THE ROLE OF POTASSIUM IN BOVINE NUTRITION

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ABSTRACT

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By

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A 110-day feeding trial, a 42-day appetite trial and a 40-day metabolism trial were conducted to study the role of potassium in bovine nutrition. Yearling steers were used in the first two experiments and yearling heifers in the last. A semi-purified ration was used in all three experiments. The levels of potassium fed during the feeding trial were 0.25, 0.47, 0.60 and 0.79% of the air-dry ration. During the first part of the appetite trial (22 days) a 0.25% potassium ration was fed in conjunction with intra-ruminal injections of either a potassium solution (1282.1 mEq) or water; and then a 0.60% potassium ration was fed during a subsequent recovery period (20 days). The daily potassium intakes during the metabolism trial were 1086.75 (high), 439.41 (medium) and 156.59 mEq (low).

Data from the feeding trial suggest that for rapidly growing steers the potassium requirement is greater than 0.47% but equal to or slightly less than 0.60% of the air-dry ration. Wound healing (gluteal incision) was significantly delayed in steers fed the 0.25% potassium ration when compared to those fed 0.47%.

Serum potassium levels of the 0.25% potassium steers were significantly lower than those of the other treatment groups. In contrast, serum magnesium and chloride concentrations of steers receiving 0.25 and 0.47% ration potassium were significantly higher than steers fed 0.60 and 0.79% potassium. However, there were no significant differences among treatments in serum levels of sodium, calcium and phosphorus, although in the metabolism trial serum phosphorus levels increased significantly as ration potassium decreased.

As ration potassium increased from 0.25 to 0.79% the pH and concentrations of sodium and potassium in rumen fluid, tended to increase. The in vitro microbial activity of rumen ingesta decreased non-significantly as ration potassium decreased.

Results of the appetite trial indicate that potassium deprivation was associated with loss of appetite and body weight, and lowered serum potassium levels.

Data from the metabolism trial show that heifers receiving the high and medium levels of potassium retained 147.67 and 15.47 mEq of potassium daily, respectively. Heifers receiving the low potassium ration were in negative balance (-25.13 mEq/day). Ration potassium did not appear to influence fecal potassium excretion, however, urinary potassium excretion increased significantly as potassium intake increased. There were no significant changes in body weight due to potassium treatment. Similarly, the

positivity of nitrogen balance was not significantly affected by the levels of potassium fed. However, there was a significant increase in urinary ammonia excretion as ration potassium decreased. The positivity of apparent water balance was not significantly affected by potassium treatment, nor were the apparent digestibilities of ration components.

Data from the metabolism trial indicate that a daily potassium intake of 439.41 mEq was adequate for maintenance of yearling heifers (average weight 239.9 kg).

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INTRODUCTION

Potassium is the most abundant cation in most animal cells, accumulating against an ionic gradient. However, it is much less abundant in the extra-cellular fluids. Intra-cellular concentration of potassium is similar to that of sodium in the extra-cellular fluid. Potassium is of great significance in maintenance of osmolarity and electroneutrality. Apart from this, there is much evidence that potassium is vital to enzyme systems associated with the utilization of energy, storage of glycogen, biological transport and cellular growth.

The potassium content of the body depends on intake and loss. Excretion of this element occurs largely in the urine and feces, and in the case of the ovine considerable potassium is lost with sweat. The homeostatic control of potassium concentration in the extra-cellular fluids (plasma potassium inclusive), however, is not well regulated since the renal mechanism is designed primarily to prevent hyperkalemia and not hypokalemia. There is an obligatory urinary excretion of potassium and, even when a potassium free diet is fed, 30-60 mEq per day are lost via the urine. There is therefore a dietary need for potassium.

In growing animals a positive potassium balance is vital, and several investigators have observed an increased need for potassium by rapidly growing animals. This is also true of the pregnant animal. The non-pregnant adult animal, however, need only remain in potassium balance.

As potassium is an important cellular constituent in both animals and plants, most common rations should supply normal potassium needs. Neal, as cited by Bredon (17), stated "that plants would cease to exist before they would have insufficient potassium to meet animal requirements."

Since the introduction of purified rations, the use of cation exchange resins to produce potassium deficiency in humans and the application of flame photometry for potassium determination, the interest of many investigators in the field of potassium metabolism has progressively increased. Considerable work has been done with rats, mice, guinea pigs, chickens, turkeys and man in regard to dietary potassium requirement and its metabolism in the body. However, there is a paucity of information concerning the role of potassium in ruminants. Further, a search of the literature gave no indications that the quantitative dietary potassium requirement of the bovine is known.

The investigation described in this thesis was designed to study the role of potassium in the bovine and also define the potassium requirement for rapidly growing yearling steers. Three experiments were conducted, a feeding trial, an appetite trial and a metabolism study. The following parameters were investigated: appetite, growth, serum concentrations of sodium, potassium, magnesium, calcium, phosphorus and chloride; also nitrogen and water balances and ration digestibility were determined in the metabolism study. Tissue healing time was also observed in potassium deficient and non-deficient steers.

LITERATURE REVIEW

Regulation of Serum Potassium

Knowledge of factors which regulate the serum potassium level is incomplete. It is not clear to what extent potassium ions can be withdrawn from the plasma and stored in the cells when serum potassium rises or, on the other hand, can be mobilized from the cells when serum potassium falls. It has been demonstrated by isotope dilution methods that there is a general lack of correlation between serum concentrations and body stores of sodium and potassium ions (49, 92, 61). However, some workers have suggested that a relatively low correlation exists between these two entities (10, 21, 71). Whilst a reduction of serum potassium indicates, in general, that a state of potassium depletion does exist, it can give no indication of the magnitude of depletion.

