single determination found to be within the normal range may be difficult to interpret. Under clinical circumstances it is seldom possible to know the base line value of hematocrit prior to the occurrence of illness. Therefore, the greatest usefulness of hematocrit lies with daily monitoring of the critical case where day-to-day changes of 3 or 4% hematocrit value, up or down, parallels the changing state of hydration.

Plasma protein determination may also be used for detection of dehydration. Plasma protein values may be read directly by refractometry.* Normal plasma protein values in cattle are: under 1 year, 6 grams%; young adults, 7 grams%; adults (lactating) 8–9 grams%.[5] Recorded values above the normal are most frequently caused by dehydration. As with hematocrit values, day-to-day changes in plasma protein concentration will parallel changing states of hydration.

Urine specific gravity is another index useful in evaluation for dehydration. The specific gravity of urine is primarily affected by the salt concentration of urine. Normal values in cattle range from 1.025 to 1.045 with an approximate mean value of 1.035.[1] Disease states accompanied by total body water deficit will result in stimulation of physiologic mechanisms for conservation of water. Hence, renal conservation of water occurs and ur'ne with high specific gravity is produced.

Acid-Base Imbalance

Certain disease conditions in cattle are accompanied by clinically significant acid-base imbalance. Failure to recognize this imbalance and take steps to alleviate it often results in loss of the case where attempts to eliminate the etiology may otherwise be successful. The first step in clinical recognition of acid-base imbalance is knowledge of the type of imbalance usually associated with the disease in question.

Acidosis occurs in the following conditions:[2,6]

---

* A. O. Refractometer, Scientific Products, Evanston, Illinois.

1. Diarrhea—loss of bicarbonate
2. Acute rumen overload — excessive lactic acid production
3. Pneumonia—failure to blow off CO_2
4. Ketosis, starvation—excess keto acid production

Alkalosis occurs in the following conditions:

1. Displacement, intussusception, torsion or volvulus of the intestinal tract—reasons may be reduced absorption of acidic gastric secretions, vomition of acidic abomasal contents into the forestomachs where they are neutralized, or absorption of products from putrefying ingesta which have an alkaline nature in herbivores.[6]
2. Renal disease and uremia — decreased elimination of cation in urine of herbivores (in carnivores uremia results in acidosis).[3]

Clinical Recognition of Acidosis and Alkalosis

For the bovine practitioner the most important facts in clinical recognition of acid-base imbalance are accurate diagnosis of the primary disease and knowledge of the nature of imbalance which accompanies that disease. Beyond this, careful evaluation of the nature of respiration is the most useful aid.

Normal respiratory rates in cattle are: adult, 30 per minute; young adult, 34 per minute; calf, 35–40 per minute.[7] The state of acidosis will result in an increased respiratory rate (attempt to compensate by blowing off acid in the form of CO_2) and increased depth of respiration. Pneumonia, which is accompanied by acidosis, will be characterized by respiratory dyspnea. Alkalosis results in a decreased respiratory rate (attempt to compensate by conservation of acid in the form of CO_2). Rates of 5 to 15 per minute have been common with alkalosis in our experience. Respiration may be shallow or deep in alkalotic states.

Urine pH may also be used for detection of acidosis or alkalosis, but in our experience has been unreliable in this regard. Normal urine pH in cattle is 7.4–8.4.[1] Values above or below this normal range would denote alkalosis or acidosis respectively. However, we have detected severe acidosis and alkalosis in cattle in which urine pH did not reflect the imbalance.

Administration of Fluids and Formulations

Suggestions:

1. Evaluate acid-base status and need for fluid.
2. Think in terms of *gallons* of fluids per day.
3. "Normal" or "balanced" electrolyte solutions are satisfactory for most cases where acid-base imbalance is not a problem.
4. For rehydration of adult cattle, avoid solutions with 5% or greater glucose concentration. Glucose will promote diuresis.
5. Avoid oral or intravenous glucose solutions which do not contain electrolytes. Attempts to rehydrate with oral or intravenous glucose will produce hyponatremia and subsequent muscular weakness.

