PHYSIOLOGICAL RESPONSES OF CATTLE TO CHANGES IN DIETARY LEVELS OF SODIUM AND POTASSIUM

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Sigurberg Ormar Thorlacius

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ABSTRACT

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by

SIGURBERG ORMAR THORLACIUS

Some aspects of sodium, potassium, calcium and magnesium metabolism of four eighteen-month-old Hereford steers were studied following changes in dietary intake of sodium and potassium. One month prior to and during the first eight days of the study each steer received 706.1 mEq daily of sodium and 1108.4 mEq daily of potassium. Sodium intake was then decreased to 21.6 mEq daily and potassium intake was increased to 4427.8 mEq daily. The study was continued for eight days immediately after the change and 16 days after the change a further eight day study was carried out. At the end of this latter period of study the steers again received the original level of sodium and potassium and the study was continued for a further eight days.

The change from the moderate intake of sodium and potassium to the high potassium low sodium intake caused a transitory increase in the weight of water in the reticulo-rumen. The change back to the moderate intake of sodium and potassium caused a transitory decrease in the weight of water in the reticulo-rumen. Weight of dry matter in the

reticulo-rumen did not appear to be altered by the change in sodium and potassium intake.

The high potassium low sodium intake caused an immediate and significant decrease in sodium concentration and increase in potassium concentration of the reticulo-rumen fluid. There was a significant decrease in sodium concentration and increase in potassium concentration of the mixed saliva but this did not occur until the steers had been receiving the high potassium low sodium intake for more than eight days. There was a rapid and complete reversal of the sodium and potassium concentration changes in the reticulo-rumen fluid and mixed saliva when the steers again received the moderate sodium and potassium intake.

The sums of sodium and potassium concentrations in the mixed saliva and in the reticulo-rumen fluid did not appear to be altered by changes in sodium and potassium intake.

Initially, after the change to the high potassium low sodium intake the steers were in negative sodium balance but this was more than compensated for by a decrease in the amount of sodium in the reticulo-rumen.

Initially, after the change to the high potassium low sodium intake a considerable amount of potassium was retained by the animals but 42% of this was retained in the reticulo-rumen.

The high potassium low sodium intake caused an immediate and significant decrease in the urinary calcium

and magnesium excretion. There was no significant change in fecal calcium excretion. Fecal magnesium excretion during the last eight days during which the steers received the high potassium low sodium intake was significantly increased but in general during the first eight days there was no increase in fecal magnesium excretion.

The high potassium low sodium intake caused an immediate and significant increase in water consumption and volume of urine excreted. Fecal water excretion and water balance were not significantly altered. Fecal dry matter excretion did not appear to be altered during the first eight days after the change to the high potassium low sodium intake, but was slightly but significantly lower during the last eight days. However, the latter was not consistently reversed when the steers again received the moderate intake of sodium and potassium.

Plasma levels of sodium, potassium, magnesium and calcium were not altered by the high potassium low sodium intake. The high potassium low sodium intake caused a consistent but non-significant increase in potassium concentration of muscle samples, but there was no consistent change in the sodium concentration of the muscle samples.

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INTRODUCTION

Normal metabolic processes take place in tissues only if the external environment of the cells is constant. This external environment has been termed the milieu interior by Claude Bernard. Its relative stability was termed homeostasis by Cannon. The simplest animals living today are small organisms whose cells are bathed in sea water. From the sea water they take their food and oxygen, and return to it their waste products. In more complex animals the cells are bathed in blood or some other extracellular fluid. Due to the need to maintain homeostasis many complex systems have developed to regulate the composition of body fluids. These regulatory processes include respiration, digestion and osmoregulation. Osmoregulation is performed not only by the excretory organs but also by the body surface and the gut lining, both of which may transport salt and water between the environment and the interior of the body. Excretory organs can play only a negative part in maintaining body fluid concentrations, conserving or excreting substances already present in the body. The activity of the gut, however, can play a positive as well as a negative role.

In the field of digestive physiology and nutrition

of the ruminant, most investigations are concerned with steady state conditions. However, the period of dietary change is worthy of investigation in its own right for two main reasons. The period of dietary change is often associated with metabolic disorders such as hypomagnesemia and bloat. Secondly, periods of dietary change allow the physiologist to observe how the homeostatic mechanisms cope with natural disturbances.

Several interesting observations have been made on the period of change from an indoor regimen to the grazing of spring pastures. Such pastures are generally low in sodium and high in potassium, particularly if they have been treated with potassium fertilizers. Following such a change in diet it has been observed that sodium ion concentration in the rumen fluid drops markedly with a concomitant increase in potassium ion concentration. Similar changes occur in saliva concentration but are generally more erratic and not necessarily concomitant with changes in the rumen fluid. It has not been established whether these changes in sodium and potassium concentration in the rumen fluid are associated with depletion of the total sodium of the body or are due to redistribution of sodium between the gut and extracellular fluid.

Hypomagnesemic tetany is a condition which occurs in cattle and sheep most frequently within the first week after the cattle or sheep are turned out to spring pastures. Numerous workers have attempted to show that hypomagnesemia is caused by the high potassium content of the grasses. These experiments have generally consisted of comparing plasma or serum magnesium levels in animals fed a high level of potassium with those found when the intake of potassium was not high. The results of these experiments have been contradictory. From *in vitro* studies there is some reason to believe that a high level of alkaline salts in the diet may cause a decrease in magnesium and calcium absorption from the gut. However, there is little evidence to show that this occurs *in vivo*.

Since "tetany prone pastures" are low in sodium content and since saliva sodium ion concentration has been found to be lowered in animals grazing these pastures, some attempts have recently been made to assess the role of aldosterone in the aetiology of hypomagnesemia. However, the results were inconclusive.

The change in diet from dry feed to spring pastures involves much more than the changes in sodium and potassium levels, e.g. the quantities and composition of proteins,

carbohydrates and lipids are probably considerably different in the two dietary regimens. The present experiment was conducted to determine the effects of marked changes in sodium and potassium levels in the ration, with other dietary factors held constant. The concentration of sodium and potassium in saliva, blood and rumen fluid were determined while steers were consuming a ration containing either moderate levels of sodium and potassium or low sodium high potassium levels, both in steady state condition and during the adaption period when the steers were switched from one treatment to the other. Balances for sodium, potassium, calcium and magnesium were also performed, as well as measurements of rumen volume and concentrations of calcium and magnesium in blood.

LITERATURE REVIEW

I

DISTRIBUTION AND BIOLOGICAL FUNCTION OF SODIUM, POTASSIUM, CALCIUM AND MAGNESIUM

The body contains approximately 0.2 per cent sodium. About one-quarter of this amount is localized in the skeleton and most of the remainder is found in the extracellular fluid where it represents approximately 90 per cent of all extracellular cations. Thus sodium is important in maintenance of acid base equilibrium and osmotic pressure. In addition, an important function of sodium is in maintenance of membrane potentials. Cellular membranes actively extrude sodium from the interior of the cells and this causes concentration gradients of sodium and potassium to develop across the cell membrane. These concentration gradients in turn cause membrane potentials that are necessary for nerve, muscle and other functions of the body (42, 57).

Sodium depletion initially causes a decrease in total osmotic pressure of the extracellular fluid. This causes an outward movement of water from the extracellular to intracellular space. The kidneys tend to excrete more water. The volume of the extracellular space is therefore decreased.

Blood changes include a fall in the plasma concentration of sodium and a rise in blood urea. There is a decrease in plasma volume leading to haemoconcentration. The blood pressure falls, which can, in severe cases, lead to circulatory failure (42, 57).

The effects of sodium loss have been studied by following the alterations in total body content, by balance techniques and correlating these with alterations in serum sodium levels. From time to time discrepancies have been revealed in such experiments which were greater than could be accounted for by errors in measurement. For example, Renwick et al (69) did not observe a drop in serum sodium level, even after thirty days of severely restricted sodium intake in man. It is now known that bone sodium content falls after loss of sodium from the body (62). Bone is the only tissue in which the sodium concentration is greater than in the extracellular fluid (62). In chronic experiments by Munro et al (62), rats were kept on a rice diet for some weeks. The total bone sodium content fell steadily on the restricted diet, but the serum sodium only declined slightly after several weeks.

POTASSIUM

Potassium is the major ion of the intracellular fluid

and it has more or less the same importance in these fluids as sodium in the extracellular fluid. In addition, potassium seems to be involved in carbohydrate metabolism, facilitating carbohydrate synthesis and translocation. This is evidenced by the fact that insulin and glucose administration cause shifts in potassium (58). Specifically, potassium has been found to be important in the following enzyme systems: phosphotransacetylase, acetyl CoA synthetase, pantothenate synthetase, pyruvate phosphokinase and myosin A T P ase (13).

In view of a certain constancy of K:N (3mEq/gN) in tissue there appears to be a relationship of potassium to protein metabolism (58).

The concentration of potassium in the extracellular fluids is low, but slight variations of this can change many body functions. Excess potassium can cause dilation of the heart, and when the extracellular concentration of potassium reaches approximately three times normal, the heart stops in diastole (42). Conversely, when the level of potassium falls to a low value in the extracellular fluids, transmission of nerve impulses becomes impaired and muscular paralysis may result. (42).

EXTRACELLULAR REGULATION OF MUSCLE COMPOSITION

Muscle electrolyte composition is usually expressed

in terms of fat free solids rather than concentration per litre of intracellular or extracellular fluid. Since the quantity of muscle solid per kilogram body weight is relatively constant and not affected by changes in extracellular volume, analysis of muscle expressed in these terms permits valid comparison of data obtained in various disturbances (19).

Metabolic acidosis of the extracellular fluid produced either by administration of HCl or NH_4Cl or loss of sodium in excess of chloride leads to an elevation of potassium content of muscle and lowering of intracellular sodium (21). There is probably a fall in concentration of undetermined anion in the serum (19). Cooke and Segar (19) suggest that in extracellular acidosis the rise in hydrogen ion concentration outside the cell decreases the gradient between cell and extracellular fluid. This results in a deceleration of the $H^+_{i_1}$ for $K^+_{e_2}$ exchange, thus reducing the magnitude of the extracellular acidosis. Deceleration of the H^+_i for K^+_e increases Na^+_i for K^+_e because of competition between these reactions. Since Nai for Ke is of greater magnitude than H^+_i for K^+_e, the net result is that more potassium is transported into the cell and high intracellular potassium results. Low intracellular sodium is the result of increased sodium transfer outward.

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[1] i = intracellular

[2] e = extracellular

Large loads of potassium usually lead to a decrease in extracellular bicarbonate concentration (19). The muscle potassium is high and the intracellular sodium of muscle is decreased (21). The mechanism by which this occurs as suggester by Cooke and Segar (18) is as follows. The concentration of extracellular potassium rises and there is increased exchange of both Na^+_i and H^+_i for K^+_e. As a result, K^+_i rises and Na^+_i falls. Due to increased transfer of hydrogen out of the cell, extracellular bicarbonate falls.

CALCIUM

More than 99 per cent of the total body calcium is contained in the skeleton and in the teeth. The remainder of the body calcium is predominantly extracellular. Although it makes up a very small part of the total body calcium, the calcium in the extracellular fluid is critical for (a) normal neuromuscular excitability, (b) capillary and cell membrane permeability, (c) normal muscle contraction, (d) normal transmission of nerve impulses, and (e) normal blood coagulation (76).