The body intake and output of potassium are of major importance in the regulation of serum potassium levels. Petersen (71) described a case of malabsorption in which fecal potassium losses were almost as high as urinary excretion, potassium balance was negative and serum potassium was low. Also, since the kidney is the major route of potassium excretion, it is not surprising that spontaneous elevations of serum potassium concentration to levels above 5.5 mEq/l may occur in some chronic renal diseases (79). In the growing animal cellular growth and multiplication involve

the accretion of cellular potassium so that a positive potassium balance is imperative. Black (10) by citing the work of McCance and Widdowson presented evidence that the infant can tolerate potassium serum levels which would be toxic to adults.

It has been suggested that when potassium loss due to deprivation is unaccompanied by excessive sodium intake, there is little tendency for hypokalemia even though a high ratio of body potassium to nitrogen loss exists together with sodium retention (62). Moore et al. (62) found that in man, the constancy of serum potassium is maintained when losses up to three times the total extra-cellular potassium occurred, and losses were as high as 40 mEq/day. Since these experiments were of short duration (3 to 7 days), there is the possibility that, during progressive potassium loss, serum levels were maintained at low normal values at the expense of intra-cellular reserves (96). When potassium loss is complicated by extra-renal salt loss which is high in chloride, or by administration of large quantities of sodium alkalosis and hypokalemia occur. This is also the case when adrenocorticotrophic hormone administration, or any form of stress is superimposed during the phase of low potassium intake. Blahd and Basset (12) reported an uncomplicated dietary depletion of potassium in one subject for 55 days during which the potassium intake was 14 mEq/day and the sodium intake was not in excess. There was no alkalosis and during the first ten days, the serum potassium gradually

fell from 4.0 to 3.5 mEq/1. Thereafter, it showed a variable tendency to rise slightly. Black and Milne (11) produced potassium depletion in two normal individuals, by dietary means, in a 6 to 7-day period. The low potassium intake was produced by passing milk through a resin column which reduced the potassium content of the milk from 43 to 3 mEq/1, and increased the sodium content from 36 to 111 mEq/1. Further, large amounts of sodium salts were added so that sodium intake during "pre" and "post" experimental periods was constantly high. During the experimental period the potassium balance was negative, showing average losses of 45 to 50 mEq/day. The serum potassium fell from 4.1 to 3.1 mEq in 6 to 7 days. These workers attributed the marked hypokalemia and alkalosis (serum bicarbonate was 36.1 mEq/1) not to the combined intake of low potassium and high sodium but only to the low intake and high loss of potassium. The induction of alkalosis increases potassium excretion (38, 27) and the potassium depletion is accompanied by metabolic alkalosis and hypokalemia (25). On the other hand, Finkenstaedt et al. (33) reported that removal of potassium by haemodialysis in the dog resulted in an acute loss of body potassium accompanied by acidosis and a marked decrease in serum potassium, while urinary potassium excretion remained almost unchanged. The common occurrence in acidosis, however, is an increase in serum potassium level (21). There is good evidence concerning a reciprocal transfer of hydrogen and potassium ions between the extra and intra-cellular fluids when acidosis or alkalosis

supervenes (21). It has been claimed that at any given level of total body potassium, there is a relationship between serum pH and the serum concentration of potassium, in that for every rise or fall of 0.1 pH unit there is, on the average, a fall or rise in serum potassium of 0.6 mEq/l. However, McCance cited by Kleeman et al. (51) has shown that the induction of alkalosis in sodium depleted subjects is not followed by increased urinary excretion of sodium and potassium.

Since the dietary intake of potassium is subject to daily variation, the regulation of serum potassium rests upon the kidney. There is evidence suggesting that all the potassium excreted in the urine could not be accounted for by the filtration reabsorption theory alone and that tubular secretion also occurs (6, 63). Recently, it has been stated that all of the filtered potassium is reabsorbed in the proximal tubules (93, 76) and that urinary potassium is evidently the result of tubule secretion (6, 76). There is in the distal renal tubule an ion exchange system by which lumen sodium ions are reabsorbed and interstitial potassium ions enter the cell and are then secreted. The rate of exchange is dependent on the availability of these two ions, and the cellular potassium must eventually come from the blood perfusing the cells (76). An equal amount of sodium is reabsorbed when potassium is secreted but this is only 1% of the filtered sodium (76). Data indicate that there is competition between hydrogen and potassium ions

for sodium in the ion exchange mechanism whereby sodium is reabsorbed in the distal tubule and that the secretion of the hydrogen ions is dependent on the enzymatic action of carbonic anhydrase (7).

Normally about 90% or more of the filtered potassium is reabsorbed (43). However, when sufficiently large quantities of potassium are administered, renal tubular secretion of the cation takes place (8). Blahd and Bassett (12) found that even when the body is in a state of potassium depletion, there is a persistently small and nearly constant excess of potassium excretion over intake, which under prolonged restriction could lead to a severe deficit. There is evidence suggesting that renal mechanisms promoting the excretion of potassium and the conservation of sodium are more active in the cow than in man and dog (2). Intravenous infusion of potassium salts into the bovine resulted in a very high rate of tubular secretion of potassium with no progressive increase in serum potassium concentration (2). However, in the dog, there was a progressive increase in serum potassium unless the animal had been made tolerant to potassium loading, by dietary means, two weeks before the infusion (8). Anderson and Pickering (2) suggested that this adaptation may be the result of high potassium and relatively low sodium content normally present in the diet of cattle. Generally, however, when a dose of potassium is given to any normal animal, it is excreted more or less quantitatively in the urine. However, potassium administration to an animal,

