Assessment of Sodium Deficiency and Polyuria/Polydipsia in Dairy Cows

W.G. Olson, *D.V.M., Ph.D*
K.R.J. Link, *D.V.M.*
Department of Large Animal Clinical Sciences
University of Minnesota
St. Paul, Minnesota 55108
D.E. Otterby, *Ph.D*
Department of Animal Science
University of Minnesota
St. Paul, Minnesota 55108
J.B. Stevens, *D.V.M, Ph.D*
Department of Pathology
School of Veterinary Medicine
North Carolina State University
Raleigh, North Carolina 27606

Summary

Sodium (Na) deficiency was observed as a cause of decreased production and associated with polyuria/polydipsia (PU/PD) in a herd of dairy cows. Subsequent investigations revealed other herds experiencing milk loss due to Na deficiency, however PU/PD was not observed in all cases. To further study Na deficiency and diagnostic tests to confirm a diagnosis of NaCl deficiency, sixteen mid-to-late lactation dairy cows were divided into 2 groups for a 30 day Na depletion study. The control group (C) received 0.75% NaCl in their concentrate while the deficient group (E) was fed a ration without supplemental Na. There was a significant difference in production decline (9.5% vs. 29.3%, $P<0.01$) between C (25.1 to 22.7 kg) and E (18.8 to 13.3 kg). A significant difference in urine Na was observed between C vs. E (29.7 to 21.4 vs. 43.4 to 1.8 mEq/L, $P<0.001$) and in FE_{Na} of C vs. E (0.357 to 0.205 vs. 0.329 to 0.016%, $P<0.001$). A significant decline in serum K was demonstrated between C vs. E (4.43 to 4.61 vs. 4.50 to 3.65 mEq/l, $P<0.01$), while FeK increased (39.27 to 36.68 vs. 39.47 to 50.78%, $P<0.001$). There were no significant differences observed in serum Na or other serum parameters between groups. The diagnostic value of FeNa and urine Na determinations was very high. A significant difference between control and depleted animals in both determinations was observed in both experimental and field conditions. Therefore the simple determination of urinary Na concentration in a herd of lactating dairy cows can be used to diagnose Na deficiency. Sampling of 5 high producing cows would be recommended since they would be at greatest risk of deficiency in borderline salt supplementation situations.

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Introduction

In the past 80 years there have been several comprehensive papers on Na deficiency.[1-6] The dairy cow is unique in its extra requirement of Na for milk production. NRC (National Research Council) recommendation for sodium chloride (NaCl) is 0.45% of the total daily dry matter intake.[7] Common guidelines recommend inclusion of 0.75% to 1.0% NaCl in the concentrate portion of the diet. However, for a variety of reasons NaCl is sometimes omitted from the ration and the error may not be recognized until several weeks to many months later.

Na deficiency is manifested in several ways such as decreased milk production, pica, polyuria-polydipsia (PU/PD), decreased water intake, decreased urinary output, decreased feed intake, loss of body weight and/or lower gains.[6,8-14]

Laboratory findings in Na deficiency include a decrease in salivary, urinary, ruminal and fecal Na concentrations, a concurrent increase in salivary, ruminal and fecal K concentrations, a decrease in salivary and ruminal Na/K ratios, and an increase in serum aldosterone concentration.[6,9-22] Urinary K concentrations may be increased or decreased.[10,13,21,22] Serum and milk concentrations of Na and K may be normal or decreased in Na deficiency.[6,9,12,14,20-24] Urine specific gravity may be low or high depending on total solute concentration and the presence or absence of PU/PD.[16] Packed cell volume (PCV) and hemoglobin may be normal or increased.[10,23-24] Histopathologic evaluation of adrenal glands and parotid salivary glands revealed increased weights of both glands, an increase in the width of the zona glomerulosa of the adrenal cortex, and a more extensive duct system in the parotid salivary glands.[6,11-12,15] Adrenal histology is a sensitive index of an individual animal's NaCl status,

however, the pathological evaluation of the adrenal glands is only useful from a survey.