The quantity of calcium in the plasma is approximately 10 mg per cent, normally varying between 9 and 11 mg per cent. The calcium in the plasma is present in three forms: (1) approximately 50 per cent of the calcium is bound to plasma proteins and is consequently non-diffusible through the

capillary membrane; (2) approximately 5 per cent of the calcium is complexed with other substances in the blood in such a manner that it is not ionized although it is diffusible through the capillary membrane; and (3) the remaining 45 per cent of the calcium in the plasma is both diffusible through the capillary membrance and also ionized. It is this ionic calcium which is important in most of the functions of calcium in the body (42).

Calcium is found in the bones partly as tricalcium phosphate and partly as calcium carbonate, and it is believed that the formula for the major crystalline salt, known as hydroxyapatite, is the following: $3Ca_3(PO_4)_2.CaCO_3$ (42). The crystals apparently admit and absorb various ionic groups without change in the geometry of the crystal lattice. Hence bone contains, besides calcium and phosphorous, variable proportions of carbonate, fluorides, citrate, sodium, potassium and magnesium. The precipitation of insoluble calcium salts at the site of bone formation is favoured by secretion of alkaline phosphatase which converts organic esters of phosphoric acid into inorganic phosphate (76).

Bone mineral is readily mobilized to maintain the level of serum calcium. It is in the cancellous bone with its extensive surface exposed to interstitial fluid that

most of the bone turnover of calcium and phosphate takes place (76).

Changing the pH of extracellular fluids alters the deposition and absorption of bone salts, principally by changing the relative quantities of $HPO_4^=$ and $H_2PO_4^-$ in the fluids (42). Since it is the dibasic phosphate ions ($HPO_4^=$) that are important for deposition of bone salts and since these are decreased by acidosis, there is a tendency for the bone salts to be reabsorbed in acidosis. Conversely, in alkalosis there is a tendency for increased deposition of bone salts.

MAGNESIUM

Magnesium is the fourth most abundant cation present in the body, being surpassed only by sodium, potassium and calcium. In cattle the magnesium content of the body in relation to body weight is given by the following equation (Blaxter and McGill, 14):

$$Mg\ (gm) = 0.655\ \text{weight}\ (kg) - 3.5.$$

About 70 percent of the total body magnesium is in the skeleton. The magnesium of the skeleton acts as a labile source of magnesium (14).

Apart from bone, magnesium occurs principally intracellularly. The concentration of magnesium in cells is about

30 mEq/l compared with a plasma concentration of about 2.4 mEq/l (76).

Intracellular magnesium acts as an activator of numerous enzymes catalyzing reactions involving adenosine triphosphate. Since A T P is required in such diverse functions as muscle contraction, protein, fat, nucleic acid, and coenzyme synthesis, glucose synthesis and utilization, methyl group transfer, sulfate, acetate, and formate activation, and oxidative phosphorylation, it seems likely that the action of magnesium extends to all major anabolic and catabolic processes (57).

Plasma magnesium can be divided into ultrafiltrable and protein bound magnesium. Present evidence suggests that the ultrafiltrable magnesium is all ionic, unlike plasma calcium where there is a non-ionic ultrafiltrable fraction. (42).

Wilson (94) suggests that the concentration of magnesium or calcium in the interstitial fluid will be very close to the ultrafiltrable magnesium or calcium in plasma and that this will be the physiologically active concentration. Although it is generally agreed that about 50 per cent of plasma calcium is ionic, there is no such general agreement regarding plasma magnesium, and values between 20 and 50 per cent have been given as the fraction of bound

magnesium in plasma (94).

Del Castillo and Engback (26) showed that magnesium in the extracellular fluid plays an important role in the production and destruction of acetylcholine, the substance necessary for the transmission of impulses at the neuromuscular junction. A low concentration of magnesium and probably of more importance, a low magnesium to calcium ratio potentiates the release of acetylcholine. It has therefore been suggested that a low concentration of magnesium in the extracellular fluid surrounding the muscle and plates may produce tetany through this mechanism (76). It is known that the activity of choline esterase is not affected during magnesium deficiency (76).

II

RENAL CONTROL OF BODY WATER AND ELECTROLYTES

Cation Excretion

In a quantitative sense the filtration and reabsorption of ions and water are by far the most significant operations of the mammalian kidney (66). From four-fifths to seven-eighths of the glomerular filtrate is normally reabsorbed in the proximal tubule while the distal tubule reabsorbs one-fifth to one-eighth of the total cation excretion in a highly variable manner suited to the needs of the moment (66). All evidence (66, 92) points to the fact that sodium reabsorption in the proximal tubule is active and that the active reabsorption creates an osmotic force which causes reabsorption of water. The colloid osmotic force exerted by the plasma proteins and the crystalloid forces developed by reabsorption of glucose and amino acids also contribute, but their contribution is believed to be small in comparison with the osmotic force developed by the active transport of sodium. In the distal tubule, sodium reabsorption occurs in exchange for potassium or hydrogen secreted. There is also some evidence (86) that sodium reabsorption may occur in conjunction with chloride absorption in the distal tubule.

The reciprocity between potassium and hydrogen ion excretions has been interpreted as evidence of competition between the two ions for exchange with sodium (15). This phenomenon is most marked in potassium loading and depletion, but is not inclusive since both hydrogen and potassium excretion may increase simultaneously as during orthostasis in humans receiving sodium sulfate (15). Although it has been shown that potassium excretion parallels increases and decreases in plasma concentrations, it does not follow that the filtered potassium is responsible for observed changes in excretion; much evidence indicates that intracellular potassium concentrations of tubular cells are more likely responsible (86). Potassium excretion is augmented by increases of blood pH (15). Since it is well known (15) that migration of potassium into cells of the body increases markedly from acidosis to alkalosis, this has been interpreted as implying that potassium secretion is dependent upon intracellular potassium concentration in the tubular cells. Similarly (15), infusion of NH_4^+ salts can inhibit potassium secretion. That NH_4^+ can replace intracellular potassium has been shown in kidney slices and erythrocytes (15).

Anderson and Pickering (2) and Sellers *et al* (73) have shown that potassium loading causes an increase in sodium excretion in cows. The experiment by Anderson and

Pickering involved intravenous infusion of 1.0 NKCl at rates of 7.0 to 9.0 ml. per minute for up to four hours. Plasma potassium concentrations rose 1 to 2 mEq/l as a result of infusion, but progressive increase in plasma potassium did not occur. The animals showed some variation but in general the increase in plasma concentration occurred within two hours of the onset of infusion. At the same time the rate of excretion of potassium increased and within two hours of the onset of infusion approximated the rate of administration. Potassium clearances rose to exceed inulin clearance values, giving ratios of potassium clearance to inulin clearance ranging as high as 2.0 towards the end of the infusion period, thus clearly indicating potassium secretion. Urine flow increased 312 to 717 per cent over the pre-infusion rate reaching a maximum within one to two hours after the onset of infusion and then declining gradually. Sodium excretion increased two to fifty times the pre-infusion figure. Like the concomitant diuresis, this reached a peak one to two hours after infusion, then fell progressively but persisted above pre-infusion level during the four-hour infusion. No changes were noted in plasma sodium concentrations.

Anderson and Pickering (2) suggested that during potassium loading a greater suppression of hydrogen-ion excretion occurs than increase in potassium. This gives rise to a net reduction in sodium-ion reabsorption, with

consequent increased restriction of the tubular reabsorption of water. In the experiment conducted by Sellers et al (73), 0.5 g. of potassium chloride per lb. of body weight, in approximately 10 gallons of tap water was introduced into the terminal part of the esophagus over a period of approximately 45 to 50 minutes. A two-hour quantitative collection of urine was made. Plasma potassium showed a prompt rise in four of five animals given potassium chloride. Twenty-four hours after the initiation of the experiment plasma potassium levels had returned to the pre-experimental levels. Similarly, plasma sodium levels rose promptly and twenty-four hours later they had returned to control levels. Potassium excretion increased from 1.2 to 6.6 times the control level. The authors noted that the rise was not as great as they had expected. Absolute excretion of sodium showed an increase in all animals ranging from 3.7 to 1230.0 times the control value. In three of the animals the increase was comparable to that attained in an experiment in which sodium was administered and greater than would be expected from a "washing out" effect of water administration. Sellers et al (73) concluded that increase in urinary sodium was due to an increase in plasma sodium. They suggested that the increase in plasma sodium was due to an inward movement of potassium into the cells in exchange for cellular sodium. Thus it

appears that Sellers et al found similar results to those of Anderson et al (2) in regard to the effect of potassium on urinary sodium. However, their results were dissimilar in regard to plasma sodium which led to different suggestions with respect to the mechanism responsible for the effect of potassium on renal excretion of sodium.

It seems possible, however, that the failure of Anderson et al (2) to find an increase in plasma sodium may have been due to an increase in plasma volume resulting from the infusion. Also it is possible in the case of the experiment by Pickering et al that the increase in urine sodium was due to a "washing out" effect since this factor was not assessed by their experiment.

In an experiment by Sellers et al (73) in which sodium was administered to cows, there was a rise in plasma potassium but the increase in urinary potassium was not marked. They concluded that the bovine kidney may distinguish between these two cations. This is in agreement with the view (86) that potassium excretion is much more dependent on cellular concentration than on plasma concentration.

Miller (59, 60, 61) found that administration of potassium salts caused an increase in the excretion of sodium in swine and rats.

In studies on swine, Miller (59) found that high potas-

sium intake increased sodium excretion during the first twenty-four hours but then sodium excretion fell to levels less than were found prior to giving the potassium salt. Similarly, studies on rats (60, 61) showed that an increase in potassium intake increased urinary excretion of sodium. However, in the case of rats, the urinary sodium remained high during the four-day period of high potassium intake.

There appears to be little available information on the mechanism of calcium excretion. This in part is due to the complexity of the physical-chemical state of this substance in the plasma (78). The total calcium content of normal human plasma ranges from 9 to 11.5 mg. per cent (78). Approximately fifty per cent of the plasma calcium is bound to protein (76). This proteinate is poorly ionized and its dissociation is affected by many factors (protein and calcium concentration, pH, temperature, albumin-globulin ratio, etc.) (78). In addition, diffusible calcium complexes (carbonate, citrate, phosphate, tartrate) are poorly ionized (78). Approximately 1.5 mg of calcium per 100 ml of plasma are bound by citrate or other organic acids (78). Thus it appears unlikely that the excretion of calcium will be related in any simple manner to the total plasma calcium. The portion bound to protein is unavailable for filtration, as is perhaps some of the phosphate complex, and it is

possible that filtrable complexes formed with carbonate, citrate and phosphate are not handled by the tubules as is the calcium ion (78).

Williamson and Freeman (93) found that the filterable fraction of plasma calcium increases in metabolic acidosis, but tends to remain within normal limits in other acid-base disturbances associated with hypercaluria. The tubular reabsorption of calcium appears to be inversely related to blood pH, plasma bicarbonate content, and sodium excretion (93).

