

## **Hypokalemia, Muscle Weakness and Recumbency in Dairy Cattle (17 Cases 1991-1998)**

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### **ABSTRACT**

Seventeen cases of severe hypokalemia (serum or plasma potassium  $\leq 2.1$  mEq/L) in association with profound muscle weakness and recumbency in lactating dairy cattle were included in a retrospective study. The cattle were from 15 different farms. Thirteen of the 17 cows were recumbent at presentation whilst the remaining 4 became recumbent within 6 hours of admission. Both multiparous (n=11) and first calf heifers (n=6) were affected. The median days in milk was 21 (range 5-110) and chronic, recurrent ketosis (15 of the 17 cases) was the most common pre-existent condition. Potential musculoskeletal and neurologic causes of recumbency were ruled out on the basis of physical examination and ancillary diagnostics. Ten of the 17 cattle were euthanized and underwent full necropsy examination that demonstrated ischemic muscle damage subsequent to recumbency and varying degrees of hepatic lipidosis. Aggressive potassium supplementation was instituted in all 17 cases either orally, intravenously or by a combination of both routes. In the 7 individuals that survived, potassium supplementation was by both oral and intravenous routes in 5, by the oral route only in 1 and intravenously only in 1.

### **INTRODUCTION**

Hypokalemia in cattle is commonly encountered secondary to anorexia as well as a number of primary conditions of the gastrointestinal and urinary systems. However, the types of clinical signs seen with these conditions are rarely attributed specifically to hypokalemia and it is not an electrolyte abnormality that is typically associated with muscle weakness, recumbency and the down cow syndrome. Recently, a syndrome of severe muscle weakness, recumbency and hypokalemia was reported in association with ketosis and the intramuscular administration of isofluprednone acetate to lactating dairy cattle<sup>1</sup>. Hypokalemia has also been documented as a potential cause of muscle

weakness in cattle of varying ages, independent of corticosteroid administration<sup>2</sup>. The purpose of this retrospective study was to characterize the history, presentation and treatment of severe hypokalemia in dairy cattle with muscle weakness and recumbency.

## **MATERIALS AND METHODS**

### **Criteria for Selection of Cases**

Medical records of cattle presented to the large animal hospital at the New York State College of Veterinary Medicine at Cornell University between 1992 and 1998 were reviewed retrospectively. Cattle that demonstrated a serum or plasma potassium of  $\leq 2.1$  mEq/L at admission and that were concurrently normocalcemic, normomagnesemic and not hypoglycemic were eligible for the study. Information obtained from the medical record for each case included age, breed, days in milk (DIM) at presentation, medical and treatment history from the current lactation, selected biochemical data at presentation, the route of potassium supplementation during hospitalization, case outcome and post mortem findings where applicable.

## **RESULTS**

Table 1 gives case details including the age, breed, DIM and selected biochemical data at presentation for each cow. The medical history for the current lactation is also summarized in Table 1, as well as whether the animal was recumbent at presentation (12 cases), and if so for how long, or able to stand but profoundly weak (5 cases). All cattle that were profoundly weak at admission became recumbent within 6 hours of admission to the hospital. The serum or plasma potassium at presentation, the mode of potassium supplementation during hospitalization (K suppl.), and the case outcome are also summarized in Table 1.

Cases tended to be concentrated in the first 45 days of lactation with a median DIM of 21 and a range of 5 to 110. There were 6 first calf heifers and 11 multiparous cows. Fifteen of the 17 cases had a history of chronic ketosis during the current lactation, where chronic ketosis is defined as moderate to severe ketonlactia or ketonuria for 5 days or more that had been treated on farm on at least 3 separate occasions. Pre-admission therapy of the 15 cases with a history of chronic ketosis was quite variable but all of them had received both 500 ml of 50% dextrose intravenously and between 6 and 18 oz of propylene glycol on at least 3 separate occasions. Ten of these 15 cases had received isofluprednone acetate parenterally on multiple occasions, 4 others had received dexamethasone parenterally or orally on multiple occasions, and one case had received both dexamethasone and isofluprednone acetate parenterally on multiple occasions. The two cattle with no history of chronic