In dairy cattle it has been shown that a linear relationship exists between dietary Na intake and urinary Na excretion.[8] Previous studies in cattle on NaCl deficient diets revealed urinary Na concentrations ranging from 0.02 to 22.0 mEq/L.[6] In lactating dairy cows, a positive milk production response to NaCl supplementation was observed when urine Na concentration was < 3.0 mEq/l prior to increased NaCl supplementation.[6,14] When NaCl supplementation was increased from 0% to 0.73%, a 4.7% increase in milk yield was noted.[25] Increasing dietary Na and K was found to increase milk yields.[26] It was therefore concluded that when the urinary Na concentration was < 3.0 mEq/l a positive milk production response to re-supplementation should be expected.[6]

In contrast, a chloride (Cl) deficiency in dairy cows (0.18% dietary Cl) produced no effect on milk production, feed intake or water consumption.[3,25]
The only significant laboratory findings were a decrease in serum, urine and fecal Cl and a slight decrease in serum K.[27] Dairy calves on 0.038% dietary Cl exhibited PU/PD. Laboratory findings included a decrease in serum, urine, fecal, and salivary Cl, which indicate an active conservation of Cl by the body.[28,29] In one study, urine Na and K concentrations were decreased; however, there was no change in total urinary excretion of Na and K. Thus, the urinary decrease in these ions was attributable to dilution.[27] Another study found deviations in PCV, blood pH. pCo_2, and HCO_3^- and a decrease in serum K.[29]

Experimentally, simultaneous K and nitrogen loading results in a negative Na balance due to a 10 fold increase in urinary Na excretion concurrent with diuresis.[30] Return to a positive Na balance was facilitated by a decrease in urinary Na concentration rather than a cessation of the diuresis.[30] High dietary K concentrations (3.1% to 6.4% of dry matter intake) were found to increase urine output 2 to 3 fold and to also increase urinary Na excretion 16% to 18%; however, total Na loss (urine plus fecal Na) remained unchanged.[8]

Materials and Methods

Experimental Study

A group of 16 lactating (midlactation) Holstein-Friesian cows from the University of Minnesota dairy herd were randomly assigned to two groups without regard to milk production. One side of the barn served as C group in a 33-day trial. Daily milk production for each cow was recorded. Water consumption for the previous 24 hours was recorded daily for each group at 9:00 A.M. The water consumption was measured by two PVC (polyvinyl chloride) Kent 3/4 P.S.M. water meters[a]. The water meters were installed such that a direct in-line measurement was made of the water consumption of each group of 8 cows. The control diet consisted of alfalfa, hay, corn silage, and a grain mixture containing corn, oats, soybean meal, minerals and NaCl. The supplemental NaCl content of the control ration was 0.4% on DM basis. The NaCl deficient diet was identical to the control diet except that NaCl was omitted from that diet. For purposes of this experiment NRC table values for Na, K and Cl in the feed stuffs were assumed.

Blood and urine samples were collected simultaneously at 9:00 A.M. on sampling days (Monday, Wednesday and Friday). Coccygeal blood was collected utilizing evacuated serum tubes[b], EDTA[b] and NaF tubes[b]. The urine samples were collected from a midstream micturition in urine specimen cups[c] after manual stimulation. A subsample was transferred into a 20 ml evacuated serum tube[b] to ensure that there was no Na or K contamination of the sample after collection. The blood analyses conducted included PCV, glucose, magnesium[d], Na^e, K^e, and SMA-12/60 Micro[f] serum profile which consisted of albumin, calcium, inorganic phosphorus, creatinine, total bilirubin, total protein, cholesterol, BUN, ALP, ALT and AST. The PCV was determined on blood collected in tubes containing EDTA, and glucose was determined on plasma from NaF-preserved blood. The urine analyses included determination of creatinine[g], Na^e and K^e concentrations, and urine specific gravity.

The derivation of the formula for the FE_{Na} is based on the amount of Na being cleared from the body by the kidney relative to the amount of creatinine.

$$FE_{Na} = \frac{\text{Sodium Clearance}}{\text{Creatinine Clearance}} \times 100 \qquad (1)$$

Urinary loss of creatinine over a 24 hour period is relatively invariable because the loss is based on muscle mass. The coefficient of variation for urinary loss of creatinine is less than for most laboratory tests. A unique feature of the FE_{Na} laboratory test is that it utilizes only serum and urine concentrations of Na and creatinine. Urine volume is eliminated as a factor; therefore, a timed urine collection period is not required.