Shohl (75), in a review of mineral metabolism, stated that alkalosis in small laboratory animals causes a shift of calcium and phosphorous from the urine to feces. Dehydration and acidosis causes an increase in the excretion of mineral cations and this excess is found almost entirely in increased amounts in the urine. This view is supported by the work of Hart et al (46). They found that daily ingestion of 115 or 230 ml of 40 per cent hydrochloric acid increased the calcium excretion in the urine of dairy cows. The acid feeding caused a reduction in urinary pH from 9.8 to 6.8. Williamson and Freeman (93) found that infusion of sodium bicarbonate solution increased calcium excretion. They interpreted this as an effect of alkalization of the urine. Recent work by Walser (89) has shown, however, that the results found by Williamson and Freeman were probably due to sodium. Walser

(89) concluded that during conditions of saline diuresis in dogs, sodium excretion is the major determinant of calcium excretion. Furthermore, the renal clearance of free ionic calcium approximately equalled the renal clearance of sodium. Walser (89) suggested that the renal tubules attempt to maintain a constant ratio of sodium ion to calcium ion in the tubular fluid, and that calcium shares a common reabsorptive transport mechanism linked to sodium transport. Walser (89) emphasized that all his observations were confined to animals with urine flows exceeding 2.5 ml per minute. At lower urine flows, another major determinant of calcium excretion becomes important. This factor is the urinary concentration of complexing anions. At lower flow rates the urinary concentrations of complexing anions, e.g. citrate, phosphate and sulphate, may be high enough to cause considerable complexing of calcium. Under these conditions the free calcium ion concentration can no longer be considered equal to total urinary calcium. However, when urine flow rates are high and plasma concentration of the complexing ions are normal, the concentrations of the anions is too low to complex a significant amount of urinary calcium.

Walser (89) attempted to determine whether calcium excretion was more closely correlated with sodium plus potassium excretion, or with sodium excretion alone. The

rise in calcium clearance resulting from KCl infusion was smaller than would be anticipated from the rise in sodium clearance despite the excretion of three times as much potassium as sodium. Although Walser did not suggest this, it would appear from examination of the data presented that potassium appeared to decrease calcium excretion. In the absence of KCl infusion the mean ratio of sodium to calcium clearance was 1.29. In the presence of KCl infusion the mean ratio of sodium to calcium clearance was 3.5. It is not known, however, whether this trend was as marked in the other three experiments conducted.

Stop flow experiments (91) in which both sodium and calcium have been measured show a similar relation to that found by Walser. Sodium concentration changes coincide with changes in calcium concentration, and the ratio of the two ions is similar to the ratio which is found in normal plasma ultrafiltrate. That calcium excretion and clearance are directly related to sodium excretion has been confirmed in humans by Kleeman et al (53). These workers found that although urinary excretion of sodium and calcium varied considerably from subject to subject, it was apparent in each that these two parameters paralleled each other. Administration of 9-alpha fluorhydrocortisone caused a distinct reduction in sodium excretion (53) and an obvious

positive balance. Despite the positive sodium balance, calcium excretion decreased in both subjects. Definite hypercaluria was caused by the high salt ingestion in four of the six subjects studied. Kleeman (53) concluded that the proper interpretation of urinary excretion of calcium in any clinical or experimental investigation cannot be made without a knowledge of simultaneous excretion of sodium.

Walser (89) also examined the relationship between urine flow and calcium excretion. The correlation between these two variables was much lower ($r = 0.61$) than for sodium and calcium ($r = 0.95$). The relationship between urinary sodium and calcium excretion was found to be only slightly modified by urinary pH. Urinary pH measured in eighty-three of the 106 collection periods ranged from 5.4 to 7.8. Although the ratio of calcium clearance to sodium clearance appeared to be slightly higher in the presence of acid urine than in the presence of alkaline urine, the difference was not marked. In an experiment (89) on a sodium-depleted dog, hydrochloric acid (0.33 N) was infused at 5 ml per minute for 22 minutes, resulting in severe acidosis. Both sodium excretion and calcium excretion remained normal. Walser concluded that in acute experiments acidosis augments calcium excretion chiefly by virtue

of its effect on sodium excretion.

Sellers et al (73) found that absolute urinary calcium excretion increased as a result of KCl administration in cows. However, the increase in urinary calcium excretion was not greater than would be expected with the concomitant water diuresis (74). In both the case of water (74) and KCl (73) administration, urinary sodium excretion increased.

In view of these results it appears possible that the same relationship between sodium and calcium excretion exists in the kidney of the cow as has been found in the dog (89) and human (53) kidney. However, in the experiment in which KCl (73) was administered to cows, sodium excretion was greater than in the case of water diuresis (74). Assuming a direct relationship exists between urinary sodium and calcium excretion, one might expect that urinary calcium excretion should have been greater with KCl administration than with water diuresis since this was the pattern followed by sodium. However, this was not the case.

These facts considered alone tend to suggest that a direct relationship between calcium and sodium does not exist in the bovine kidney. It should be pointed out, however, that control values in both the KCl (73) and water diuresis (74) were carried out under non-diuretic conditions. The situation is thus complicated (91) by the fact that a

large proportion of the urinary calcium in the control experiments was probably not ionic. The explanation could also be that potassium suppresses urinary calcium excretion (91).

Little is known about renal excretion of magnesium for much the same reasons as for calcium. A considerable fraction of the plasma magnesium is known to be bound to plasma protein and magnesium excretion is influenced by complexing agents in the urine. Wilson (94), in 1960, reviewed existing knowledge of renal magnesium excretion. He concluded that the urinary output of magnesium can best be described by a filtration reabsorption mechanism, and that magnesium is a threshold substance and only appears in the urine if the filtered load is greater than the maximum tubular reabsorption rate. Experiments by Wilson (94) showed that in ewes with a filtration rate of 100 ml/minute, the threshold concentration in the plasma is 2 mg per cent. Rook et al (70) found a correlation between serum concentration and urinary output of magnesium in cows and estimated that the renal threshold in cows was not greater than 2.15 mg per cent. Wilson (94) pointed out that the amount of magnesium filtered and reabsorbed in twenty-four hours is more than ten times the total amount of magnesium in the extracellular field. Experiments with ^{24}Mg reviewed by Ulrich (86)

support the view that most of the magnesium enters the tubule at the glomerulus and not through the tubular cell.

Both Bradley (15) and Wilson (94) noted that a number of workers have shown that aglomerular teleost fishes actively excrete magnesium through their tubules. Jabir et al (50) and Barker (8) found that intravenous infusion of magnesium salts in humans decreased the urinary excretion rate of potassium. Wilson (194) found a similar effect in ewes. It has been suggested (94) that observed decreases in urinary excretion of potassium may be a result of a competition between magnesium and potassium for a transport mechanism. Sellers et al (74) found in conditions of water diuresis a constancy of absolute magnesium and potassium excretion despite approximately three to seven-fold changes in urine volume. They suggested that magnesium and potassium are handled in like manner by the bovine kidney. However, under conditions of high potassium intake (73) the absolute excretion of magnesium did not appear to be markedly different from the control values whereas the absolute excretion of potassium, as would be expected, was much higher than the control values.

Bicarbonate Excretion

Bicarbonate excretion appears to be largely a function of acid-base balance (14). The bulk of filtered bicar-

bonate is reabsorbed in the proximal tubule (66). The mechanism here involves an exchange of cellular hydrogen for filtered sodium (66). Hydrogen combines with bicarbonate in the urine to form carbon dioxide and water. The carbon dioxide diffuses into the cell, where it undergoes hydration to form carbonic acid, a reaction which is catalyzed by carbonic anhydrase. Subsequent dissociation provides the hydrogen ions, which are exchanged for sodium ions across the luminal membrane, and the bicarbonate ions, which diffuse down a potential gradient into the peritubular fluid.

Potassium and hydrogen competitively occupy in part a common transport mechanism, so that circumstances which favour potassium excretion reduce hydrogen excretion and vice versa (78). This is well illustrated in experiments with dogs (66). In these experiments it was found that when plasma concentrations of potassium fall below the normal value of 4 mEq/l, bicarbonate reabsorption is enhanced. Conversely, when potassium salts are infused and plasma potassium concentration exceeds 4 mEq/l, bicarbonate reabsorption is reduced below the normal value. Pitts (66) suggests the significant factor controlling bicarbonate reabsorption in these cases is not the plasma concentration of potassium but rather the intracellular concentration.

This is based on the fact that infused potassium rapidly enters all cells in exchange for hydrogen. The hydrogen ions leaving the cells are buffered by extracellular bicarbonate, thus decreasing plasma concentration of bicarbonate. The renal tubular cells would respond in a similar manner, accumulating potassium ions from, and extruding hydrogen ions into the peritubular fluid. As a result of reduced hydrogen ion concentration and increased potassium ion concentration of collecting duct cells, potassium ions displace hydrogen ions from the luminal exchange mechanism. Bicarbonate reabsorption would be reduced and potassium secretion enhanced. The urine contains large amounts of potassium bicarbonate and is therefore highly alkaline. In conclusion Pitts suggests that loading with potassium salts would lead to a hyperkalaemic metabolic acidosis. The acidosis is extracellular; the cell contents being relatively alkaline.

Bicarbonate reabsorption appears to be related to plasma chloride. In hypochloremia plasma bicarbonate increases. Conversely, in hyperchloremia, plasma bicarbonate decreases.(66)

Water Excretion

Renal water reabsorption is believed to occur by passive diffusion of water along an osmotic gradient created by active reabsorption of ions (66). In water diuresis when

the titer of circulating anti-diuretic hormone is low, the epithelium of the distal tubule is impermeable to water. The hypotonicity of the tubular urine leaving the loops of Henle is maintained throughout the remainder of the nephron and the final urine is dilute. Osmotic diuresis occurs due to reduced absorption of filtered solutes (24). This may be due to presenting the proximal tubules with material that cannot be absorbed (mannitol). High intakes of sodium chloride cause osmotic diuresis and this is believed to be due to diminished isosmotic absorption of filtrate in the proximal tubule, leading to an increased load on the distal tubule (24). Administration of potassium chloride to cows has been shown to cause osmotic diuresis (2, 73). Anderson et al (2) suggested that the osmotic diuresis was in part due to an increased solute load in the gomerular filtrate. They also suggested that during potassium loading a greater suppression of hydrogen ion excretion occurs than increase in potassium, giving rise to a net reduction in sodium ion reabsorption, with consequent increased restriction of the tubular reabsorption of water. Water diuresis has also been shown to occur in cattle (74).

Hormonal Regulation of Renal Electrolyte Excretion

The adrenal cortex is a well-recognized source of hormonal agents that exert a powerful influence upon renal tubular function. The most recently discovered of these, aldosterone, dominates the steroids released in terms of its effect upon electrolyte excretion (15) and is approximately thirty times more effective than deoxycorticosterone. Aldosterone appears to promote tubular sodium reabsorption and potassium secretion (15). There are two main hypotheses regarding the control of aldosterone secretion (86). The first states that peripheral nervous receptors, possibly located in the upper arterial tree, serve as the afferent limb of a reflex arc whose effector is a neurohormone or a pineal hormone. The second hypothesis states that a peripheral receptor affects the release of renin from the juxtaglomerular apparatus in the kidney. Renin leads to the liberation of angiotensin which in turn stimulates aldosterone secretion. Available evidence appears to favour the latter of these two hypotheses (86). Increased aldosterone secretion has been shown to be caused by potassium administration as well as sodium depletion (86).