ketosis (cases 12 and 13) had a history of clinical mastitis that had been treated with intramammary infusions of 20-40 mg of isofluprednone acetate after each of 15 consecutive milkings. Cases 2, 3, 10, 11 and 15 had received 10-20 mg of isofluprednone acetate parenterally on 3 separate occasions, case 4 had received 20 mg of isofluprednone acetate parenterally on 4 occasions, cases 6, 8 and 14 had received 10-20 mg of isofluprednone acetate parenterally on 5 occasions and case 17 had received 10 mg of isofluprednone acetate parenterally on 6 occasions. Cases 1 and 9 had received 25 mg of dexamethasone parenterally on 3 occasions, case 16 had received 12.5-25 mg of dexamethasone parenterally on 4 occasions and case 5 had received dexamethasone at an unspecified amount on 5 or more occasions both parenterally and orally. Case 7 had received 20 mg of isofluprednone acetate twice and 25 mg of dexamethasone twice parenterally, each steroid administration occurring on a separate occasion.

Cases 2, 3 and 8 had a history of insulin administration as part of the therapy for chronic ketosis. Adjunct therapy for ketosis included niacin boluses in 5 cases (case 2, 3, 6, 10 and 17) and multivitamin preparations in 8 cases (cases 2, 3, 5, 6, 8, 10, 14, 16, and 17).

The clinical signs at presentation observed in the cattle of this report included muscle fasciculations, weakness, difficulty rising and recumbency. Five of the cases (cases 2, 7, 15, 16 and 17) were documented to be in atrial fibrillation at initial presentation. Twelve of the 17 cows were recumbent and unable to stand at presentation, whilst the 5 remaining individuals were profoundly weak and became recumbent within 6 hours of admission. In 13 of the 17 cases the weakness was so severe at some point during hospitalization that cattle could not rise out of lateral recumbency and required external support to maintain them in a sternal position (Figure 1). Of these 13 cases, 8 were euthanized and 5 recovered. Of the 4 cases that could maintain sternal recumbency unassisted (cases 2, 11, 15 and 17), 2 were ultimately euthanized and 2 recovered, suggesting that the ability to remain in sternal recumbency was not related to recovery.



Figure 1. Dairy cow with severe hypokalemia (case 17) in lateral recumbency. The cow was completely unable to support the weight of the head and could not rise out of lateral recumbency.

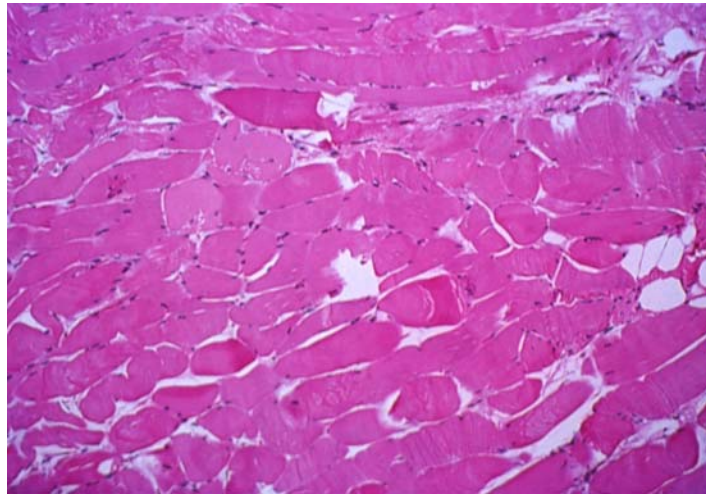
Table 1 gives selected biochemical data obtained at admission from all 17 cases. In addition to severe hypokalemia, other pertinent findings on serum biochemistry included elevations in the muscle specific enzyme creatine kinase in all cases (17 cases). In addition all 17 cattle had elevations in the levels of aspartate aminotransferase (AST). This enzyme is also released into serum subsequent to hepatocellular injury and 8 cattle also had elevations in serum activity of the enzyme gamma glutamyl transpeptidase (GGT), which is specific to biliary epithelium. It is not possible to say what proportion of the elevation in the serum activity of AST was the result of myopathy or of hepatocellular origin. Only one cow (case 17) had normal acid base status and serum chloride levels on admission, all other cases demonstrated either hypochloremic metabolic alkalosis (9 cases), hypochloremic metabolic acidosis (5 cases), or normochloremic metabolic acidosis (2 cases). All 7 of the cattle demonstrating metabolic acidosis at presentation were ultimately euthanized, compared to only 2 of the 9 cattle with metabolic alkalosis. Hypophosphatemia was documented in 4 cases at presentation. On presentation all cattle demonstrated ketonuria that was at least moderate (40 mg/dl) on urine dipstick examination.