whose body has been previously depleted of the element, will result in a progressive rise of serum potassium to normal level and complete tissue repletion. Once the stores are repleted, any excess potassium is eliminated quantitatively in the urine (11, 12, 33, 37, 62). The slow rise of serum potassium during repletion may be more closely linked with the acid-base balance than with body potassium content. It has been shown that with repair of alkalosis by administration of ammonium chloride there is, with no addition of potassium to the body, a serum rise of potassium from 3.3 to 4.4 mEq/l (62). It has been proposed that during the production of alkalosis by potassium deficiency, three potassium ions leave the cell fluid in exchange for two sodium and one hydrogen ions from the extra-cellular fluid, thereby elevating the plasma pH (7). The kidneys will then excrete a somewhat alkaline urine containing potassium and bicarbonate ions. As intra-cellular potassium is depleted, serum potassium concentration soon falls and the now depleted renal tubular cells secrete excessive quantities of hydrogen and ammonium ions, thus aggravating the existing acid-base imbalance. In acidosis, the hydrogen ions are excreted at the expense of the potassium ions and retention of potassium occurs (43).

Hyperkalemia is a very uncommon occurrence even in chronic renal diseases (7). The diseased kidney, like the normal organ, is capable of secreting potassium ions by the

process of ion exchange for sodium ions in the distal tubular lumen (7). When renal function ceases abruptly, as in the case of lower nephron nephrosis or in certain chronic renal diseases, the resultant hyperkalemia may even increase to toxic levels (79).

The renal tubular activity in regulating potassium excretion depends also on the level of adrenocorticoid activity. These hormones, particularly aldosterone, control the handling of sodium and potassium by the kidneys. Under the influence of aldosterone the excretion of sodium, and less regularly of chloride, is lowered while that of potassium and magnesium is increased. When potassium is withdrawn from the diet, aldosterone secretion is also lowered. On the other hand, an intake of large amounts of potassium brings about an increase in aldosterone production. When the supply of aldosterone is low, as in Addison's disease, serum potassium concentration often rises and serum sodium concentration falls. When an excessive amount of this hormone is in circulation such as in Cushing's Syndrome, under stress conditions, or in the course of aldosterone administration, the serum potassium concentration is reduced. It has been stated that regulation of serum potassium is under the influence of adrenalin rather than the activity of adrenal cortex secretion (28). However, this does not lessen the effect of cortical hormones upon potassium absorption and excretion, since the experiment of Fluckiger and Verzar (35) suggested an extra-renal effect of the mineralo-corticoids

on sodium and potassium regulation. These workers incubated diaphragm muscle, from adrenalectomized and normal rats, in Ringer's solution containing radio-active sodium and potassium both with and without added glucose and insulin. Their results indicate that, with or without glucose and insulin, the diaphragm muscle of the adrenalectomized animals took up more potassium and, in the absence of glucose and insulin, much less sodium than those of the normal rats. The authors concluded that adrenalectomy impairs the ability of muscle to maintain the normal equilibrium for sodium and potassium.

The genesis of Familial Periodic Paralysis is not certain but it is characterized by a fall in serum potassium. It appears that the low serum level is not due to any increase in renal excretion but rather to a movement of potassium from the extracellular fluids into the cells. This paralysis can be accelerated by the administration of glucose or insulin, a process which results in a decrease of serum potassium due to cellular storage of glycogen.

Accumulative data indicate that magnesium has an effect on potassium balance (71, 48, 94). The mechanism by which magnesium ions decrease renal potassium and increase sodium and chloride excretion remains unknown. Jabir et al. (48) suggested that magnesium might enter into a tubular ion exchange mechanism causing a decrease in tubular secretion of potassium. However, Whang and Welt (94) presented data in support of an effect of magnesium on potassium metabolism not through the kidney but at the cell level.

The Biological Functions of Potassium

The content of potassium in the body is similar to that of sodium but unlike the latter, it exists primarily as a cellular constituent. Intra-cellularly, potassium is mainly in the ionic state and many of its biological effects are produced at the cell level. The modern concept of membrane permeability has necessitated the postulation of a sodium-potassium coupled pump mechanism. This concept helps to explain the existence of a high intra-cellular potassium concentration in which an electrochemical potential gradient across the cell membrane is set up by the active extrusion of sodium. This pump is an energy assisted transport reaction. It depends not only on cellular adenosine triphosphate (ATP) but also on the absolute concentrations of cations which are known to affect either directly or indirectly the ATP-ase activity of the cell (72, 54). This concept of active transport plays an important part in influencing many essential phenomena which are potassium dependent. These include the following: (a) The electrical activity of nerve and muscle cells and the process of synaptic transmission (43). (b) The distribution of ions and water between the various body fluid compartments and the regulation of interstitial and intra-cellular pH. (c) The secretive (81) and absorptive processes (72, 75) of the gastro-intestinal tract. (d) Cellular respiration (9, 3). (e) The formation of urine by the kidney.

Potassium is necessary as an activator of certain enzyme systems, which include ATP-pyruvate phosphotransferase, myosin ATP-ase and choline acetylase. Also potassium is intimately involved in the transfer of high energy phosphate bonds to the adenylic system (15, 16). Thus potassium deficiency lends to uncoupling of oxidative phosphorylation, which in turn may result in impaired carbohydrate and protein synthsis. Since sodium is antagonistic to potassium in relation to enzymatic activity, a concomitant cellular influx of this cation during potassium depletion may very well aggravate the disturbance in cellular metabolism (10). In vitro studies have shown that potassium is also important in the synthesis of phospholipids, in that large amounts of sodium and potassium are found associated with lecithin, cephalin and suphatides of the brain (20). It is believed that these cations compete with each other for combination with lipids, and also that changes in lipid structure may affect electrolyte balance which in turn could affect the metabolic activities of brain cells (72).