Adrenal cortical hormones appear to affect bicarbonate reabsorption, although the mechanism is not entirely

clear. Hyper secretion of gluco- and mineral corticoids induces metabolic alkalosis associated with increased bicarbonate reabsorption. In contrast, a deficiency of adrenal cortical hormones induces metabolic acidosis associated with reduced bicarbonate reabsorption (65). There have been some attempts in recent years to assess the role of adrenal cortical hormones in renal excretion of calcium and magnesium. Care et al (17) found that when normal sheep were injected intramuscularly with DOCA, urinary, excretion of calcium increased but magnesium excretion remained unchanged. In adrenalectonized sheep the same workers (17) found that neither DOCA nor D-aldosterone-trimethyl acetate had a significant effect on renal excretion of magnesium. Similarly, Scott and Dobson found that D-aldosterone injected intramuscularly (66) had no effect on urine excretion of magnesium but in contrast to Care and Ross they also found no effect on the urinary excretion of calcium. Scott and Dobson (71) found, however, that the injection procedure, i.e. intramuscular injection of an isotonic solution of saline alone, caused a significant increase in urinary excretion of magnesium. Scott and Dobson (71) found the intravenous infusion of aldosterone appeared to cause an increase in urinary magnesium excretion although sufficient data were not collected for statistical analysis. In sodium-depleted sheep where

aldosterone secretion would be expected to increase, Dobson and Scott (71) found a decrease in urinary excretion of calcium and magnesium. They suggested that this may have been due to a reduction of the extracellular compartment due to depletion of sodium.

III

REGULATION OF THE VOLUME AND MINERAL COMPOSITION OF RUMEN FLUID

The volume of rumen fluid is normally maintained by the consumption of water and salts and the secretion of saliva. Consumption of water and salts together with absorptive mechanisms counterbalance the effects of microbial metabolism so as to maintain pH, solute concentration and osmotic pressure of the rumen fluid within the range favourable to normal rumen function.

Water Intake

Water intake of cattle depends on a large number of factors but only those related to mineral intake will be discussed here. Beilharz and Kay (11) reported that the osmotic pressure of the blood appears to be important. They found that sheep became very thirsty when they were given intravenous injections of hypertonic sodium chloride solutions. However, Beilharz and Kay (11) also found that intravenous injections of hypertonic glucose did not have the same effect as sodium chloride and they concluded that the sheep's osmoreceptors respond selectively to solutes which do not penetrate the cell membrane readily. The osmotic pressure of the rumen contents does not appear to be involved since

Beilharz and Kay (11) found that sheep showed no sign of thirst when hypertonic sodium bicarbonate solutions were introduced into the rumen. That low blood osmotic pressure does not fully inhibit thirst was shown by Beilharz et al (10) who found that sheep depleted of sodium by loss of saliva continue to drink water although the concentration of sodium in the plasma is falling.

Salivary Secretion

Ruminants secrete large volumes of alkaline well buffered saliva which stabilizes the rumen pH and supplies most of the water and salts of the rumen fluid. Bailey (5) has shown that cattle secrete as much as 98 to 190 litres of saliva daily and sheep (5) have been estimated to secrete 6 to 16 litres daily.

Saliva is made up of secretions of a number of glands. Kay (51) divided the salivary glands of sheep into three groups. First, the parotid and inferior molar glands which are purely serous. They secrete continuously and may be stimulated from the mouth, the oesophagus and the rumen. These glands are particularly active during eating and rumination. The saliva secretion is isotonic with plasma, alkaline and well buffered. Kay (51) placed the small glands lying beneath the epithelium of the cheeks, palate and pharynx in the second group. They are largely composed of

mucous cells and secrete little when not stimulated. Like the serous glands they respond well to stimulation of the mouth, oesophagus and rumen. Although the palatine saliva is extremely mucous it does resemble parotid saliva in its inorganic constituents and alkalinity. The third group, which is both serous and mucous, consists of the submaxillary, sublingual and labial glands. The submaxillary is little affected by stimulation of the oesophagus and rumen. It secretes rapidly during feeding but only slightly during rumination. The saliva of the third group is mucous, hypotonic and poorly buffered.

Parotid saliva from normal sheep contains on the average the following: (mEq/l) sodium, 177; potassium, 8; bicarbonate, 104; monohydrogen phosphate, 52; chloride, 17; and less than one mEq/l each of calcium and magnesium (52). Mixed saliva in sheep is similar in composition, according to Kay (51). In cattle it has been found that mixed and parotid saliva are also similar in composition (5, 6, 31).

The most important factor that affects the composition of parotid saliva is sodium depletion. The remarkable inversion of the relative concentrations of sodium and potassium in parotid saliva when sheep are depleted of sodium was first described by Denton (27). Denton found that when sodium supplement was withdrawn from a sheep with

a parotid fistula that the sodium ion concentration of the saliva decreased from 180 to 60 mEq/l. There was an almost equivalent increase in potassium ion concentration; it rose from 10 to 120 mEq/l. Thus the saliva appeared to retain its hypertonicity. Apart from a small increase of monohydrogen phosphate with an equivalent decrease in bicarbonate, the anion pattern was unchanged. There was a gradual decrease in the saliva volume. The average volume per day during the control period was 2.68 litres whereas the average over the eleven days of sodium depletion was 1.71 litres. The rise in monohydrogen phosphate was commensurate with the decrease of secretion volume so that the total phosphate secreted was unchanged. Within forty-eight hours of sodium withdrawal, the sodium excretion by the kidney was reduced to virtually nil. At the same time, the renal potassium excretion was greatly reduced. Denton concluded that in the face of gross sodium depletion, the animal had ceased excreting sodium in the urine, greatly reduced it in the saliva, and transferred the large urinary potassium excretion characteristic of herbivora to the parotid secretion. Dobson (33) showed that the concentration changes of sodium and potassium ions of mixed saliva of sheep during sodium depletion followed those of the parotid saliva to a highly significant degree, but were not as marked. The potassium concentration of mixed

saliva was slightly more effective than its sodium concentration as a basis for predicting the potassium concentration of parotid saliva. Bailey and Balch (5, 6) and Dobson (31) have shown that the changes that occur in the concentration of sodium and potassium of both parotid and mixed saliva of cattle during sodium depletion are similar to those found in sheep. Dobson (31) has suggested that mixed saliva of cattle obtained by sponge sampling may be more closely related to parotid saliva than is the case for sheep.

A number of workers have shown that there is a relationship between the rate of secretion of parotid saliva and its composition. Coats and Wright (18) varied the rate of secretion in sheep by electrically stimulating the motor nerve at various frequencies. They found that as the rate of saliva secretion increased, there occured a rise in sodium concentration and a fall in potassium concentration. Similarly, Bailey and Balch (5) found in a small steer that the concentration of sodium increased and potassium decreased as the rate of parotid saliva secretion increased. This appeared to be the case whether or not sodium intake was adequate. Dobson (31) found similar results with respect to sodium and potassium concentration in the parotid saliva of a cow receiving an inadequate intake of sodium. However, when the cow was receiving an adequate intake of sodium,

Dobson (30) found that potassium concentration rose slightly with an increase in flow rate with a corresponding fall in sodium concentration. Dobson (31) suggested that the apparent discrepancy between his work and that of Bailey and Balch could have arisen from differences between animals or from small differences in sodium status.

Osmotic Pressure and Electrolyte Concentrations in the Rumen Fluid

The work of Parthasarathy and Phillipson (64) suggests that the rumen fluid tends towards isotonicity with the blood. They found that solutions which were hypotonic or isotonic to serum lost water when put in the isolated rumen of anaesthetized sheep while hypertonic solutions gained water. The movement of water with hypertonic and hypotonic solutions indicated that water moved across the rumen epithelium as a result of osmotic gradients. The absorption of water from the isotonic solution was probably due to net uptake of ions. Kay (52) suggests that three mechanisms may account for the near isotonicity of rumen contents: the permeability of the rumen epithelium to water, the absorption of salts by the rumen epithelium, and the secretion of large volumes of nearly isotonic saliva.

If ruman contents are to remain isotonic any solutes absorbed must be accompanied by an appropriate volume of

water. Kay (52) has pointed out that Hyden's estimate of the rate of absorption of water from the rumen contents of a sheep, 3.6 litres daily, is enough to render roughly isotonic the 600 mEq of sodium (plus anion) that Dobson(29) estimated is absorbed from the rumen. Kay (52) suggested that volatile fatty acids and other fermentation products which are continuously found in the rumen and absorbed from it would be expected to have little long term effect on the osmotic pressure and water balance of the rumen; intermittent surges of fermentation may have more substantial effects. Fatty acids will only increase the number of osmotically active particles in solution to the extent that they are not neutralized or are neutralized by monohydrogen phosphate to give dihydrogen phosphate plus acid radical since neutralization by bicarbonate is accompanied by the evolution of gaseous carbon dioxide.

Elam (36) found that rate of passage of digesta through the rumen is accelerated by including large amounts of sodium chloride in the diet, and Lloyd Davies (55) found that the addition of four or eight litres of water to the rumen daily caused only very slight increases in food intake. Similarly, Murray et al (63) found that provision of a salt lick during an experiment when a sheep was feeding continuously led to an increased rumen outflow. However, they also found that

regular addition of three grams of sodium chloride to each meal when the animal was fed twice daily gave no increase in the daily rumen outflow over that of a control period with no salt addition, but there was a slight decrease in the weight of the rumen contents (63). If the isotonicity of the rumen contents is mainly due to the permeability of the rumen epithelium to water, then the rate at which rumen fluid passes on to the omasum will depend more on the amount of salts that enter the rumen than on the water intake. The experiments reported above suggest that this is the case.

The concentration of individual electrolytes in the rumen fluid depend largely on the amounts of salts and water that are ingested, the amounts absorbed by the rumen epithelium and the amounts secreted in the saliva (52). Experiments by Sellers et al (72) and Dobson et al (33) in sheep, and by Bailey (4) in cattle have shown that sodium and potassium account for most of the cations present in the rumen fluid and their concentrations show a strong inverse correlation. The principal anions are chloride, monohydrogen phosphate, bicarbonate and the anions of volatile fatty acids (4, 85).

The relationship between the concentrations of sodium, potassium, chloride and inorganic phosphate in mixed saliva and rumen fluid of cattle was studied by Bailey (4). The

studies were carried out on four dry shorthorn cows, given five different diets supplying from four to sixteen grams of sodium daily. The sodium concentration in the rumen fluid ranged from 160 to 60 mEq/l, and in the saliva from 168 to 28 mEq/l. The observations in which sodium concentration in rumen fluid exceeded 145 mEq/l were mainly on cows receiving sodium chloride ad lib. The concentration of sodium in the rumen fluid was parallel to, but lower than, the concentration in saliva. The mean value for rumen fluid was 17 per cent lower than the mean value of saliva for all pairs of observations. The consistency of the relationship between the concentration of sodium in the rumen fluid and saliva was high. Bailey suggested that when food was eaten, and salivary flow increased, absorption of sodium and the voluntary water intake during the meal was sufficient to prevent any rise in sodium concentration in the rumen. The results showed that at any one time the concentration of sodium in the saliva was clearly the dominant factor determining the concentration found in the rumen fluid. The concentration of sodium in the saliva and in the rumen fluid tended to be lower when the cows were consuming the diets containing the lower amounts of sodium. Bailey suggested that over a long period with a sodium-deficient diet the amount of sodium supplied could have an effect on sodium concentration in rumen fluid owing

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to a progressive depletion of body sodium and a consequent decline in salivary sodium concentration. There was considerable difference in the sodium concentration in saliva and rumen fluid between cows consuming the same diet. Bailey suggested that this probably reflected the varying length of time the cows had been consuming the diets.

Both chloride and potassium concentration in rumen fluid were highest shortly after eating, and declined steadily thereafter to low values fourteen hours after eating. Like sodium, the concentration of potassium in rumen fluid reflected differences in the amounts of potassium supplied by the saliva. However, the concentrations of potassium in rumen fluid were invariably higher than the concentrations in saliva, which suggested that dietary potassium was a more important fraction of the potassium in the rumen than was the dietary sodium of the sodium in the rumen. The concentration of chloride in the rumen fluid was invariably greater than the concentration in the saliva. This fact as well as the uniformity in the concentration of chloride in the rumen fluid in different cows consuming a given diet confirmed that the dietary chloride was important in establishing these concentrations.