## **CASE MANAGEMENT**

Treatment of the cattle in this report included aggressive potassium supplementation, intravenous fluid support and nursing care. Table 1 details whether potassium supplementation was intravenous or oral or both for each case. The amount of potassium administered to each case varied quite significantly between individuals. Intravenous potassium was typically administered in the form of supplemental potassium chloride added to polyionic fluids, at potassium administration rates that varied from 60 mEq/hr to 315 mEq/hr. The dose and frequency of oral potassium supplementation were also highly variable, with individual cattle receiving between 120 grams (1600 mEq) three times daily and 500 grams (6800 mEq) twice daily diluted in warm tap water by orogastric tube. Several of the cattle that received the highest levels of oral potassium supplementation developed moderate to severe diarrhea. In animals that died and in those that survived, restoration of normokalemia was challenging. The median duration of potassium supplementation required to achieve normokalemia in those that recovered was 3 days, with a range of 1 to 7 days. A variety of devices and techniques were used to assist down cattle to stand including hip lifters, slings and a flotation tank, in some cases more than one device was used during the management of that individual.

## **CASE OUTCOME**

Ten of the 17 cattle in this study were euthanized at the owner's request. The remaining 7 individuals recovered and were discharged from the hospital. The decision to euthanize cattle was based upon continued recumbency of greater than 72 hours duration and/or financial considerations. All euthanized individuals underwent a full necropsy examination to rule out other potential causes of weakness or recumbency such as traumatic musculoskeletal injuries, spinal neoplasia or severe systemic disease. Gross and histologic evidence of myopathy was identified in weight bearing muscles of all euthanized animals. In addition in cases 6, 8 and 17 muscle tissue was obtained from non-weight bearing muscles (diaphragm, cervical and intercostal musculature) and demonstrated lesions of acute, multifocal, myofiber degeneration and necrosis consistent with hypokalemic myopathy in humans and cats<sup>3,4,5</sup> (Figure 2). Moderate (cases 5, 6, 7, 8, 11, 13, and 17) to severe (cases 2, 4, and 10) hepatic lipidosis was also noted at post mortem.



**Figure 2. Photomicrograph of intercostal muscle obtained from case 17 demonstrating acute, multifocal myofiber degeneration, vacuolation and necrosis. Acute myolysis with myofiber fragmentation along Z lines and amorphous residual eosinophilic material are visible**

## **DISCUSSION**

There are a number of important factors that could have contributed to the development of hypokalemia in the cattle of this report. The typical forage based diets offered to dairy cattle are relatively high in potassium and as a consequence urinary potassium excretion is high<sup>6,7</sup>. This adaptation to a high potassium intake diet significantly contributes to the development of hypokalemia with any condition causing a reduction in voluntary feed intake because urinary potassium excretion cannot be reduced sufficiently or rapidly enough to maintain external potassium balance<sup>7</sup>. However hypokalemia from anorexia alone tends to be mild and asymptomatic. Physiologically hypokalemia can also be the consequence of the intracellular movement of potassium due to metabolic alkalosis or insulin release secondary to hyperglycemia or pathologically as the result of increased potassium loss through the alimentary or urinary systems in cases of intestinal or renal disease<sup>6,8,9</sup>. However, it is worth remembering that only about 10% of the body's total potassium is found extracellularly, with the plasma level representing only 0.4% of the total<sup>10</sup>. For these reasons plasma or serum potassium measurement can be an imprecise means of establishing

whole body potassium status. Case 17 provides an example of an individual with one of the higher serum potassium measurements at admission but clinical evidence of severe hypokalemia resulting in a complete inability to rise out of lateral recumbency and histologic features in non-weight bearing muscles consistent with hypokalemic myopathy in other species<sup>3,4</sup>. The severity of the hypokalemia at presentation, the chronicity of their illness and the histologic lesions consistent with hypokalemic myopathy in non-weight bearing muscles such as those of the diaphragm, neck and intercostal area would at least be suggestive of severe whole body potassium depletion in several cattle of this report.