Potassium seems to be involved in carbohydrate metabolism, facilitating its systhesis, translocation and storage. In vitro studies indicate that optimum glycogenesis in liver slices requires the presence of potassium (41, 89). Fenn (32) presented evidence that deposition of glycogen in the liver is also accompanied by potassium deposition. Further, Hastings et al. as cited by Tuerkischer and Wertheimer (89), demonstrated that in rat liver slices synthsis of glycogen

takes place only in a potassium-rich, sodium-free medium corresponding in ionic composition to intra-cellular fluid. On the other hand it has been reported that potassium, even in physiological concentrations, will inhibit glycogenesis by diaphragm tissue (89). At higher concentrations of potassium, glycolysis was shown to occur. This observation of opposite effects of potassium in the liver and diaphragm muscle is of great physiological importance in the regulation of synthesis and breakdown of glycogen. During muscle activity there is a decrease in muscle potassium content (95) with as much as 30% being released into the extra-cellular medium (82). Considerable amounts of this are taken up by the liver, and released during muscle recovery (32). Boyer (15, 16) has shown that potassium is necessary for the transfer of phosphate from 2-phosphopyruvate to creatine which results in the formation of ATP. Creatine phosphate serves as a reservoir of energy for the re-phosphorylation of adenosine diphosphate (ADP). In fact, some data suggest that phosphocreatine exists in skeletal muscle as a di-potassium salt (64). Thus, it would seem that the phenomena of glycogenesis and glycolysis result in the translocation of potassium with its concomitant production of energy for tissues according to need.

Accumulated *in vitro* data concerning the effect of potassium on carbohydrate metabolism has been very inconsistant. Gardner *et al.* (41) reported that potassium deficiency

does not cause diminution of liver potassium. His rats, after a potassium depletion of 60 days, showed an abnormally high content of post-prandial liver and muscle glycogen. However, when the depletion period was extended to 120 days, there were only traces of tissue glycogen. In contrast, Fuhrman (40) reported low post-prandial liver and muscle glycogen levels in rats depleted of potassium over 8 days. Further, the liver and skeletal muscle content of glycogen increased above normal during fasting. Both groups of workers reported enlarged adrenal glands in the potassium deficient rats and attributed the increase in tissue glycogen to gluconeogenesis, brought about by the over production of adrenal cortical hormones. But the difference in their results may be due to the fact that, in the experiment of Gardner et al. (41), the rats were not as completely depleted of potassium as were those of Fuhrman (40). In the former experiment the rats gained weight slowly until the 40th day, after which time they showed deficiency signs. However, in Fuhrman's experiment 40% of muscle potassium was lost after the 7th day and mortality began after the 8th day of the experiment.

In view of a certain consistency in the ratio of potassium to nitrogen in muscle tissue, there appears to be a relationship between potassium and protein metabolism. This relationship has been complicated in many experiments, however, by anorexia which is often associated with potassium deprivation. Recently, careful potassium and nitrogen studies

in which caloric intakes were sufficient to prevent excessive catabolism of protein have been conducted (23, 28, 39). Cannon et al. (23) demonstrated the need for potassium in tissue synthesis. They found that rats depleted of protein and then given special diets containing a complete mixture of essential amino acids failed to respond when potassium was withheld during the repletion period. Addition of small amounts of potassium chloride to the deficient ration resulted in prompt, effective protein repletion. Frost and Smith (39) re-emphasized the need for a certain dietary level of potassium to facilitate weight gain and tissue protein synthesis. Davis and Loosli (28) citing the work of Frost et al. reported a similar relationship of potassium to amino acid utilization in rats.

Consequences of Potassium Depletion

Potassium deficiency is accompanied by compositional changes in the body tissues and fluids. This condition results not only in changes in potassium concentration but also in alterations in acid-base balance and changes in cellular structure and activities. The total amount of body potassium, however, can be changed markedly without disturbing potassium metabolism. For example, in starvation, the potassium excreted comes from the cells, partly from the breakdown of proteins and partly from the breakdown of other compounds which contain potassium (43). The potassium set free from combination with protein is generally excreted in the urine in the

ratio of 3 mEq potassium to 1 g nitrogen (62). Potassium depletion, therefore, may not exist under some conditions since there is an equal decrease in both potassium content and the body tissues (capacity) which hold the potassium ions (21). Burnell and Scribner (21) define potassium depletion as "a decrease in potassium content in relation to capacity".

In general, however, a low level of serum potassium is indicative of potassium depletion but, as previously indicated, the correlation is by no means a strict one. In potassium depletion there is a shift of potassium ions from the cells into the extra-cellular fluid (25, 91). This loss of cellular potassium is accompanied by partial replacement with sodium, and occurs in skeletal (22, 24, 41, 68, 91) and cardiac muscles (68, 91) and to a lesser extent in kidneys (68, 91). Campbell (22) and Ziegler et al. as cited by Welt et al. (91) reported increased liver potassium levels in depleted animals. However, the majority of reports on rats have indicated no change in liver potassium content (41, 91). The apparent stability of liver potassium in face of a significant deficit of body potassium may be the result of a concomitant loss of liver organic material along with the cation (91). One argument in favour of this suggestion is the observation that following partial hepatectomy, the potassium level in normal rat liver did not change significantly during liver regeneration (65). In contrast, Dodgen and Muntwyler as cited by Welt et al. (91) observed

an actual increase in liver glycogen in potassium deficient rats. Fenn (32) demonstrated that in rat liver, glycogen deposition is accompanied by increases in the amounts of water, potassium and phosphate.