Creatinine clearance is defined as volume of plasma (in

[a] Power Process Equipment Inc., Minneapolis, MN.

[b] Vacutainer Brand, Becton-Dickinson and Company, Rutherford, NJ.

[c] Sweetheart Plastics Inc., Wilmington, MA.

[d] IL Atomic Absorption/Atomic Emission Spectrophotometer, Model 251, Instrumentation Laboratory Inc., Boston, MA.

[e] IL flamephotometer, Model 143, Instrumentation Laboratory Inc., Boston, MA.

[f] Technicon SMA-12/60 Micro, Technicon Instruments Corporation, Tarrytown, NY.

[g] Jaffä reaction with Lloyd's Reagent, Henry RJ: *Clinical Chemistry: Principles and Technics*. New York, Harper & Row, 1974.

milliliters) that contains the amount of creatinine excreted by the kidney per minute. Mathematically this is expressed as:

$$\text{Creatinine Clearance} = \frac{C_u \cdot Vol_{u/min}}{C_p} \quad (2)$$

Where: C_u = Creatinine concentration per ml of urine

Vol_u = ml of the urine produced per minute

C_p = Creatinine concentration per ml of plasma

Sodium clearance is the same except that it is calculated from milliequivalents (meq) rather than milligrams (mg) as is creatinine.

$$\text{Sodium Clearance} = \frac{Na_u \cdot Vol_{u/min}}{Na_p} \quad (3)$$

Where: Na_u = Sodium concentration per liter of urine

Vol_u = ml of urine produced per minute

Na_p = Sodium concentrations per liter of plasma

Referring back to formula 1 and substituting the formula for Na and creatinine clearance, the result is:

$$FE_{Na} = \frac{\frac{Na_u \cdot Vol_{u/min}}{Na_p}}{\frac{C_u \cdot Vol_{u/min}}{C_p}} \times 100 \quad (4)$$

By rearrangement of formula (4)

$$FE_{Na} = \frac{Na_u \cdot Vol_{u/min}}{Na_p} \times \frac{C_p}{C_u \cdot Vol_{u/min}} \times 100 \quad (5)$$

The terms for volume can be cancelled yielding:[31]

$$FE_{Na} = \frac{Na_u}{Na_p} \cdot \frac{C_p}{C_u} \times 100 \quad (6)$$

The FE_k can be derived in a similar manner from formula 1 by substituting K clearance for Na clearance.

The important clinical implication of this is that simultaneous collection of a serum sample and a urine sample from an animal is all that is needed. It must be realized that FE_{Na} and FE_k can also be stated as a simple ratio; i.e., clearance ratio rather than percent. The FE_{Na} and FE_k were calculated using formula 6. Data were statistically evaluated utilizing means, standard deviations and Duncan's multiple range test for variance.[33] Comparisons were considered significant when $p<0.01$ was present.

Field Studies

Several herds with the limited complaints were assessed for NaCl status (Table 1). The laboratory procedures were the same as those used in the experimental study.

Results

Experimental Study

Selected serum and urine parameters, and milk production are presented as means and standard error of the mean in Table 3 (one animal from each group was eliminated because of stage of lactation). Mean serum Na and K and urine Na, K, FE_{Na} and FE_K values are presented in Figures 1 and 2. There were no significant differences in urine specific gravity (Table 3) between the NaCl deficient and control groups; however, the E group tended to have slightly higher urine specific gravities. Also, a significant difference in serum Na (Figure 1) between the two groups was not demonstrated. The urine Na concentration (Figure 1) of the E group exhibited a significant ($p<0.001$) steady decline over time in comparison to the C group. The FE_{Na} declined from 0.329 to 0.0156% in 29 days while urine Na concentration declined from 43 to 1.8 mEq/L. FE_{Na} and urine Na mEq/L were both consistently lower by day 5 after NaCl deletion from the diet. Urine Na concentration of less than 3 mEq/L was equivalent to FE_{Na} as a diagnostic test to detect Na deficiencies as evidenced by perfect concordance when the urine Na data were ranked from lowest value to highest value and then compared visually with the FE_{Na} data. The serum K concentration (Figure 2) of the E group showed a significant ($p<0.001$) decline over time compared to the C group. There were no appreciable changes in the other ion species. Acid-base balance was not assessed. The urine K concentration was significantly higher in the E group throughout the trial (Figure 2). The FE_k (Figure 2) of the E group was higher than the C group during the experimental period. Serum BUN, creatinine, and inorganic phosphorus for the C and E groups were unchanged within and between groups, which indicated normal renal function. All other serum parameters were also within normal limits.