Sellers and Dobson (72), and Dobson and McDonald (34), have studied the changes which occur in the composition of

saliva of sheep feeding on grass grown on fields heavily fertilized with potassium salts. These grasses are characteristically high in potassium content and low in sodium. Sellers and Dobson (72) found that when two sheep accustomed to hay or hay and meals were fed cut grass heavily fertilized with potassium chloride and ammonium sulphate, sodium concentrations in the rumen fluid fell and potassium rose. Concentration of about 25 mEq/l for sodium and 100 mEq/l for potassium were reached after a week, a reversal of the concentrations found when sheep were on hay diets. Changes in concentration of sodium and potassium in the mixed saliva were much more erratic. However, the lower ratios of sodium to potassium tended to be associated with the grass diets, and Sellers and Dobson concluded that the changes in the sodium and potassium concentration in the rumen fluid were due at least in part to changes in concentrations of these ions in the saliva. Electrical potentials between plasma and rumen contents were also measured by Sellers and Dobson (72). They found that the potential was correlated negatively with the sodium concentration and positively with the potassium concentration in the rumen. The effects of the two ions could not be separated because of the close negative correlation between sodium and potassium concentrations. Sellors and Dobson concluded, however, that although changes in concen-

tration of sodium and potassium in the rumen account for part of the changes in potential encountered, other unidentified factors must play a role, especially in the grass regimen.

Dobson and McDonald (34) studied changes in the rumen fluid and saliva concentration of sodium and potassium when eight sheep accustomed to a diet of hay and meals were fed a diet of grass heavily fertilized with potassium chloride and ammonium sulphate. In general they found lowered concentration of sodium and increased concentration of potassium in the saliva and rumen fluids, thus confirming earlier observations of Sellers and Dobson (72). The change in saliva sodium and potassium concentration varied greatly between animals in both extent and timing. However, Sellers and Dobson (72) observed changes in salivary potassium during the first week at grass, whereas Dobson and McDonald (34) observed no change in salivary potassium during the first week at grass. Dobson and McDonald (34) suggested that the difference was due to the fact that in the previous work the sheep consumed less sodium. Dobson and McDonald (34) found that the fall in sodium and rise in potassium concentration in the rumen fluid were nearly completed by the end of the first week at grass. They thus concluded that changes in rumen concentration do not necessarily imply changes in saliva compo-

sition as had been implied earlier by Sellers and Dobson (72). Dobson and McDonald (34) suggested that the changes in rumen concentration of sodium and potassium may have been due to changes in rate of flow into or out of the rumen or in absorption of water and sodium, together with the increased intake of potassium. Dobson (32) studied changes in rumen and saliva sodium and potassium when mature ewes which had previously been receiving a steady intake of grass cubes were subjected to three dietary changes. The first change was to fresh frozen grass, then onto hay and meals and finally back to the frozen grass. The dietary intake of sodium was about 30 mEq per day on the grass cubes, 10 mEq per day on frozen grass and 55 mEq per day on the hay and meals. The saliva potassium concentration was low on the diets of grass cubes and grass diets but was high when the ration was changed to hay and meals. The high level of potassium in saliva was maintained though it showed some signs of falling just before the final dietary change. Dobson suggested that since in other experiments a low concentration of saliva potassium was found with a diet of hay and meals, the salivary response was associated with the dietary change rather than with the diet itself. The change in sodium intake from grass cubes to frozen grass was accompanied by an approximately equivalent fall in the amount of sodium excreted in the urine. In contrast, as soon as feed-

ing of the hay and meals ration was begun, sodium almost disappeared from the urine. This was followed by an enormous increase in urinary sodium during the first few days of the second period on frozen grass. The urinary output of potassium rapidly and consistently reflected these changes in intake. The loss of sodium on change from hay and meals to frozen grass was accompanied by a loss of water. The loss of water was shown to occur by changes in both live weight and apparent water balance. Dobson suggested that this probably reflected a decrease in rumen volume. Sodium concentration in the rumen fluid decreased from 90 mEq/l to 55 mEq/l. Dobson concluded that the sodium which appeared in the urine thus appeared to originate from the rumen contents.

The change from hay and meals to frozen grass was marked by a retention of water and an increase in the concentration of sodium in the rumen from 55 mEq to 90 mEq/l. The changes of potassium concentration in the rumen fluid were roughly equivalent to those of sodium but in the opposite direction.

Dobson suggested that the raised concentration of potassium in mixed saliva together with the reabsorption of sodium from the urine which followed the change to hay and meals indicated an increase in the level of aldosterone in

the blood. Dobson also suggested that since the stimulus to the adrenal gland accompanied an increase in the amount of sodium in the ration, any explanation in terms of a sodium depletion of the whole animal may be ruled out. Dobson hypothesized that the change to hay and meals gave rise to an immediate retention of sodium in the rumen which could happen only by depleting the extracellular space outside the gut, thereby stimulating the secretion of aldosterone. Denton (27) and Kay (52) have suggested that the rumen acts as a store for sodium which can be drawn upon at times when dietary intake is low. This suggestion appears to be based mainly on the observed decrease in saliva sodium concentrations which occurs when sheep are depleted of sodium. Dobson pointed out that in the change from hay and meals to frozen grass a large fraction of the sodium in the rumen was rapidly transferred to the urine at a time when the dietary intake of sodium was reduced. Dobson therefore concluded that in this case the sodium in rumen contents was by no means secured for the use of the animal.

An electrical potential gradient exists across the rumen epithelium of a sheep (35). The blood has been shown to be approximately 30 mv positive with respect to the rumen contents (35). This fact has been observed in both conscious sheep with normal rumen contents and in anaesthetized sheep

with artificial solution in the rumen. In considering absorption of electrolytes from the rumen contents one must thus consider both the electrical and the chemical gradient existing. Dobson (29) showed that sodium movement takes place against its electrochemical gradient and therefore the absorption of sodium is active. Dobson (29) concluded that about one-half the salivary sodium of a sheep is absorbed in the reticulo-rumen and that therefore the absorption of sodium from the reticulo-rumen is of considerable importance to the sodium economy of the animal. Dobson (29) also suggested that since water moves passively down its osmotic gradient, the sodium uptake takes place without greatly affecting the concentration of sodium, as was found by Sperber and Hyden (78). Dobson (29) also pointed out that since the sodium pump will tend to make the rumen contents electrically negative to the plasma, this will decrease the forces driving potassium into the plasma. This, Dobson (29) suggested, may be of importance to ruminants whose diet is rich in potassium and poor in sodium, whereas their plasma has high sodium and low potassium concentration.

Little is known about the absorption of potassium from the rumen contents (29). The epithelium contains a high concentration of potassium and this hampers the in-

vestigation of potassium movements in acute experiments, since changes in a solution in the rumen may arise in part from changes in amounts in the epithelium (30). Sperber and Hyden (79) observed that potassium accumulated in a rumen pouch to a concentration of 27 mEq per litre. Dobson (30) pointed out that this could be accounted for by an electrical potential which made the blood 45 mv positive to rumen contents. A potential of this size and sign is well within the observed range

He (87) calculated that about 64 g of sodium per day were absorbed from the large intestine of the cows under experiment. Van Weerden (87) pointed out that, since a cow of 500 kg,

44. Hansard, S. L., C. L. Comar and M. D. Plumlee. 1952. Absorption and tissue distribution of radiocalcium in cattle. J. Animal Sci. 11:524.

45. Hansard, S. L., C. L. Comar and M. P. Plumlee. 1954. The effects of age upon calcium utilization and maintenance requirements in the bovine. J. Animal Sci. 13:25.

46. Hart, E. B., H. S. Steenbock, O. L. Kline and G. C. Humphrey. 1931. Dietary factors influencing calcium assimilation. XIV. The influence of mineral acids and sugar on the calcium metabolism of milking cows. J. Dairy Sci. 14:307.

47. Head, M. J. and J. A. F. Rook. 1955. Hypomagnesemia in dairy cattle and its possible relationship to ruminal ammonia production. Nature. 176:262.

48. Horrocks, D. and G. D. Phillips. 1964. The concentration of certain mineral elements in the alimentary tracts of European and Zebu steers. J. Agric. Sci. 63:359.

49. Instruction Manual for Hitachi Perkin-Elmer Flame Photometry Attachment (139-0400) for Model 139 UV-VIS Spectrophotometer. 1963. Hitachi Perkin-Elmer.

50. Jabir, F. K., S. O. Roberts and R. A. Womersly, 1957. Studies on magnesium metabolism. Clin. Sci. 16: 119.

51. Kay, R. N. B. 1960. The rate of flow and composition of various salivary secretions in sheep and calves. J. Physiol. 150:515.

52. Kay, R. N. B. 1963. Reviews of the progress of dairy science. Section A. Physiology. Part 1. The physiology of the rumen. J. Dairy. Res. 30:261.

53. Kleenan, C. R., J. Bohannan, D. Bernstein, S. Ling and M. H. Maxwell. 1964. Effect of variations in sodium intake on calcium excretion in normal humans. Proc. Soc. Exptl. Biol. Med. 115:29.

54. Kunkel, H. O., K. H. Burns and B. J. Camp. 1953. A study of sheep fed high levels of potassium bicarbonate with particular reference to induced hypomagnesemia. J. Animal Sci. 12:451.

55. Lloyd-Davies, H. 1962. Intake studies in sheep involving high fluid intakes. Proc. Aust. Soc. Animal Prod. 4:167.

56. Masson, K. J. and A. T. Phillipson. 1952. The composition of the digesta leaving the abomasum of sheep. J. Physiol. 116:98.

57. Maynard, L. A. and J. K. Loosli. 1956. Animal Nutrition, 4th Edition. McGraw-Hill Book Co. Inc., Toronto.

58. Meier, H. 1963. Fluids and Electrolytes. Clinical Biochemistry of Domestic Animals. C. E. Cornelius and J. J. Kaneko. Academic Press, New York.

59. Miller, H. G. 1923. Potassium in animal nutrition. I. Influence of potassium on urinary sodium and chlorine excretion. J. Biol. Chem. 55:45.

60. Miller, H. G. 1926. Potassium in animal nutrition. III. Influence of potassium on total excretion of sodium, chlorine, calcium and phosphorus. J. Biol. Chem. 67:71.

61. Miller, H. G. 1926. Potassium in animal nutrition. V. Influence of potassium on urinary and fecal excretion of sodium, chlorine, calcium and phosphorus. J. Biol. Chem. 70:593.

62. Munro, D. S., R. S. Sataskar and G. M. Wilson. 1957. The exchange of bone sodium with isotopes in rats. J. Physiol. 139:474.

63. Murray, G. M., R. S. Reid and T. M. Sutherland. 1962. The rate of passage of digesta from the reticulorumen of the sheep. J. Physiol. 164:262.

64. Parathasarathy, O. and A. T. Phillipson. 1953. The movement of potassium, sodium, chloride and water across the rumen epithelium of sheep. J. Physiol. 121:452.

65. Phillips, G. D. 1965. Personal Communication. University of Manitoba, Winnipeg, Manitoba.

66. Pitts, R. F. 1963. Physiology of the Kidney and Body Fluids. Year Book Publishers, Inc., U.S.A.

67. Poutsiaka, J. W., B. G. H. Thomas and C. R. Linegar. 1957. Effect of fluorohydrocortisone on gastro-intestinal and renal excretion of cations by the dog. Proc. Soc. Exptl. Biol. Med. 96(3):712.