In accord with Cooke's concept, (25) it seems reasonable to assume that the alkalosis observed in potassium deficient rats is accompanied by cellular acidosis. Kleeman (51) reported that potassium-alkalosis in man is characterized by urinary excretion of large amounts of organic acids and appreciable amounts of ammonia in an alkaline urine. A suggested explanation is that renal glutaminase activity is increased in potassium depletion, therby favouring hydrogen-sodium exchange in the renal tubules (26). Thus a distortion of the acid-base balance results. This is frequently associated with a fall in extra-cellular chloride (91) and invariably the carbon dioxide combining power and pH of the serum increases (11, 51, 61), as do the sodium content and extra-cellular space (51, 91). Campbell (22) and Telle et al. (88) reported an increase in serum calcium and a decrease in serum magnesium in potassium deficient sheep. A fall in the serum hematocrit (12, 88) and organic acid levels (51) has also been observed in potassium deficiency.

Potassium depletion leads to impairment of cellular function. Smith and Blaxter (81) reported that potassium deficient rats secreted a much smaller volume of gastric juice than normal rats. Cannon et al.(23) have shown that the specific effect of potassium depletion is a failure of

tissue protein synthesis. Other workers have demonstrated that nitrogen balance is less positive in potassium depleted than in normal animals (22, 62). Riggs et al. (74) showed that depletion of cellular potassium with its concomitant replacement by sodium was the common denominator linking together a great variety of toxic actions suppressing amino acid transport at the cell level. Carbohydrate metabolism is also affected by potassium depletion. In the intact animal there is a decreased rate and absolute amount of glucose uptake at the cellular level (77). Since potassium is an essential cofactor for activation of certain enzyme systems concerned with energy utilization, a malfunction of such a system could be related to degenerative changes. It is thus understandable that severe potassium depletion will elicit adverse effects in different types of muscle. In striated muscle there is weakness with diminished reflex action (10); whereas in smooth muscle the atonicity may result in paralytic ileus (10, 66). Cardiac action is also weakened and is usually accompanied by arrhythmia, tachycardia and electrocardiographic changes (10, 50, 84).

The gross symptoms associated with potassium deficiency are anorexia and pica. Campbell (22) reported wool pulling, listlessness and emaciation in potassium depleted lambs. Poor growth and loss of weight are classic symptoms of potassium deficiency, apparently due, in part, to anorexia (19, 22, 41). General weakness is a prominant symptom of

severe potassium depletion. Hughes and Ittner (47) reported that pigs fed a ration devoid of potassium were generally unable to stand and some exhibited unsteady gait. Such a ration resulted in a 100% mortality at the end of 4 weeks. Diarrhea (41), edema, polyuria and polydipsia (66) have been observed in potassium deficient rats, but occurrence of these symptoms are inconsistant in other species (91, 22).

Some of the morphologic and histochemical changes in potassium depletion include inflammation of mucosa of stomach and large intestines (47), impaired rumen papillae growth (19) and Zinker's necrosis in the fibres of striated muscle. Fuhrman (40) reported adrenal hypertrophy and thymus hypotrophy in depleted rats. However, the most classical histological and histochemical changes are found in the kidneys and heart. Reports indicate a bilateral enlargement of the kidneys, interstitial fibrosis, cloudy swelling, hyaline droplets and abnormal mitotic figures of the tubular cells (22, 66, 91). In addition, the distal convoluted segments may be dilated due to the presence of hyaline casts in the lumen. A variety of enzymatic changes have also been reported to occur in the kidney (91). Pearse and Macpherson (70) reported increases in acid phosphatase in the collecting tubules and non specific esterases in the proximal tubules. These workers also observed an increase in niacinamide adenine dinucleotide phosphate (NADP)- diaphorase with a concomitant decrease in niacinamide adenine dinucleotide

(NAD)-diaphorase. They interpreted this as suggesting a disturbance in cellular respiration and oxidative phosphorylation, particularly in the tubules. The myocardial lesions observed include noninflammatory degenerative changes, widespread necrosis, cellular infiltration (66) and abnormal mitotic figures (66, 91). It has also been reported that there is no abnormality in size of the heart (66).

Potassium Requirements

All animals require a dietary source of potassium, which can be satisfactorily supplied by natural rations or by addition of the element to purified rations. Gills (42) has shown that chicks require 0.2% potassium in an otherwise adequate diet for maximum growth. This level, however, should be increased to 0.24% to insure optimum performance when phosphorus content is sub-optimum. However, under these conditions ration levels of potassium above 0.24% depressed growth. McWard and Scott (57) reported the potassium requirement of the chick for maximum growth in the presence of 0.38 and 0.11% sodium to be 0.21 and 0.28%, respectively. These values compare quite well with those reported by Gillis. In contrast, Davies and Loosli (28) stated "the requirement of the chick is about 0.4% of the diet but is affected by the rate of growth and the sodium content". If the sodium content of the diet is 0.8%, then a potassium level of 0.33% is adequate for maximum growth (28). In a study on the effects of zinc and potassium on bone

formation, feathering and growth of poults, Supplee et al. (85) reported that a dietary potassium level of 0.36% is required. Sullivan (87) suggested the potassium requirement to range between 0.32 and 0.38% for maximum performance in growing turkey poults. A dietary level of 0.175% or less resulted in 100% mortality within 21 days. In contrast, Supplee and Combs (86) reported that for adequate growth, a dietary level of 0.6% potassium is required for unmedicated feeds and 0.45% for antibiotic medicated feeds. The relationship of antibiotic to potassium utilization remains to be explained.