Formulations:

1. Rehydration of adult cattle by oral route:
   sodium chloride
   (NaCl) . . . . 240 g. (1 Cup)
   water . . . . 5 gallons
   Give 5 to 10 gallons in 24 hours.
2. Rehydration of adults or calves by intravenous route:
   a. any commercial preparation referred to as a balanced electrolyte solution
   b. self-prepared formulation[4]
      sodium chloride
      (NaCl) . . . . 5.5 g.
      sodium acetate
      ($Na\ C_2H_3O_2 \cdot 3H_2O$) . 5.0 g.
      potassium acetate ($KC_2 H_3O_2 \cdot 3H_2O$) . . . . . . 1.0 g.
      distilled water q.s. . . . . 1.0 liter
      Give an adult 20 to 40 liters in 24 hours.

Give a calf 3 to 5 liters in 24 hours.

3. Correction of alkalosis and dehydration in adults:
   sodium chloride (NaCl) .. 9.0 g.
   distilled water q.s. ...... 1.0 liter
   Give an adult 20 to 40 liters intravenously in 24 hours.
4. Correction of acidosis in adults:
   sodium bicarbonate ($NaHCO_3$) .... 15.0 g.
   sterile distilled water q.s. . 1.0 liter
   (Final bicarbonate solution is unstable if sterilized.)
   Give 2 to 3 liters (1000 lb. cow) intravenously.
   The cow will hyperventilate during administration.
   Overdosage may cause tetany which is easily corrected with Ca therapy.
5. Correction of acidosis and dehydration in calves (calf diarrhea) by oral route:[2]
   sodium chloride (NaCl) ... 117 g.
   potassium chloride (KCl) .. 150 g.
   sodium bicarbonate ($NaHCO_3$) .... 168 g.
   potassium phosphate (KH_2PO_4) .... 135 g.
   glucose (dextrose) .... 500 g.
   TOTAL 1070 g.
   Add 56 g. of above mixture to 1 liter distilled water.
   Give 2 pints orally every 2 hours.

Intravenous apparatus:

1. Materials needed:
   a. 20 liter container with bottom faucet.
   b. Spiral coil, (527 F), drip chamber, (527 C) and Tuohy-Borst adapter, (527 C). These are available from Peder-Pederson Ltd., 31 Manor Park Cresent, Box 818, Guelph, Ontario.
   c. Rubber tubing
   d. Intramedic polyethylene tubing, Mfg. by Clay Adams, Division of Becton Dickinson and Co., Parsippany, New Jersey
      P E 205, 0.062″ I.D. (for adults)
      P E 100, 0.034″ I.D. (for calves)

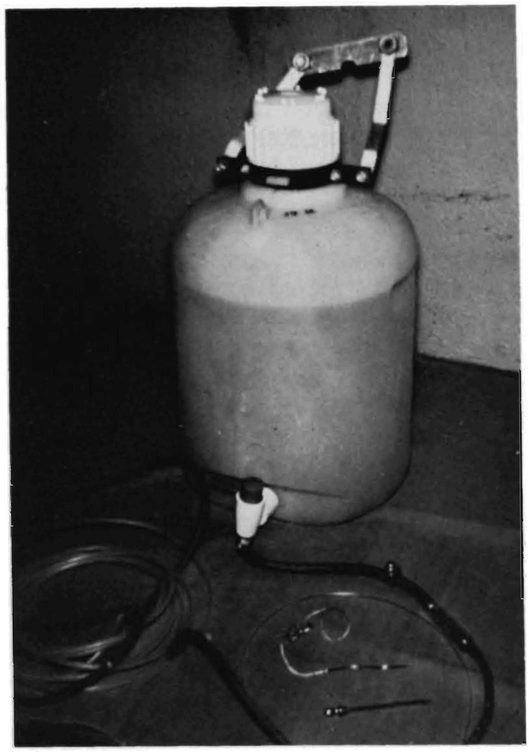

Figure 1. Assembled IV Apparatus.