68. Renkema, J. A., T. Senshu, B. D. Gaillard and E. Brower. 1962. Regulation of sodium excretion and retention by the intestine in cows. Nature. 195: 389.

69. Renwick, R., J. S. Robson and C. P. Stewart. 1955. Observations upon the withdrawal of Nacl from the diet in hypersensitive and normotensive individuals. J. Clin. Invest. 34:1037.

70. Rook, J. A. F., and C. C. Balch. 1958. Magnesium metabolism in the dairy cow. I. Metabolism on stall rations. J. Agric. Sci. 51:189.

71. Scott, O. and A. Dobson. 1965. Aldosterone and the metabolism of magnesium and other minerals in the sheep. Quart. J. Exptl. Physiol. 50:42.

72. Sellers, A. F. and A. Dobson. 1960. Studies on reticulo-rumen sodium and potassium concentration and electrical potentials in sheep. Res. Vet. Sci. 1:95.

73. Sellers, A. F., T. L. Gitis and M. H. Roepke. 1951. Studies of electrolytes in body fluids of dairy cattle. III. Effects of potassium on electrolyte levels in body fluids in mid-lactation. Am. J. Vet. Res. 12:296.

74. Sellers, A. F. and M. H. Roepke. 1951. Studies of electrolytes in body fluids of dairy cattle. I. Effects of water diuresis on renal excretion of several elements. Am. J. Vet. Res. 12:183.

75. Shohl, A. T. 1939. Mineral Metabolism. Monogr. Ser. Amer. Chem. Soc. No. 82. Reinhold Publishing Co., New York.

76. Simensen, M. G. 1963. Calcium, inorganic phosphorus, and magnesium in health and disease. Clinical Biochemistry of Domestic Animals, C. E. Cornelius and J. J. Kaneko. Academic Press, New York.

77. Sjollema, B. and L. Seekles. 1932. The magnesium content of the blood, particularly in tetany. Klin. Wachshr. 11:989.

78. Smith, H. W. 1956. Principles of Renal Physiology. Oxford University Press, New York.

79. Sperber, I. and S. Hyden. 1952. Transport of chloride through the ruminal mucosa. Nature. 169:587.

80. Steel, R. G. O. and J. H. Torrie. 1960. Principles and Procedures of Statistics. McGraw-Hill Book Co. Inc., Toronto.

81. St. Omer, V. V. E. 1965. The role of potassium in bovine nutrition. M. Sc. Thesis, University of Manitoba, Winnipeg, Manitoba.

82. Storry, J. E. 1961. Studies on calcium and magnesium in the alimentary tract of sheep. I. The distribution of calcium and magnesium in the contents taken from various parts of the alimentary tract. J. Agric. Sci. 57:97.

83. Storry, J. E. 1961. Studies on calcium and magnesium in the alimentary tract of sheep. II. The effect of reducing the acidity of abomasal digesta *in vitro* on the distribution of calcium and magnesium. J. Agric. Sc. 57:103.

84. Talapatra, S. K., S. C. Ray and K. C. Sen. 1948. Calcium assimilation in ruminants on oxalate-rich diet. J. Agric. Sci. 38:163.

85. Turner, A. W. and E. V. Hodgetts. 1955. Buffer systems in the rumen of the sheep. II. Buffering properties in relationship to composition. Australian J. Agric. Sci. 6:115.

86. Ulrich, K. J. and D. J. Marsh. 1963. Kidney, water and electrolyte metabolism. Annual Review of Physiology. 25:91.

87. Van Weerden, E. J. 1961. The osmotic pressure and the concentration of some solutes of the intestinal contents and faeces of the cow, in relation to the absorption of minerals. J. Agric. Sci. 56:317.

88. Visek, W. J., R. A. Munroe, E. W. Swanson and C. L. Comar. 1953. Calcium metabolism in dairy cows as studied with Ca^{45}. J. Dairy Sci. 36:373.

89. Walser, M. 1962. Calcium clearance as a function of sodium clearance in the dog. Amer. J. Physiol. 200:1099.

90. Walser, M. 1960. Determination of free magnesium ions in body fluids. Anal. Chem. 32:711.

91. Wesson, L. G. and D. P. Lauler. 1959. Nephron reabsorption site for calcium and magnesium in the dog. 101:235.

92. Wiendhager, E. E. and Giebish. 1961. Micropuncture study of renal tubular transfer of sodium chloride in the rat. Amer. J. Physiol. 200:581.

93. Williamson, B. J. and S. Freeman. 1957. cited in--Kidney. Annual Review of Physiology. S. E. Bradley. 19:513.

94. Wilson, A. A. 1960. Magnesium homeostasis and hypomagnesemia in ruminants. Veterinary Reviews and Annotations. 6:1.

APPENDIX

TABLE 1

SODIUM CONCENTRATION OF THE MIXED SALIVA

(mEq per litre)

	Steer A	Steer B	Steer C	Steer D
Period I				
Day 1	142.0	122.5	139.0	130.3
2	122.0	139.0	140.0	150.0
3	105.0	139.0	130.0	150.0
4	145.3	150.0	152.5	163.5
5	138.0	139.0	155.0	144.5
6	145.0	139.0	158.0	158.7
7	141.7	139.0	142.0	167.0
8	138.0	171.5	142.0	167.0
Period II				
Day 1	139.0	141.0	138.0	152.5
2	145.0	139.0	125.0	150.0
3	113.0	122.0	150.0	160.0
4	122.0	122.0	145.0	150.0
5	125.0	92.5	141.5	120.0
6	141.5	92.5	141.5	133.0
7	128.5	98.8	136.5	133.0
8	125.0	71.3	145.0	124.0
Period III				
Day 1	96.5	55.0	105.5	90.0
2	94.5	37.5	111.5	101.5
3	100.0	45.0	117.5	126.5
4	90.0	35.5	114.5	101.5
5	112.5	40.0	105.0	102.5
6	81.5	28.0	101.5	101.0
7	77.5	43.0	96.5	92.0
8	88.0	35.5	105.0	101.0
Period IV				
Day 1	87.5	42.5	96.0	75.5
2	67.5	55.5	104.0	110.0
3	94.0	87.5	110.0	126.0
4	123.5	142.5	126.0	132.5
5	117.5	132.5	152.5	145.0
6	135.5	105.0	145.0	150.0
7	123.5	101.5	155.0	175.0
8	123.5	-----	154.0	150.0

TABLE 2

POTASSIUM CONCENTRATION OF THE MIXED SALIVA

(mEq per litre)

	Steer A	Steer B	Steer C	Steer D
Period I				
Day 1	12.0	11.0	9.2	13.2
2	12.0	10.7	10.0	15.8
3	11.0	10.0	18.3	14.5
4	10.0	10.0	11.7	11.7
5	11.0	15.7	15.0	11.7
6	13.0	13.0	13.2	15.0
7	11.0	13.0	15.0	13.2
8	12.0	12.0	11.7	11.7
Period II				
Day 1	13.2	13.2	16.7	16.0
2	13.2	23.3	23.3	18.5
3	16.7	26.0	20.9	15.0
4	18.3	32.3	18.8	22.0
5	12.5	50.0	16.5	26.0
6	15.6	50.0	18.8	30.0
7	13.5	50.0	25.0	28.5
8	20.9	58.8	14.6	27.5
Period III				
Day 1	40.0	101.5	61.5	82.5
2	45.5	104.0	45.0	73.0
3	58.0	105.5	45.0	52.5
4	50.5	107.0	60.0	77.5
5	52.5	107.5	71.0	50.0
6	42.5	119.5	50.0	65.0
7	44.5	107.0	66.0	60.0
8	50.5	107.5	60.0	65.0
Period IV				
Day 1	62.5	90.0	55.0	80.0
2	45.5	88.0	50.0	50.0
3	20.0	55.0	32.5	35.0
4	16.0	37.0	20.0	37.5
5	16.0	25.5	14.0	20.0
6	15.0	31.0	14.0	10.5
7	12.5	42.5	15.5	10.0
8	20.5	42.5	----	14.0

TABLE 3

SODIUM CONCENTRATION OF THE RETICULO-RUMEN FLUID

(mEq per litre)

	Steer A	Steer B	Steer C	Steer D
Period I				
Day 1	128.9	125.0	125.0	130.0
2	125.0	128.9	125.0	128.9
3	126.3	122.5	125.0	130.0
4	126.3	125.0	125.0	127.5
5	126.3	131.3	126.3	130.0
6	128.9	132.5	126.3	126.3
7	125.0	135.0	122.5	130.0
8	125.0	135.0	128.9	130.0
Period II				
Day 1	111.3	110.0	111.3	100.0
2	85.0	79.0	102.5	94.0
3	81.5	86.0	88.0	86.0
4	78.5	79.0	88.0	85.0
5	78.5	-----	85.0	85.0
6	80.0	68.8	88.0	75.0
7	80.0	65.0	86.0	71.0
8	75.0	63.8	83.5	67.0
9	71.0	55.0	83.5	70.0
Period III				
Day 1	80.0	21.3	71.3	72.0
2	80.0	21.3	----	58.5
3	30.0	21.3	63.8	57.0
4	65.0	22.5	75.0	60.5
5	68.8	20.0	71.3	55.0
6	69.0	22.5	70.0	59.0
7	60.0	21.3	58.8	59.0
8	55.0	22.5	65.7	59.0
Period IV				
Day 1	77.0	45.0	65.0	58.5
2	100.0	65.0	87.0	64.0
3	102.0	----	110.0	96.5
4	107.0	87.5	125.0	112.5
5	127.5	87.5	133.0	116.0
6	127.5	107.0	140.0	131.0
7	137.5	103.5	137.5	137.5
8	127.5	90.0	137.5	131.0

TABLE 4

POTASSIUM CONCENTRATION OF THE RETICULO-RUMEN FLUID

(mEq per litre)

	Steer A	Steer B	Steer C	Steer D
Period I				
Day 1	30.0	27.5	34.5	32.0
2	30.5	33.0	33.0	30.5
3	29.5	25.0	29.5	30.5
4	32.0	29.5	27.5	30.0
5	30.0	30.0	26.3	31.3
6	32.5	32.0	30.0	30.5
7	35.0	31.3	32.0	32.5
8	33.0	32.0	31.3	32.5
Period II				
Day 1	55.5	45.0	60.0	65.0
2	80.0	56.0	67.5	70.0
3	77.5	68.0	72.5	73.0
4	79.0	79.0	80.0	75.0
5	85.5	----	72.5	85.0
6	----	85.0	69.5	84.0
7	87.0	87.5	73.8	84.0
8	84.0	91.3	68.5	90.0
9	83.0	91.3	81.0	93.0
Period III				
Day 1	86.3	128.8	96.3	103.0
2	81.3	125.0	----	103.0
3	131.3	131.3	85.0	95.0
4	85.0	131.3	95.0	101.0
5	85.0	128.8	96.3	101.0
6	106.0	131.3	91.3	100.5
7	100.0	131.3	91.3	110.5
8	95.0	118.8	87.5	111.0
Period IV				
Day 1	68.0	106.0	75.0	110.0
2	58.0	100.0	70.0	71.5
3	53.5	----	58.0	69.0
4	40.0	70.0	43.0	55.0
5	42.5	64.5	39.0	33.5
6	33.5	52.5	30.5	31.5
7	37.0	56.0	27.5	29.0
8	32.0	63.0	27.5	30.5

TABLE 5

URINARY SODIUM

(mEq/day)