The relationship of potassium and sodium to growth has been studied by Miller (60). He fed rats a high potassium-low sodium diet and reported satisfactory growth. But when a high sodium-low potassium diet was fed, it was observed that sodium did not replace potassium for rats. He also demonstrated that a ration level of 0.1% potassium was sub-optimum and retarded the growth of rats. Orent-Keiles and McCollum (68) demonstrated pica and subnormal growth when they fed rats a purified diet containing 0.01% potassium.

A number of reports have appeared in the literature with reference to the potassium requirements of growing swine, but there is no information available for reproducing or lactating sows. Hughes and Ittner (47) suggested that for optimum growth young pigs require about 0.15% potassium and that 0.08% potassium will result in deficiency

symptoms. These workers reported a 100% mortality when a ration completely devoid of potassium was fed to young pigs for 4 weeks. Meyer et al. (59) showed that pigs require a ration containing 0.23 to 0.28% potassium. Other workers (50) reported similar results. The requirement reported by Meyer et al. is about double that obtained by Hughes and Ittner. The rates of growth reported by Meyer et al. were much higher than those of Hughes and Ittner which may explain the higher potassium requirements by pigs in the former experiment.

Recently there has been much interest in defining the potassium requirements for ruminants. Telle et al. (88) reported severe potassium deficiency in lambs fed less than 0.2% potassium and observed near optimum performance when the ration contained 0.3% potassium. However, the feedlot performance was generally superior at 0.5% potassium. Brink (19) and Campbell (22) found that the potassium requirement for optimum growth of lambs was greater than 0.3% but not greater than 0.5% of the ration. Delvin and Roberts (30) reported the daily maintenance potassium requirement of wether lambs in the presence of 2.9 g of sodium to be 1.2 g. Campbell's (22) data suggests the potassium maintenance requirement for lambs, when 2.1 g. of sodium is fed, to be greater than 0.7 but less than 2.2 g/day. Both these workers (22, 30) determined their maintenance values from balance trials. In contrast, Telle et al. (88) used data

from a feeding trial and estimated the potassium requirement for maintenance of wether lambs (averaging 27.7 kg in weight) to be close to 65 mg/kg body weight (1.8 g/day). This would mean that lambs (average weight 33.6 kg) investigated by Campbell (22) in a potassium feeding trial, would require 2.2 g potassium daily for maintenance.

Evidence for the bovine potassium requirement, however, is very poor. Du Toit et al.(31) fed Friesland heifers, averaging 363.2 kg, a daily basal ration consisting of hay (1.58 kg), maize (4.54 kg) and blood meal (20 g) over a period of 2.5 years. Two animals were fed the basal ration which was considered low in potassium (0.34% of the dry ration) while a control heifer received the basal ration plus adequate potassium supplementation. The animals passed through two gestation and two lactation periods. During growth and pregnancy a daily intake of less than 24.9 g of potassium appeared to be adequate and failed to elicit signs of deficiency. After the first gestation period, one of the treatment heifers died. During the lactation period the basal ration was modified in potassium content by the addition of 2.27 kg of maize ensilage. It was then observed that a daily potassium intake of 31.5 g satisfied the minimum requirement for lactation. Over the 2.5 years, however, growth was slow and the heifer fed the low potassium ration gained 154.7 kg, which was not significantly less than that gained by the control.

EXPERIMENTAL PROCEDURE

Experiment 1

Twenty-four yearling Hereford steers, fairly similar in phenotype and weight, were selected and used in a 110-day feeding trial to define the potassium requirement for rapidly growing steers. The animals were group fed and adjusted during 23 days from an all hay ration to a semi-purified basal ration, the composition of which is listed in Table 1. After the initial adjustment period the steers were randomly sorted into four groups of six, and equilibrated on a ration containing 0.60% potassium for an additional 7 days. Ration potassium levels (expressed as percentage of the air-dry ration) of 0.25, 0.47, 0.67 and 0.79% were made by adding appropriate quantities of K_2CO_3, at the expense of dried brewers grains, to the basal ration. These rations were mixed in a vertical mixer in batches of 227 kg, and were randomly assigned to the four groups. The ration levels of potassium are designated for future reference in the text as groups 1, 2, 3 and 4, respectively. At initiation of the trial, the average weights of steers in the four groups were 282.2, 281.4, 291.8 and 277.5 kg, respectively. The animals were group fed ad libitum and had free access to tap water (0.13 mEq of potassium/l). No bedding was provided since it was likely that the animals would eat a certain amount of bedding and thus obtain additional potassium.

Daily inspections of the steers were made and any

TABLE 1. BASAL RATION COMPOSITION***

INGREDIENT	PER CENT
Corn starch	15.04
Animal tallow	3.00
Dehydrated alfalfa	1.00
Dried brewers grains	74.70
Barley	5.01
Sodium chloride	0.50
Tricalcium phosphate	0.67
Vitamin A supplement*	0.07
Trace mineral supplement**	0.01

* Vitamin A supplement was prepared by mixing 1980 g of wheat middlings with 20 g of Vitamin A palmitate (250,000 I.U./g).

** Trace mineral supplement contained the following in g/100 g: $FeSO_4.5H_2O$, 71.8; $MnSO_4.H_2O$, 15.5; $CuSO_4.5H_2O$, 12.7.

*** Contained (a) 0.08% potassium and (b) 0.23% sodium of which 0.02% was inherent in the ration.

abnormality in behaviour and physical appearance noted. The steers were individually weighed every week and average gains recorded. Also bi-weekly venous blood samples were drawn by the jugular puncture technique. The blood samples were allowed to clot for 2 hours, centrifuged at 2200 rpm for 30 minutes and the extracted serum stored at -18°C until analysed.