There was significant differences ($p<0.01$) in milk production decline during the experimental period between the E and C group. Milk production declined 29.3% in the E group while the control's declined 9.5% (Table 3). A confounding problem is that the C group produced 6.3 kg of milk per cow per day higher than the E group at the start of this experiment.

TABLE 1. Summary of Salt Deficient and Salt Adequate Herds Studied.

Herd No.	Complaint	Amount of Salt Supplemented gm/cow/day	duration	Predominant Forage
		– Deficient –		
1	PU/PD, decreased production	<28	1 ± years	Alfalfa hay
2	decreased production	28	Unknown	Alfalfa hay
3	decreased production	28	1 ± years	Alfalfa hay, corn silage
4	PU/PD, decreased production	42	1 ± years	Alfalfa hay
5	decreased production	28	6–8 months	Alfalfa hay, corn silage
		– Adequate –		
6	decreased production, intermittent diarrhea	56	—	Alfalfa hay
7	decreased production	70	—	Alfalfa hay, corn silage
8	PU/PD	98	—	Alfalfa hay
9	PU/PD	98	—	Alfalfa hay haylage
10	decreased production	56	—	Corn silage

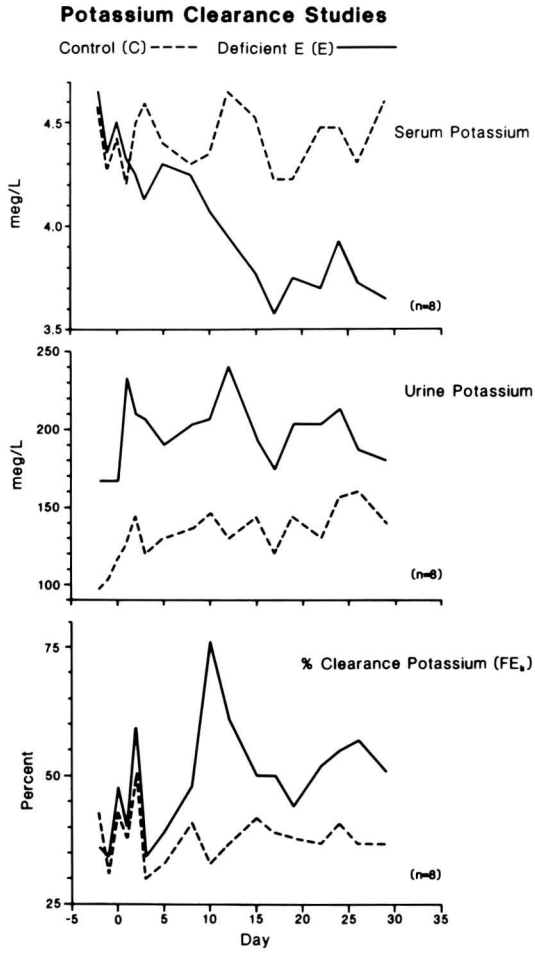

FIGURE 1. Mean values for serum and urine Na and FE_{Na}.