2. Method: See figure 1 for assembled appartus.
   a. Puncture the jugular vein with a 12 ga. needle.
   b. Thread 6 to 8 inches of 0.062″ I.D. polyethylene tubing into the vein through the needle. Use a 14 ga. needle and 0.034″ I.D. polyethylene tubing in calves.
   c. Pull out the needle leaving the polyethylene tubing in the vein.
   d. Attach the Tuohy-Borst adapter to the catheter.
   e. Suture the adapter to the skin.
   f. Attach the rubber tubing to the adapter and the spiral coil. The drip chamber may be placed in a section of rubber tubing between the coil and the fluid container.
   g. By counting the drops through the drip chamber (15 drops/ml $\times$ time) calculate the rate of flow. A satisfactory rate is about 2 liters per hour (running fluid as fast as possible and still forming drops in the drip chamber).

Conclusion

We have attempted to provide the practitioner a foundation upon which indication for fluid therapy and proper selection of fluid may be considered when confronted with the clinical case. Simplicity in approach and efficient use of the practitioner's time can be maintained if initial time is spent preparing formulations and equipment. The addition of a regimen of supportive fluid therapy to one's armament of therapeutic measures will add a very professional and rewarding dimension to the rural practice of bovine medicine.

REFERENCES

1. Benjamin, M. M. 1961. Outline of Veterinary Clinical Pathology, 2nd. ed., Iowa State University Press, Ames.
2. Donawick, W. J. and Christie, B. A. 1971. Clinicopathologic conference. J.A.V.M.A. 158: 501-510.
3. Fisher, E. W. 1969. Hydrogen ion concentration—anion—cation (acid-base) balance. *In* Veterinary Clinical Pathology. W. Medway, J. E. Prier and J. S. Wilkinson eds.. Williams and Wilkins, Baltimore.
4. Hammond, P. B. 1962. Supportive therapy in the rural practice. Minn. Vet. 2: 20-22.
5. Schalm. O. W. 1965. Veterinary Hematology, 2nd ed., Lea and Febiger, Philadelphia.
6. Tasker, J. B. 1971. Fluids, electrolytes, acid-base balance. *In* Clinical Biochemistry of Domestic Animals Vol. II, 2nd. ed., J. J. Kaneko and C. E. Cornelius eds., Academic Press, New York.
7. Tenney, S. M. 1970. Respiration in mammals. *In* Dukes' Physiology of Domestic Animals, M. J. Swenson ed., Cornell University Press, Ithaca.

Transplacental Passage of Drugs and Other Exogenous Compounds: A Review-Part II

by Gary A. Eckhoff,* D.V.M., M.S.

METABOLISM OF DRUGS BY THE PLACENTA

In addition to and in conjunction with its transport functions, the placenta posesses metabolic activity consisting of many enzyme systems which function in the biosynthesis, degradation, and biotransformation of numerous endogenous compounds. Claude Bernard was the first to discover glycogen in the placenta[57]. The role of placental glycogenolysis in maintaining early fetal glucose stores has also been elucidated[54]. In 1965 Mori discovered protein biosynthesis in the placenta[38]. This study utilized rabbits and involved placental S^{35} incorporation into protein. Numerous active enzyme systems exist which function in the biosynthesis and degradation of steroids. It was also discovered that conjugation of steroids markedly decreased their placental transfer[26,11]. Thus there are extensive enzymatic capabilities in the placenta for endogenous compounds. It would be interesting to speculate on the effects of drugs on these metabolic activities of the placenta. This is an area with very little investigative effort and resultant alteration in placental function could be of considerable importance to fetal physiology.

Only within the last five years has investigative effort revealed the ability of the placenta to function in the biotransformation of drugs and other foreign compounds. Consideration of molecular change of pharmacologic agents given to the pregnant female and assayed in the fetus have led workers toward investigate efforts in this area. As noted above, pharmacologically administered steroids to the mother are generally considered to pass through the placenta only in a nonmetabolized state[26]. It is thus essential to under-

* Dr. Eckhoff is an instructor of Veterinary Physiology and Pharmacology, Iowa State University.

† Presented as a partial fulfillment of a master's degree from the University of Minnesota, College of Veterinary Medicine, St. Paul, Minnesota.