On days 55 to 58 inclusive rumen contents were collected from steers in all the groups (one animal from each group daily) by use of a stomach tube and suction pump. The pH of each sample was immediately determined using a glass

electrode pH meter. As soon as possible after collection (approximately 90 minutes) duplicate 15 g subsamples of rumen contents from each steer were transferred to 160 ml Warburg flasks. In vitro rumen microbial activity was then determined by a modification of a manometric method described by McBee (56). Dry matter of each rumen sample was also determined. The remaining portion of each sample was strained through four layers of cheese cloth and the rumen fluid stored at -18°C until analysed.

At conclusion of the trial (110 days), the steers from group 3 and 4 were slaughtered and the shrunken live weights (19-hour shrink period) and hot carcass weights recorded. Government carcass grades were obtained in order to get an indication of carcass finish.

Two days following the end of the trial, muscle biopsy (skin incision technique) of the right gluteal muscle was made from three animals randomly selected from each of groups 1 and 2. The samples were preserved in formalin for slide preparation and histopathological examination. In addition, the number of days required for the incisions to apparently heal was visually estimated over a subsequent 23-day period.

Experiment 11

After Experiment 1 was concluded a 42-day appetite trial was conducted with the steers that had been fed the 0.25 (group 1) and 0.47% (group 2) potassium rations. All

steers were initially fed the same potassium ration (0.25%) for a period of 14 days in order to adjust the steers from group 2 to the lower level of potassium. The animals within each group were then randomly divided into two subgroups of three steers each. These will be referred to as groups 1a, 1b, 2a and 2b. Groups 1a and 2a were used as controls because groups 1 and 2 were initially in different physiological planes of potassium. Each steer was then intraruminally injected (a 14 gauge hypodermic needle 9 cm long and a 50 cc glass syringe were used) through the left paralumbar fossa with either a potassium solution or deionized water. The steers in groups 1b and 2b received 50 g potassium (1282.1 mEq) as K_2CO_3 dissolved in 100 ml of deionized water every second day for 22 days. The control steers (groups 1a and 2a) were treated in a similar manner except only 100 ml of deionized water was injected. The animals were group fed ad libitum the 0.25% potassium ration and had free access to drinking water. Each animal was weighed at initiation of the experiment and again, 1 day following its termination. Average daily feed consuption was recorded. Blood and rumen samples were collected 1 day after the last injection and processed as in Experiment 1. After the last injection period, the ration of all steers was changed to a diet adequate in potassium (0.6%) and fed for an additional 20 days. At the end of this period the steers were weighed and blood and rumen samples were collected for analyses. The average feed consumption was recorded, and average daily body weight

gains were calculated.

Experiment 111

A metabolism study was conducted with yearling Hereford heifers, which involved three separate trials and three different animals in each trial. These heifers ranged in weight from 209.8 to 258.3 kg. Each animal was initially adjusted from an all hay ration to a 0.40% potassium ration over a 20-day period, and then equilibrated on a similar ration (described later in the text) for a period of 6 days prior to initiation of the trial. Each metabolism trial was divided into an 8-day pre-experimental and a 40-day experimental period. The former consisted of a 5-day collection period, followed by a 3-day non-collection period; while the latter consisted of three consecutive 5-day collection periods and a 10-day non-collection period followed by three consecutive 5-day collection periods. During the collection periods the heifers were kept in metabolism crates and then returned to stalls during the non-collection periods.

During the period of equilibration each heifer was fed twice daily with a total intake of 2.72 kg basal ration (Table 1), 30 g vitamin-mineral supplement (Table 1) and 386.24 mEq of potassium as K_2CO_3. The same feeding regime was continued throughout the 8-day pre-experimental and 40-day experimental periods, except during the latter when the daily potassium intakes (from K_2CO_3) were changed to 1032.87, 386.24 and 103.29 mEq. Therefore, the total daily potassium intakes

which included that suppled by the basal ration and water were 1086.75, 439.41 and 156.59 mEq, and these levels will be referred to as high, medium and low potassium, respectively, throughout the remainder of the text. Thus, during the experimental period three heifers received each level of potassium. Sodium intake, including that supplied by the water, was relatively constant throughout the trial and averaged 256.43 mEq/day for the nine heifers. The gross energy of the basal ration was 5.0401 kcal/g. The nitrogen, crude fibre, moisture and ether extract contents of the ration were 3.53, 12.65, 6.87 and 10,19%, respectively. Tap water (containing 0.13 and 0.10 mEq of potassium and sodium/l, respectively) was offered ad libitum and the daily amount consumed recorded. The heifers were weighed at the beginning and end of each trial, while blood samples were collected for analyses on the last day of each trial.

During the collection periods, urine and feces were collected separately. Each day the feces were removed from the metal collection trays, weighed, mixed once and aliquots stored at -18°C. Aliquot samples from five consecutive daily feces collections were thawed and mixed in a Hobart mixer and a subsample retained for analyses. Urine was collected from each heifer by means of a retained vaginal catheter which emptied into a plastic collection bottle containing 15 ml concentrated H_2SO_4 plus 50 ml toluene. The daily quantity of urine voided was measured, and 2% of each daily collection was removed and frozen (-18°C). Composite samples

of five consecutive daily collections were later made and used for analyses. Daily aliquot samples of urine were taken during the pre-experimental and also during the last 5 days of the experimental period. These samples were analysed for ammonia-nitrogen within 1 hour after collection. In addition, urea-nitrogen was determined on each 5-day composite sample for the same collection periods.