Hypochloremia and metabolic alkalosis were the most common serum electrolyte and acid base abnormalities accompanying hypokalemia at presentation in the cows of this report, although metabolic acidosis was identified in 7 cases. All 7 cases that demonstrated metabolic acidosis at presentation were ultimately euthanized. Five of the 7 individuals exhibiting a metabolic acidosis had been recumbent for a period of several hours to days prior to admission. Three of these plus the 2 with metabolic acidosis that were still able to stand on admission also had a documented increase in the anion gap consistent with an increase in unmeasured anions. Possible sources of these unmeasured anions would include both lactate, potentially from muscle damage due to intermittent or complete recumbency, poor tissue perfusion or a combination of both, and ketoacids (all individuals demonstrated urine ketone measurements of at least 40 mg/dl on admission). Somewhat paradoxically it is interesting to note that 1 of the 2 individuals with metabolic acidosis but a normal anion gap, also had the highest creatine kinase at admission (case 10). However, the fact that all 7 animals with metabolic acidosis at presentation were ultimately euthanized suggests that this is a negative prognostic sign.

Hepatic lipidosis was present at post mortem in all 10 of the euthanized animals and 7 of these individuals also had elevations in the hepatobiliary enzyme GGT at admission. A mild degree of hepatic lipidosis is expected, and considered normal, during the period of negative energy balance in early lactation<sup>11,12</sup>. However, chronic ketosis would have predisposed the cattle of this report to more significant hepatic lipid infiltration and could have contributed to some of the clinical signs and biochemical abnormalities detected because severe hepatic lipidosis can itself lead to severe anorexia, weakness, recumbency and death. Metabolic acidosis would also be an expected acid-base abnormality with advanced hepatic lipidosis and fulminant hepatic failure<sup>11</sup>. Metabolic acidosis and biochemical evidence of advanced liver disease (elevations in GGT, low BUN) alongside hepatic lipidosis that was histologically classified as severe, were documented in cases 4 and 10.

Hypophosphatemia was documented in 4 of the cattle in this report and was a feature of 40% of the cows with hypokalemic myopathy and weakness in the study by Sielman *et al.*<sup>1</sup>.

Hypophosphatemia was also documented in 35% of the cattle demonstrating hypokalemia in the study by Sattler *et al.*<sup>2</sup>. The potential contributory role that hypophosphatemia may have played in the development of weakness and recumbency in the cattle of this study is uncertain. The clinical signs of severe weakness were seen with both hypophosphatemic (4 cases) and normophosphatemic (13 cases) cattle. The fact that no specific phosphorus supplementation was given to those cattle that recovered, and that their clinical signs improved with specific restoration of normokalemia only, would argue against a significant contribution from low serum phosphorus.

Recurrent ketosis was an antecedent condition in 15 of the 17 individuals in this study, a finding consistent with the report by Sielman *et al.*<sup>1</sup> The 2 cases (cases 12 and 13) that were not associated with ketosis were representative of a herd problem of weakness and recumbency in lactating cows and heifers that was associated with clinical mastitis. During a 7-week period 10 lactating animals on this farm exhibited signs of progressive weakness and recumbency. All individuals had received intramammary infusions with 20 – 40 mg isoflupredone acetate three times a day as therapy for clinical mastitis for 5-7 days immediately prior to the development of clinical signs. Although detailed biochemical information is only available for cases 12 and 13, all 10 animals showed clinical signs consistent with other animals in this study including profound weakness with an inability to support themselves in sternal recumbency.

Although all of the cows in the report by Sielman *et al.*<sup>1</sup> had received the corticosteroid, isoflupredone acetate as part of the treatment for the ketosis, this was not a feature of all the cattle in this study. Twelve of the 17 cases documented here had received isofluprednone only, whilst 4 had received dexamethasone only and one had received both isoflupredone and dexamethasone. It should be noted that in all cases where isoflupredone acetate was used, the amount used, dose frequency, or route of administration were contrary to the manufacturer's current recommendations. The package insert stipulates that 10 to 20 mg of isofluprednone acetate should be administered by intramuscular injection, and that treatment can be repeated in 12 to 24 hours if necessary. The potential for enhanced renal and gastrointestinal potassium losses due to some mineralocorticoid activity may exist following isoflupredone acetate administration, particularly when the product is used repeatedly. Although the specific mineralocorticoid activity of isoflupredone acetate is unknown, it has been shown to possess as potent a mineralocorticoid effect as aldosterone both *in vivo* and *in vitro* in an adrenalectomized rat model<sup>13</sup>. By comparison, dexamethasone is considered to have minimal mineralocorticoid effect. Values of 55%, 126%, 81%, 128% and 122% for the urinary fractional excretion of potassium were obtained from cases 7, 8, 10, 14 and 17 respectively prior to intravenous or oral potassium supplementation. Each of these 5 individuals had received multiple doses of