	Steer A	Steer B	Steer C	Steer D
Period I				
Day 1	250.1	(450.0)	(429.0)	398.8
2	385.0	358.1	282.1	526.1
3	332.1	480.7	313.8	447.0
4	258.8	312.8	370.5	468.8
5	391.3	210.0	447.0	466.1
6	248.6	258.8	279.5	349.7
7	390.0	360.0	532.7	543.9
8	-----	475.0	733.0	-----
Period II				
Day 1	264.1	190.2	457.7	205.3
2	374.5	15.3	54.4	143.2
3	34.6	20.5	65.1	49.4
4	12.8	20.0	25.4	61.3
5	24.2	25.3	40.1	36.1
6	19.0	11.7	15.1	21.0
7	6.8	26.8	22.9	48.2
8	10.3	17.2	16.1	55.1
Period III				
Day 1	5.1	17.0	(6.0)	4.4
2	3.5	(9.8)	6.5	3.2
3	5.4	21.3	21.5	(11.2)
4	7.2	27.3	16.7	5.3
5	5.3	18.9	11.5	4.9
6	4.7	7.4	10.3	5.6
7	5.9	7.3	29.4	15.4
8	9.1	4.7	6.4	9.1
Period IV				
Day 1	3.5	13.7	4.1	4.1
2	2.5	12.1	12.1	4.1
3	2.2	17.2	6.4	2.3
4	2.6	5.9	42.2	4.1
5	16.5	116.0	87.5	6.1
6	48.8	364.0	204.0	5.8
7	240.0	255.0	280.5	54.4
8	533.0	168.0	663.0	8.8

TABLE 6

FECAL SODIUM

(mEq/day)

	Steer A	Steer B	Steer C	Steer D
Period I				
Day 1	439.7	(127.5)	(95.3)	145.2
2	397.5	101.8	109.9	116.3
3	268.6	278.4	97.5	314.2
4	358.1	279.3	95.4	120.4
5	273.8	259.1	116.1	85.3
6	341.4	278.0	101.2	57.1
7	378.5	254.3	181.0	92.0
8	(422.6)	369.5	143.0	-----
Period II				
Day 1	266.0	251.4	170.4	89.6
2	366.7	30.6	80.2	25.4
3	252.3	12.6	56.7	18.8
4	14.7	31.9	52.4	15.0
5	48.2	7.7	97.5	9.6
6	48.2	10.3	15.5	5.8
7	32.7	13.0	24.8	9.2
8	27.0	6.7	16.5	7.0
Period III				
Day 1	26.1	7.7	26.8	4.4
2	22.7	(8.7)	33.9	9.2
3	29.6	12.2	22.3	9.0
4	19.2	4.7	30.7	11.0
5	16.1	6.7	24.7	7.5
6	13.6	5.0	23.2	9.2
7	(12.6)	2.8	16.1	11.6
8	20.0	12.0	14.5	10.5
Period IV				
Day 1	15.5	13.7	27.1	10.8
2	25.4	12.1	21.9	11.6
3	20.4	17.2	24.9	12.6
4	20.2	5.9	28.5	11.5
5	87.3	116.0	67.6	12.0
6	98.7	364.0	116.0	22.9
7	311.9	255.0	179.5	58.0
8	320.6	168.0	368.2	70.7

TABLE 7

URINARY POTASSIUM

(mEq/day)

	Steer A	Steer B	Steer C	Steer D
Period I				
Day 1	703.1	964.8	830.0	725.0
2	751.9	940.5	869.5	722.8
3	770.0	1063.8	846.6	726.4
4	842.5	(977.5)	877.5	515.6
5	1092.8	930.0	675.8	899.7
6	1017.0	1012.6	785.0	565.5
7	1080.0	816.0	854.3	768.8
8	-----	1094.4	(874.3)	-----
Period II				
Day 1	2690.4	2808.0	2174.4	2649.6
2	3877.5	3488.0	3915.0	3412.8
3	3552.0	3387.3	2288.0	3627.1
4	3162.0	3273.5	3968.6	3645.6
5	3883.4	3913.8	4171.5	3687.2
6	4056.0	3978.0	4152.5	3995.0
7	3902.0	3952.5	4351.0	3280.5
8	4053.8	3841.5	3672.0	4357.5
Period III				
Day 1	3567.0	3248.0	(3450.0)	3645.0
2	(3543.8)	(3931.3)	3964.8	3570.0
3	3378.4	3740.0	3686.5	3897.9
4	3720.2	4173.1	3605.0	3838.4
5	3718.8	3831.3	4275.0	4075.0
6	3965.0	3822.0	3613.8	3545.4
7	4018.0	3810.3	3796.3	3756.1
8	3736.6	4284.0	3471.5	(3615.4)
Period IV				
Day 1	1914.0	1832.0	1037.5	1246.9
2	1248.0	1074.7	1014.3	868.8
3	1274.0	1230.0	1120.0	1200.0
4	1152.0	757.9	843.8	1076.1
5	1004.9	1421.0	968.8	998.4
6	1088.8	650.0	798.0	1082.8
7	1155.0	462.0	726.8	580.0
8	------	430.5	682.5	606.0

TABLE 8

FECAL POTASSIUM

(mEq/day)

	Steer A	Steer B	Steer C	Steer D
Period I				
Day 1	405.8	202.9	162.5	360.5
2	368.5	353.4	274.9	392.1
3	249.4	167.2	383.0	262.3
4	129.1	(179.5)	310.7	258.3
5	148.4	139.1	168.8	189.9
6	164.8	228.0	195.1	165.7
7	153.8	128.0	242.9	176.0
8	(111.8)	172.4	(192.9)	-----
Period II				
Day 1	87.7	241.9	748.8	327.1
2	112.1	176.4	583.6	276.0
3	160.7	224.1	372.3	274.5
4	181.4	231.4	368.9	197.2
5	204.6	253.8	158.7	220.5
6	176.7	247.3	256.8	118.5
7	242.7	271.1	179.4	148.0
8	346.8	257.5	186.5	141.4
Period III				
Day 1	632.4	283.5	(669.2)	382.2
2	(585.4)	(396.1)	706.0	335.8
3	587.6	435.7	556.0	316.0
4	537.7	360.7	537.9	321.3
5	461.7	393.4	431.9	258.0
6	372.7	268.2	466.0	262.4
7	302.4	386.6	491.6	345.1
8	238.1	526.8	301.1	264.6
Period IV				
Day 1	320.6	311.3	640.5	310.4
2	326.8	433.5	607.3	227.7
3	200.2	286.2	444.7	295.9
4	132.1	184.3	370.1	225.8
5	137.9	93.8	402.8	164.6
6	127.0	76.6	435.7	240.8
7	123.9	193.4	427.7	324.3
8	96.2	118.1	464.5	215.5

TABLE 9

URINARY CALCIUM

(mEq/day)

	Steer A	Steer B	Steer C	Steer D
Period I				
Day 1	111.3	89.1	26.0	74.8
2	135.8	59.2	31.0	107.9
3	119.9	56.1	42.3	81.6
4	115.9	(51.7)	36.2	54.1
5	94.1	45.8	(33.4)	42.2
6	35.3	49.1	36.6	25.2
7	30.0	37.8	41.9	20.7
8	----	25.4	31.6	----
Period II				
Day 1	3.8	2.2	12.0	1.4
2	2.9	1.6	1.0	1.3
3	1.8	1.7	nil	1.3
4	2.0	1.6	2.1	1.2
5	1.2	2.1	2.2	1.5
6	0.7	1.7	2.5	1.3
7	1.3	2.6	1.9	1.0
8	1.8	2.0	1.8	0.9
Period III				
Day 1	(2.0)	(1.3)	1.8	0.9
2	1.1	2.0	1.2	1.2
3	1.4	1.0	0.5	1.6
4	1.8	1.1	1.1	1.3
5	2.3	1.7	1.5	(0.7)
6	1.8	1.8	1.2	0.8
7	2.0	2.9	(1.4)	1.1
8	2.5	3.4	0.9	0.7
Period IV				
Day 1	5.6	24.4	33.8	6.6
2	46.0	37.0	47.6	10.7
3	66.8	64.9	37.7	61.1
4	104.8	59.6	54.7	85.2
5	112.1	120.1	70.2	101.1
6	117.3	101.5	77.5	90.1
7	90.0	93.1	89.9	49.4
8	55.7	36.6	77.8	48.7

TABLE 10

FECAL CALCIUM

(mEq/day)

	Steer A	Steer B	Steer C	Steer D
Period I				
Day 1	433.8	366.2	248.2	299.6
2	315.8	311.5	280.3	294.0
3	387.4	291.8	304.3	429.8
4	310.5	(326.8)	283.0	234.1
5	313.1	298.1	(294.1)	248.6
6	376.4	395.5	323.2	216.6
7	329.4	247.3	328.0	251.2
8	(318.6)	315.5	270.9	-----
Period II				
Day 1	253.8	386.5	304.3	261.3
2	291.0	212.3	299.4	200.7
3	334.3	247.7	223.3	232.8
4	237.5	335.0	345.4	188.4
5	327.0	268.1	306.3	247.0
6	323.1	244.1	287.2	176.7
7	342.4	285.9	266.0	206.4
8	373.3	211.3	266.0	248.7
Period III				
Day 1	(444.0)	315.3	346.7	246.4
2	391.1	318.3	391.7	238.1
3	376.8	362.4	328.7	251.6
4	353.4	293.9	283.9	261.3
5	305.5	316.8	283.9	(219.3)
6	174.2	259.4	323.5	265.2
7	276.7	229.4	(367.6)	238.1
8	449.8	263.3	355.4	170.7
Period IV				
Day 1	437.4	285.3	421.3	238.3
2	450.5	340.3	426.6	304.7
3	330.7	283.9	372.3	299.1
4	290.3	223.4	358.5	303.0
5	314.8	258.6	402.3	308.5
6	293.0	281.5	376.3	320.4
7	329.7	442.8	432.5	321.5
8	296.5	181.8	512.6	265.7

TABLE 11

URINARY MAGNESIUM

(mEq/day)

	Steer A	Steer B	Steer C	Steer D
Period I				
Day 1	275.2	189.0	231.0	174.9
2	278.2	(155.8)	271.8	190.9
3	243.7	166.9	(323.3)	157.3
4	206.8	166.7	244.6	119.7
5	171.5	135.9	211.0	180.6
6	111.8	176.5	132.8	122.3
7	124.6	124.0	210.0	125.7
8	-----	192.0	263.0	-----
Period II				
Day 1	85.5	73.9	161.4	77.3
2	107.9	69.1	nil	0.1
3	139.7	87.1	nil	6.2
4	135.1	64.7	95.1	49.7
5	155.1	59.5	nil	120.0
6	125.3	nil	96.5	91.9
7	141.0	65.1	108.7	79.4
8	148.5	69.3	77.9	138.5
Period III				
Day 1	116.5	95.6	134.5	112.4
2	138.5	59.7	(144.2)	92.5
3	167.0	(86.5)	124.5	(99.0)
4	(183.4)	104.2	137.8	110.6
5	139.6	102.2	128.2	113.4
6	144.4	89.7	99.3	114.2
7	177.4	91.3	117.6	107.9
8	167.5	103.8	106.2	126.4
Period IV				
Day 1	193.8	134.8	200.2	126.4
2	279.0	115.6	176.9	105.4
3	290.4	147.9	152.6	211.2
4	284.9	136.9	198.5	224.8
5	306.7	296.3	189.4	287.5
6	271.2	246.5	234.8	292.2
7	249.4	179.8	295.0	116.7
8	153.5	64.3	151.3	110.6