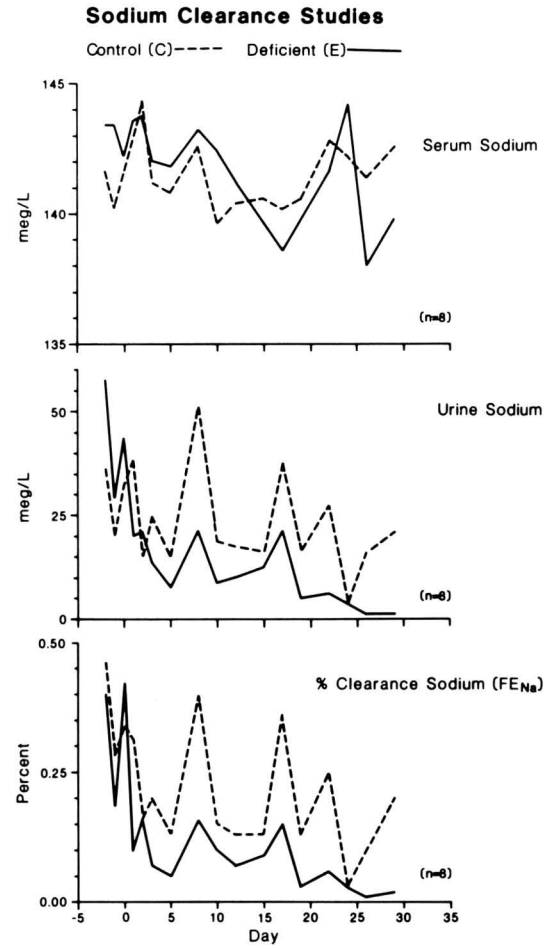

FIGURE 2. Mean values for serum and urine K and FE_K.

TABLE 2. Summary of Na and K status (X ± SD) in Salt Deficient and Salt Adequate Herds.

Herd No.	# of Cows	Urine Sp.G. mEq/L	Serum Na+ mEq/L	Urine Na+ mEq/L	Serum K+ mEq/L	Urine K+ mEq/L	FE_{Na} %	FE_K %
				– Deficient –				
1	9	1.017 ± 0.006	138.5 ± 3.0	0.33 ± 0.12	4.79 ± 0.39	179.8 ± 63.3	....	....
2	5	1.032 ± 0.004	138.6 ± 1.6	1.60 ± 1.8	4.28 ± 0.31	254.2 ± 31.3	....	....
3	5	1.035 ± 0.002	141.3 ± 4.0	0.00 ± 0.0	4.65 ± 0.45	273.4 ± 43.4	0.0000 ± 0.0000	43.93 ± 14.42
4	5	1.029 ± 0.002	138.8 ± 4.0	8.20 ± 10.7	4.50 ± 0.25	289.2 ± 63.7	....	....
5	6	1.017 ± 0.007	141.3 ± 6.9	20.50 ± 26.0	4.80 ± 0.24	260.7 ± 64.4	0.3478 ± 0.8390	57.18 ± 24.32
				– Adequate –				
6	6	1.025 ± 0.005	141.7 ± 1.7	152.6 ± 72.3	4.70 ± 0.39	185.4 ± 66.1	1.7796 ± 1.2086	54.29 ± 7.20
7	8	1.030 ± 0.003	139.4 ± 1.4	35.5 ± 37.9	4.63 ± 0.37	327.0 ± 38.2	0.3057 ± 0.3437	72.76 ± 15.81
8	6	1.030 ± 0.005	139.5 ± 1.7	42.1 ± 36.3	4.97 ± 0.15	301.9 ± 33.3	0.3819 ± 0.3216	79.56 ± 12.20
9	7	1.025 ± 0.005	138.6 ± 1.6	130.9 ± 59.8	4.44 ± 0.08	202.2 ± 53.4	1.3348 ± 0.7321	58.95 ± 6.64
10	10	1.104 ± 0.009	142.7 ± 1.3	44.6 ± 40.2	4.38 ± 0.35	115.3 ± 58.5	1.1245 ± 1.1590	73.84 ± 39.31

TABLE 3. Na and K Concentrations in Serum and Urine and Fractional Excretion in Salt Deficient and Control Lactating Cows.