isofluprednone acetate in the days before presentation. However, it should be pointed out that in calculating these fractional excretion values urine potassium measurement was performed using an ion-selective electrode technique, and this methodology has been demonstrated to consistently underestimate urine potassium concentrations in cattle, putatively due to chelation by a low molecular weight compound<sup>14,15</sup>. Therefore the values calculated in these 5 individuals may actually underestimate the true fractional potassium excretion values. Of the several studies reporting values for urinary fractional excretion of potassium in cattle<sup>16,17,18</sup> only one gives data for lactating dairy cattle in early lactation, citing a reference range of 26.9% to 120%<sup>18</sup>. Based upon the data from cases 7, 8, 10, 14 and 17 it would appear that these individuals had values for urinary fractional excretion of potassium that were either within or above the normal reference range for healthy cattle in early lactation. The fact that 4 of the individuals in this report had received dexamethasone only, a steroid with minimal to no mineralocorticoid activity, suggests that severe hypokalemia with muscle weakness can certainly occur in cattle independent of isofluprednone administration.

The specifics of any association between chronic ketosis, recurrent corticosteroid administration and clinically significant hypokalemia are uncertain. Chronic ketosis or clinical mastitis could have resulted in a significant reduction in dietary potassium intake due to prolonged anorexia in each of the cows in this report. Furthermore, hyperglycemia subsequent to exogenous glucose administration and enhanced gluconeogenesis following steroid administration would further reduce plasma potassium due to intracellular potassium shifting subsequent to insulin release<sup>19,20</sup>. However, it is also worth remembering that cases 12 and 13 had no history of chronic ketosis, and therefore no history of exogenous dextrose, propylene glycol or insulin administration, and had experienced a relatively short period of mild anorexia (5-7 days). These 2 individuals, along with several others from the same farm, had received intramammary isofluprednone acetate on multiple occasions, and went on to develop severe hypokalemia and weakness, that was indistinguishable clinically from the other cattle in this study. This suggests that severe hypokalemia leading to muscle weakness can occur independent of chronic ketosis, but that it may be predisposed to by overuse of isofluprednone acetate. The possibility that recurrent corticosteroid administration could contribute to severe potassium depletion, independent of mineralocorticoid activity is suggested by those cases that received repeated doses of dexamethasone alone. However, a mechanism for enhanced intracellular potassium loss due to either a direct or indirect effect of repeated corticosteroid administration is not apparent from a review of the literature.

Potassium supplementation for cows with severe hypokalemia should ideally include both intravenous and oral administration, although from a practical standpoint oral supplementation is

frequently the chosen route. It should be remembered that kaluresis will be a consequence of diuresis with all intravenous fluids and so proprietary or homemade preparations should always contain supplemental potassium. However, in all cases of supplemental intravenous potassium administration practitioners are cautioned not to exceed a maximum infusion rate of 0.5 mEq/kg/hr, so as to avoid potential pathologic cardiac arrhythmias<sup>7</sup>. It is the opinion of the authors, based upon experience with the cattle of this study and others with less severe hypokalemia, that oral supplementation either alone or in combination with intravenous potassium supplementation more rapidly restores normokalemia than exclusively intravenous supplementation. Similar observations have been made by Sielman *et al.*<sup>1</sup> and Sattler *et al.*<sup>2</sup>. Recommendations would be not to exceed 0.5 lb of potassium chloride orally twice daily, due to the risks of inducing severe osmotic diarrhea at higher doses.

Hypokalemia should be considered in the differential diagnosis of weakness and recumbency in dairy cattle, particularly in individuals with a history of prolonged anorexia associated with chronic refractory ketosis and corticosteroid administration during the first month of lactation. Although the cases in this report tended to be concentrated in the first month of lactation one cow was 110 days in milk. Based upon this report recurrent ketosis appears to be a particular risk factor for the development of severe hypokalemia but the potential for other conditions to at least contribute to the development of this problem should not be overlooked<sup>2</sup>. Although repeated use of the corticosteroid isofluprednone acetate has previously been associated with severe hypokalemia<sup>1</sup>, this was a common, but not invariant feature, of the cattle in this report. Based upon the observations in this retrospective study it is not possible to establish a cause and effect between any or all of the various therapeutic modalities that cattle had received prior to admission and the development of severe hypokalemia. Reduced potassium intake, intracellular shifting of potassium subsequent to metabolic alkalosis and hyperglycemia, kaluresis due to hyperglycemic osmotic diuresis, and increased potassium loss due to the mineralocorticoid effects of exogenously administered corticosteroids are all potential contributory factors to the development of clinically significant hypokalemia in the chronically ketotic cow. Excessive use of corticosteroids with mineralocorticoid activity for conditions other than ketosis may also predispose cattle to the development of severe hypokalemia. Subsequently, oral potassium supplementation to at risk animals, particularly those showing signs of early hypokalemia including muscle fasciculations and weakness is recommended.