TABLE 12

FECAL MAGNESIUM

(mEq/day)

	Steer A	Steer B	Steer C	Steer D
Period I				
Day 1	386.5	510.7	374.0	483.2
2	342.4	(327.3)	392.2	431.8
3	446.2	422.6	(520.8)	678.2
4	369.0	503.1	415.9	379.2
5	397.5	452.2	400.2	444.9
6	538.4	584.6	432.8	412.7
7	444.4	423.5	457.7	432.9
8	(519.5)	391.6	377.1	-----
Period II				
Day 1	352.6	542.4	409.9	527.3
2	477.9	327.6	542.0	439.4
3	503.0	383.0	404.6	470.8
4	413.3	475.2	648.0	370.5
5	475.7	504.7	454.1	473.8
6	405.0	476.7	461.8	386.3
7	440.4	575.9	545.3	455.2
8	528.3	423.8	573.3	495.9
Period III				
Day 1	583.2	617.1	601.3	540.3
2	466.4	579.3	(677.0)	589.3
3	488.0	(628.9)	506.9	622.0
4	629.9	604.4	519.1	669.2
5	600.5	563.4	649.6	578.6
6	292.4	526.7	673.7	665.8
7	418.9	705.6	684.1	598.6
8	717.2	458.6	440.6	417.7
Period IV				
Day 1	661.6	577.0	670.5	625.7
2	531.9	566.5	603.7	733.8
3	411.8	481.0	603.9	625.1
4	333.8	357.6	454.1	504.8
5	391.9	372.1	489.1	465.0
6	397.6	351.5	452.8	452.2
7	494.2	484.0	501.3	470.1
8	377.5	257.1	428.2	344.3

TABLE 13

WATER CONSUMPTION

(l/day)

	Steer A	Steer B	Steer C	Steer D
Period I				
Day 1	14.1	11.7	13.4	13.3
2	9.6	10.7	13.0	12.8
3	10.8	12.0	12.4	8.1
4	11.1	----	14.4	----
5	14.5	10.5	12.1	15.8
6	9.3	13.2	12.7	7.7
7	----	----	13.8	10.4
8	11.5	12.7	----	10.4
Period II				
Day 1	15.1	14.1	21.8	18.4
2	21.0	21.2	15.2	17.0
3	15.9	19.0	----	18.3
4	20.3	20.1	20.4	18.3
5	15.3	15.5	21.8	18.9
6	15.6	15.8	19.0	16.8
7	19.1	19.2	----	15.9
8	16.7	18.0	17.8	15.7
Period III				
Day 1	15.5	16.5	16.2	17.0
2	17.8	(16.4)	14.5	14.2
3	----	16.3	(14.6)	14.7
4	18.9	16.5	16.7	14.7
5	19.4	15.1	17.4	17.4
6	14.5	18.0	16.1	(16.6)
7	15.1	16.5	16.8	17.3
8	20.9	15.0	17.1	12.4
Period IV				
Day 1	12.9	13.0	12.2	10.3
2	9.6	11.9	11.4	11.1
3	16.3	10.1	12.4	----
4	11.3	11.2	12.2	15.0
5	12.9	12.8	13.6	----
6	9.6	11.8	----	12.4
7	11.4	17.4	10.4	17.9
8	----	----	15.3	----

TABLE 14

URINE VOLUME

(l/day)

	Steer A	Steer B	Steer C	Steer D
Period I				
Day 1	3.75	3.60	4.40	2.90
2	4.01	3.45	3.70	3.45
3	3.85	3.94	4.15	2.98
4	4.06	3.40	3.90	2.50
5	7.05	3.00	3.52	3.39
6	4.52	3.45	3.14	2.60
7	4.80	3.00	4.02	3.00
8	3.85	3.80	5.38	----
Period II				
Day 1	7.60	6.80	6.55	6.90
2	10.75	7.72	7.70	7.90
3	8.60	7.63	7.70	7.30
4	9.20	8.42	11.72	7.40
5	9.75	9.10	11.10	7.80
6	9.40	9.20	10.00	8.40
7	10.76	9.20	10.45	7.10
8	9.81	8.85	8.60	9.50
Period III				
Day 1	7.75	7.00	9.00	7.36
2	9.30	9.86	10.20	7.40
3	9.45	9.20	9.30	8.15
4	9.00	10.60	10.40	8.62
5	9.40	8.81	8.80	9.00
6	8.80	8.90	9.33	8.33
7	9.10	9.40	9.10	8.95
8	11.25	9.10	9.60	7.95
Period IV				
Day 1	7.70	8.16	4.00	4.25
2	5.24	3.83	3.83	2.62
3	5.60	3.92	4.12	3.80
4	5.40	2.35	3.50	3.73
5	5.60	4.80	4.00	3.90
6	5.50	5.50	3.80	3.95
7	5.00	5.00	4.10	2.20
8	4.20	3.00	4.20	2.50

TABLE 15

FECAL WATER

(kg/day)

	Steer A	Steer B	Steer C	Steer D
Period I				
Day 1	6.51	4.40	4.40	5.72
2	5.62	4.92	3.70	4.93
3	4.31	4.50	4.15	6.00
4	4.88	4.76	3.90	4.51
5	3.94	4.32	3.52	4.57
6	4.62	5.46	3.14	4.23
7	4.30	3.80	4.02	4.46
8	4.33	5.01	5.38	----
Period II				
Day 1	3.56	5.20	7.34	5.82
2	4.22	3.11	6.31	4.71
3	5.13	3.92	4.25	5.12
4	5.18	5.39	6.94	4.38
5	4.35	3.93	4.84	3.98
6	3.93	4.00	4.37	3.46
7	4.26	4.60	4.99	3.87
8	5.01	3.82	4.43	4.03
Period III				
Day 1	5.97	4.74	6.58	4.84
2	5.72	4.58	6.76	4.79
3	4.95	5.08	5.47	4.78
4	5.81	4.79	5.05	5.61
5	4.88	4.03	5.21	4.37
6	4.29	5.39	6.17	5.05
7	4.63	3.57	6.27	5.74
8	6.67	4.27	4.20	3.12
Period IV				
Day 1	4.62	5.24	8.13	6.93
2	4.88	6.07	5.28	3.60
3	4.08	4.74	6.20	4.90
4	3.47	3.64	5.93	4.08
5	3.78	2.46	5.70	4.18
6	3.71	1.81	6.68	4.68
7	4.73	3.28	7.52	5.61
8	3.90	3.04	7.64	1.40

TABLE 16

FECAL DRY MATTER

(kg/day)

	Steer A	Steer B	Steer C	Steer D
Period I				
Day 1	1.79	1.60	1.61	1.98
2	1.58	1.58	1.75	1.67
3	1.39	1.45	1.76	2.05
4	1.72	1.64	1.73	1.44
5	1.46	1.38	1.65	1.58
6	1.83	1.84	1.79	1.47
7	1.77	1.30	1.95	1.49
8	1.67	1.64	1.72	----
Period II				
Day 1	1.44	1.80	1.86	1.78
2	1.68	1.19	1.89	1.49
3	1.97	1.38	1.35	1.63
4	1.77	1.81	2.21	1.52
5	1.60	1.52	1.66	1.37
6	1.72	1.45	1.58	1.19
7	1.84	1.53	1.81	1.33
8	1.79	1.18	1.72	1.47
Period III				
Day 1	2.29	1.36	1.92	1.34
2	1.67	1.37	2.04	1.31
3	1.58	1.63	1.63	1.32
4	1.35	1.36	1.73	1.49
5	1.52	1.27	1.60	1.23
6	1.52	1.61	1.93	1.45
7	1.11	1.08	1.88	1.56
8	1.63	2.33	1.30	1.23
Period IV				
Day 1	1.73	1.36	1.77	1.27
2	1.67	1.88	1.62	1.40
3	1.52	1.61	1.80	1.55
4	1.43	1.26	1.87	1.42
5	1.52	0.74	2.00	1.47
6	1.29	0.59	1.82	1.42
7	1.67	1.17	1.78	1.59
8	1.54	0.96	2.06	1.30

TABLE 17

MEAN SQUARES OF MEASUREMENTS MADE DURING PERIOD I AND PERIOD III

Source of Variation	Treatment		Animals		Interaction		Error	Error Degrees of Freedom
Degrees of Freedom	1		3		3			
Sodium Conc. of Reticulo-Rumen Fl.	88,381.8	**	1,558.7	*	2,095.4	**	462.3	55
Potassium Conc. of Reticulo-Rumen Fl.	87,500.1	**	970.4	**	1,158.1	**	48.2	55
Sodium Conc. of Saliva	54,785.3	**	7,163.8	**	3,326.7	**	125.7	56
Potassium Conc. of Saliva	522,808.2	**	27,315.2	**	27,807.8	**	379.8	56
Sum of Sodium plus Potassium Conn.of Reticulo-Rumen Fl.	20.0		133.4	*	147.5	*	36.8	55
Sum of Saliva Sodium plus Potassium Conc.	28.6		1,839.1	*	155.1		126.7	56
Urinary Sodium	1,992,642.5	**	15,007.0	**	81,704.0	**	1,698.3	48
Fecal Sodium	563,706.1	**	42,604.5	**	42,300	**	2,318.9	48
Urinary Potassium	119,957,657.0	**	74,368		34,198		40,992	48
Fecal Potassium	454,554.5	**	22,352.1		24,516.4		9,864.3	48
Urinary Calcium	46,535.1	**	2,018.6	**	1,944.2	**	403.9	48
Fecal Calcium	3,582.4		18,680.1	**	4,948		2,935.4	48
Total Calcium Excretion	75,103.2	**	27,435.4	**	10,985.5		4,025.5	48

* ($P < 0.05$)
** ($P < 0.01$)

TABLE 18

MEAN SQUARES OF MEASUREMENTS MADE DURING PERIOD I AND PERIOD III

Source of Variation	Treatment	Animals	Interaction	Error	Error Degrees of Freedom
Degrees of Freedom	1	3	3		
Urinary Magnesium	62,548.5 **	8,985.3 **	2,605.2	1,186.9	48
Fecal Magnesium	222,093.0 **	11,985.7	6,738.8	7,544.0	48
Total Magnesium	49,474.4 **	2,667.7	475.1	7,883.2	48
Water Consumption	276.4 **	5.7	3.9	3.3	48
Urine Volume	445.99 **	5.40 **	0.47	0.58	55
Fecal Water	0.17	3.34 **	0.39	0.52	55
Fecal Dry Matter	0.164*	0.184**	0.076	0.057	55
Water Balance	7.1	1.6	9.6	2.9	46

* ($P < 0.05$)
** ($P < 0.01$)

TABLE 19

MEAN SQUARES FOR

COMPARISON OF MUSCLE AND PLASMA ELECTROLYTE CONCENTRATIONS

ON THE LAST DAY OF PERIOD I WITH THOSE

ON THE LAST DAY OF PERIOD III

Sources of Variation	Treatment	Animals	Error
Degrees of Freedom	1	3	3
Plasma Sodium	6.3	5.5	7.1
Plasma Potassium	0.07	0.14	0.16
Plasma Calcium	0.003	0.354 *	0.017
Plasma Magnesium	0.033	0.369 **	0.001
Plasma Chloride	325.2 *	66.4	29.1
Muscle Sodium	3.28	3.61	2.88
Muscle Potassium	14.50	1.58	2.33

* ($P < 0.05$)
** ($P < 0.01$)