Day	Urine Sp.G.	Serum Na mEq/L	Urine Na mEq/L	Serum K mEq/L	Urine K mEq/L	FE_{Na} %	FE_k %	Milk kg/cow
				– Deficient –				N=7
0*	1.026 ± 0.005	143.0 ± 1.9	43.4 ± 32.1	4.50 ± 0.38	165.9 ± 41.2	0.3290 ± 0.2911	39.47 ± 13.52	18.8 ± 7.2
15	1.028 ± 0.005	139.6 ± 1.4	12.4 ± 12.3	3.79 ± 0.29	192.8 ± 29.4	0.0945 ± 0.1192	50.34 ± 11.90	16.5 ± 11.6
29	1.025 ± 0.007	139.8 ± 2.0	1.8 ± 03.1	3.64 ± 0.33	179.5 ± 50.0	0.0156 ± 0.0301	50.78 ± 12.73	13.3 ± 11.5
				– Control –				N=7
0*	1.020 ± 0.009	141.4 ± 1.4	29.7 ± 32.4	4.43 ± 0.28	106.6 ± 53.0	0.3573 ± 0.2880	39.27 ± 11.49	25.1 ± 10.8
15	1.021 ± 0.009	140.6 ± 1.2	15.9 ± 13.7	4.53 ± 0.39	144.0 ± 56.9	0.1285 ± 0.0975	42.09 ± 16.07	24.6 ± 19.9
29	1.021 ± 0.009	142.5 ± 1.5	21.4 ± 20.3	4.61 ± 0.36	141.3 ± 49.1	0.2050 ± 0.2015	36.68 ± 11.18	22.7 ± 19.7

* = mean of 3 pre-experimental days (X ± SD)

TABLE 4. Na and K after 2 weeks of salt supplementation in herd 1.

		Serum Na+ mEq/L	Urine Na+ mEq/L	Serum K+ mEq/L	Urine K+ mEq/L
Before $\bar{x} \pm$ SD		138.5 ± 003.0	0.33 ± 0.12	4.79 ± 0.39	179.8 ± 063.3
n=9	range	134.0 — 143.5	0.20 — 0.50	4.20 — 5.30	70.5 — 251.5
After x ± SD		143.7 ± 001.3	6.05 ± 06.86	4.40 ± 0.22	274.90 ± 048.1
n=10	range	142.0 — 146.0	0.50 — 18.50	4.00 — 4.80	207.00 — 346.0

Field Studies

The first herd which prompted these investigations exhibited evidence of PU/PD and declining milk production (Herd No. 1, Table 1). Urinalysis revealed an extremely low urine Na concentration indicative of an Na deficiency (Table 2). In our subsequent investigations serum Na, urine Na, urine specific gravity and the FE_{Na} were utilized to assess Na status because of the ease of collection and analysis of serum and urine samples versus collection and analysis of saliva and/or feces. The FE_{Na} and urine specific gravity were assessed to determine if they would better assess Na status than the use of either serum or urine Na alone.

Table I is a summary of herd historical information 10 herds investigated. Table 2 is a summary of selected serum and urine laboratory data. The primary complaint of herds in the NaCl deficient group was either PU/PD or decreased production or both, both of which partially or totally resolved upon the resupplementation of NaCl in the diet. All of the Na deficient herds were supplementing < 42 grams of NaCl per cow per day.

The laboratory findings in the 10 private herds revealed very large differences in urine Na concentrations. In general, the Na deficient herds had extremely low urine Na concentrations in comparison to the Na adequate herds

Table 4 exhibits the clinical laboratory data on Herd No. 1 on the initial visit and 2 weeks after Na re-supplementation. After re-supplementation it was noted that serum Na, urine Na and urine K concentrations all increased by approximately 4 kg/cow/day 2 weeks after NaCl re-supplementation.

Herds No. 8 and 9 (Tables 1, 2) in the NaCl adequate group exhibited PU/PD as the primary complaint. Subsequent analysis of the forages fed revealed that the alfalfa hay from Herd No. 8 contained 2.94% K, while Herd No. 9, alfalfa hay and haylage, contained 3.06% and 3.6% K on a dry matter basis, respectively.

Discussion

Experimentally, Na deficiency resulted in a milk production decline. It can be expected that in higher producing cows, milk production would decline more rapidly and to a greater degree as is usually observed in feed mixing errors[h]. A possible reason for the difference in urine K concentrations between deficient and control groups (Figure 2) is lower milk production. The slightly higher specific gravity of urine may be attributable to increased K concentration and higher PCV values of the deficient group may both be related to milk production.