<sup>a</sup>Isofluprednone acetate: Predef® 2X, Pharmacia and Upjohn, Kalamazoo, MI, USA.

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## Preconvention Seminar 7: Dairy Herd Problem Investigation Strategies

Case	Age (yr)	Breed	DIM	Medical History	Status at Admission	K (3.9-5.8 mEq/L)	K suppl.	AST (48-107 IU/L)	CK (83-1357 IU/L)	GGT (13-39 IU/L)	BUN (10-29 mg/dl)	P (3.9-9.2 mg/dl)	Cl (96-104 mEq/dl)	Venous pH (7.35-7.45)	Base Excess (0-6)	Anion Gap (6-20 mEq/L)	Outcome
1	2	Hol	22	Chronic ketosis	Down, 2 hours	2.1	Oral and i.v	901	24555	35	24	4.8	87	7.51	18.4	16	Recovered
2	4	Hol	22	Chronic ketosis	Down, 6 hours	1.8	i.v	155	27800	276	22	3.8	97	7.21	-12.0	32	Euthanized
3	3	Hol	11	Chronic ketosis, metritis	Down, 3 hours	2.1	i.v	397	3751	20	15	8.9	92	7.50	12.2	13	Recovered
4	3	Hol	18	Chronic ketosis	Profoundly weak	1.6	i.v	210	893	91	9	8.3	95	7.56	16.3	5	Euthanized
5	2	Hol	5	Chronic ketosis, metritis	Profoundly weak	2.0	i.v	1510	17730	22	26	3.5	73	7.28	-8.7	22	Euthanized
6	8	Hol	11	Chronic ketosis, mastitis	Down 48 hours	1.7	i.v	788	27600	39	10	4.4	93	7.33	-0.7	11	Euthanized
7	2	Hol	21	Chronic ketosis	Down 12 hours	1.8	i.v	1111	24082	43	22	4.3	82	7.25	-10.9	27	Euthanized
8	4	Hol	32	Chronic ketosis	Down 24 hours	1.7	i.v	198	3188	67	9	6.5	85	7.53	13.2	16	Euthanized
9	4	Hol	28	Chronic ketosis, pneumonia	Profoundly weak	2.0	Oral and i.v	115	1860	17	13	4.6	85	7.54	17.8	18	Recovered
10	6	Hol	42	Chronic ketosis	Profoundly weak	1.9	Oral and i.v	1760	97600	121	7	6.3	96	7.25	-5.7	19	Euthanized
11	4	Hol	10	Chronic ketosis, metritis	Profoundly weak	1.8	Oral	211	8884	35	21	4.8	95	7.27	-9.9	29	Euthanized
12	5	Hol	110	Mastitis	Down 12 hours	2.1	Oral	413	2378	18	11	5.0	94	7.48	15.0	17	Recovered
13	2	Hol	64	Mastitis	Down 6 hours	1.8	Oral	169	12346	65	8	1.1	77	7.16	-13.9	43	Euthanized
14	6	Jer	7	Chronic ketosis	Down 12 hours	2.1	Oral and i.v	148	1844	71	30	6.0	87	7.57	15.5	13	Recovered
15	2	Hol	12	Chronic ketosis, mastitis	Profoundly weak	1.9	Oral and i.v	453	24590	44	16	6.1	91	7.49	11.2	15	Recovered
16	2	Hol	11	Chronic ketosis, metritis	Down 24 hours	1.9	Oral and i.v	180	9180	23	23	4.9	81	7.53	18.9	14	Recovered
17	3	Hol	18	Chronic ketosis	Down 12 hours	2.1	Oral and i.v	298	2068	60	18	0.9	100	7.35	1.1	16	Euthanized

Table 1. Selected Clinical and Biochemical Data From 17 Dairy Cattle With Severe Hypokalemia (all biochemical data represent admission values, normal ranges in parentheses)