Many factors are involved in the control of Na balance. Excess dietary Na or renal tubular damage will cause an increase in FE_{Na} and FE_{Na} will decrease with stress or steroid administration. Experimentally, dietary Na deficiency may be detected utilizing urine Na Concentration and FE_{Na}. These tests could also be used in clinical practice to assess Na supplementation status. Like most laboratory tests, the result is only interpretable by someone acquainted with the animal patients. Animals studied on a herd basis are rarely treated with steroids, and if particularly stressed, it would be apparent. Blood sampling may be stressful, but the urine in the bladder is formed before sampling by venipuncture takes place. Therefore, a low mean FE_{Na} from 5 randomly selected cows in a herd would reflect dietary Na status; i.e., FE_{Na} = 0% would indicate a 0.0 Na concentration. Under the conditions of these experiments and visually comparing the data in Table 1, urine Na concentration was as reliable as FE_{Na} in confirming a diagnosis of Na deficiency.

The ultimate usefulness of these tests is to convince the dairyman or other persons involved that NaCl was deficient in the diet. Once accomplished, appropriate recommendations to meet requirements for NaCl will be followed. This was necessary in the first herd studied to

[h] Consultation with several veterinary practitioners and dairy nutritionists.

convince the owner to add the proper amount of NaCl. The follow-up visit after adequate resupplementation and resampling, revealed that production increased significantly in 2 weeks and that total body Na was normalized (8 of 10 animals had urine Na concentrations above 3 mEq/L, which is in agreement with previous studies that stated that whenever urine Na concentrations were below 3 mEq/L, additional slat supplementation will result in an increase in milk production[6,14]). In herd 5, the normal concentrations of Na found in the urine suggest that either the information about Na supplementation given by the owner was inaccurate, or that there are higher than normal Na concentrations in the water supply or in the feeds or forages fed.

The complaint of PU/PD is perhaps the most perplexing. Many field complaints of omitting a supplemental source of Na report decreased water consumption, urinary output and milk production, and drier feces. The finding of high urinary K concentration and normal urinary Na concentration in some of the PU/PD herds certainly implicate high dietary K as a possible cause of PU/PD.

There are several possible explanations for the PU/PD observed in the herds studied. Dehydration was not observe either as increased PCV or upon physical examination, and was not further assessed. Dehydration was not considered to be a factor in these studies. Excess dietary K may induce an osmotic diuresis. This mechanism responsible for this effect may be related to the effect of K on the adrenal gland and kidney. An increase in dietary K initially produces an increase in serum K and serum aldosterone.[32-38] Also, angiotension II receptor concentration increases in the aldosterone secreting zona granulosa cells.[36,37] In contrast to Na deficiency, where both rennin and angiotension II can be elevated, excess dietary intake of K either decreases or has no effect on rennin or angiotension II secretion.[34,36,38-40] In chronic excess K, intake serum K eventually returns to normal;[34] however, there is a sustained hypersecretion of aldersterone and an increase in the width of the zonma glomerulosa over time.[34,36,39] Excess K intake has a direct effect on the kidney by increasing both K and Na excretion.[40] The increase in cation excretion is due to decreased reabsorption of K and Na from proximal tubulear fluid.[41] A mechanism of accounting for PU/PD is the continued chronic loss of Na and undetected declines in serum Na, which could lead to medullary soluite gradient depletion "medullary washout," thereby reducing the concentrating ability of the nephron.[33] Water consumption increases to compensate for PU.

PU/PD in lactating cattle on a herd basis was associated with a diet high in K concentration. We speculate that the medullary gradient is washed out and that any of the previously mentioned mechanisms may account for that effect. PU/PD is a nuisance problem and if there are no concurrent defined disorders, it is not associated with any medical problems or production losses.

These studies support a hypothesis that in cattle, decreases in milk production and Na excretion occur while serum Na concentrations remain normal. Only when Na depletion is extreme for extended periods of time will Na concentrations decline. The use off urine Na determination to confirm Na deficiency is very effective and inexpensive to perform. The authors suggest testing five high producing cows in a suspect herd, since those cows would be at greatest risk when salt is borderline or deficient.

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