On the first day of the third 5-day collection period of the last trial, however, the heifer receiving the low potassium ration ruptured its urethra and voided the catheter. The animal was then removed from the trial because it was not possible to replace the catheter.

Analytical Methods

Nitrogen in the ration, feces and urine were determined by the macro-Kjeldahl method (Association of Official Agricultural Chemists, 4). Two ml of urine and approximately 1 g each of air-dry feces and ration were used for nitrogen analysis. Dry matter of ration and feces was determined by a modification of the oven drying method described by A.O.A.C.(4). The ration and feces samples were dried at 60°C for 72 hours and then allowed to equilibrate at room temperature. The samples were then ground in a Wiley mill and subsamples used for analyses. Gross energy of ration and feces samples, from the last 5-day collection period, was determined in a Parr adiabatic oxygen bomb calorimeter (69). The ether extract and the crude fibre content of these

samples were determined according to A.O.A.C. (4) methods. Apparent digestion coefficients for nitrogen, dry matter, ether extract and crude fibre were calculated according to the total collection method.

Sodium and potassium analyses were determined on samples of the ration, feces, urine, rumen fluid, serum and tap water by flame photometry. A "Hitachi" Perkin-Elmer specto-photometer with a flame attachment, which is a direct intensity instrument, was used. The method used was as described by the manufacturers (45). However, since potassium is present in low concentrations in serum, dilutions of 1:25 and 1:20 were used. At these dilutions the effects of interfering cations, mainly sodium, resulted in abnormally high potassium values when a calibration curve using standards containing only KCl was made. To overcome the problem of interference an artificial serum was made and used in the working standards. The artificial serum consisted of the following in g/l: NaCl, 80.72; KCl, 0.209; KH_2PO_4, 0.176; $CaCl_2.2H_2O$, 0.370; and $MgSO_4.7H_2O$, 0.250 in deionized water (1). For greatest accuracy the bracketing technique, rather than reading from a calibration curve was used for the serum potassium analysis. For sodium and potassium analyses, all fluid samples were diluted with various quantities of deionized water so as to fall within the reading range of the prepared standards and analysed directly. The feces and ration samples were analysed in a similar manner after wet ashing with concentrated HNO_3 followed by 70% $HClO_4$ (approximately 5 ml HNO_3 and 2 ml

$HClO_4$/g of dry sample were used).

Serum chloride, inorganic phosphorus and total calcium and magnesium were determined according to the methods of Schales and Schales (78), Fister (34) and Walser (90), respectively. Urine ammonia and urea were determined by the modified aeration method of Van Slyke and Cullen as described by Hawk et al. (44).

Statistical evaluation of the data was by analysis of variance and Duncan's multiple range test (83).

RESULTS AND DISCUSSION

Experiment 1

Effects of ration potassium levels on steer feedlot performance. There was a marked treatment effect on steer feedlot performance. Body weight changes are illustrated in Figure 1, and total average weight gains were 9.9, 98.8, 145.8 and 138.0 kg for groups 1 to 4, respectively, during the 110-day experimental period (Table 2). Rations containing 0.47, 0.60 and 0.79% potassium supported significantly ($P<0.01$) greater gains than the ration containing 0.25% potassium. There was no significant ($P>0.01$) difference in weight gains between groups 3 and 4.

The average daily feed consumption during the experiment revealed a very marked effect of ration potassium upon appetite (Table 2). Steers fed the 0.25% potassium ration consumed only 3.82 kg daily which was about 60% that of steers fed the 0.47% potassium ration and less than 50% of that consumed by steers receiving the two highest levels of potassium. Feed consumption (expressed in weekly intervals) of steers receiving the different treatments are shown in Figure 2, and in general they parallel the body weight gains shown in Figure 1. A slow but progressive depression in appetite was observed in group 1 while, with the exception of an initial depression by group 2 steers, there was a progressive increase in feed intake by the other groups. Group 3 steers attained and maintained the highest feed

TABLE 2. EFFECTS OF RATION POTASSIUM UPON GROWTH AND VARIOUS SERUM ELECTROLYTES IN STEERS

Item	Treatment Group			
	1	2	3	4
Ration potassium %	0.25	0.47	0.60	0.79
Number of steers	6.0	6.0	6.0	6.0
Av. initial wt, kg	282.2	281.4	291.8	277.5
Av. final wt, kg	292.1	380.1	437.6	415.5
Av. total gain, kg	9.9±18.37	98.8^{A}±14.14	145.8^{Aa}±5.30	138.0^{Aa}±6.26
Av. daily feed, kg	3.82	6.28	8.14	7.84
Av. feed/kg gain, kg	42.44	6.98	6.17	6.27
Serum potassium, mEq/l	3.71±0.82	4.11^{A}±0.09	4.28^{A}±0.04	4.32^{A}±0.05
Serum sodium, mEq/l	144.17±1.52	146.67± 1.56	145.00± 0.10	147.00± 1.31
Serum chloride, mEq/l	110.08^{A}±1.35	106.89^{AB}±1.24	103.79^{BC}±0.87	101.24^{C}±0.85
Serum calcium, mg/100 ml	10.22± 1.63	9.95± 1.63	9.77± 0.12	9.46± 0.25
Serum magnesium,mg/100 ml	2.88± 0.07	2.27± 0.06	1.70^{A}±0.03	1.88^{A}±0.15
Serum phosphorus, mg/100ml	10.04± 0.66	9.26± 0.24	9.13± 0.64	8.49± 0.03

A,B,C Treatment means within an item not showing the same superscript letter are significantly ($P < 0.01$) different.

a,b,c Treatment means within an item not showing the same superscript letter are significantly ($P < 0.05$) different.

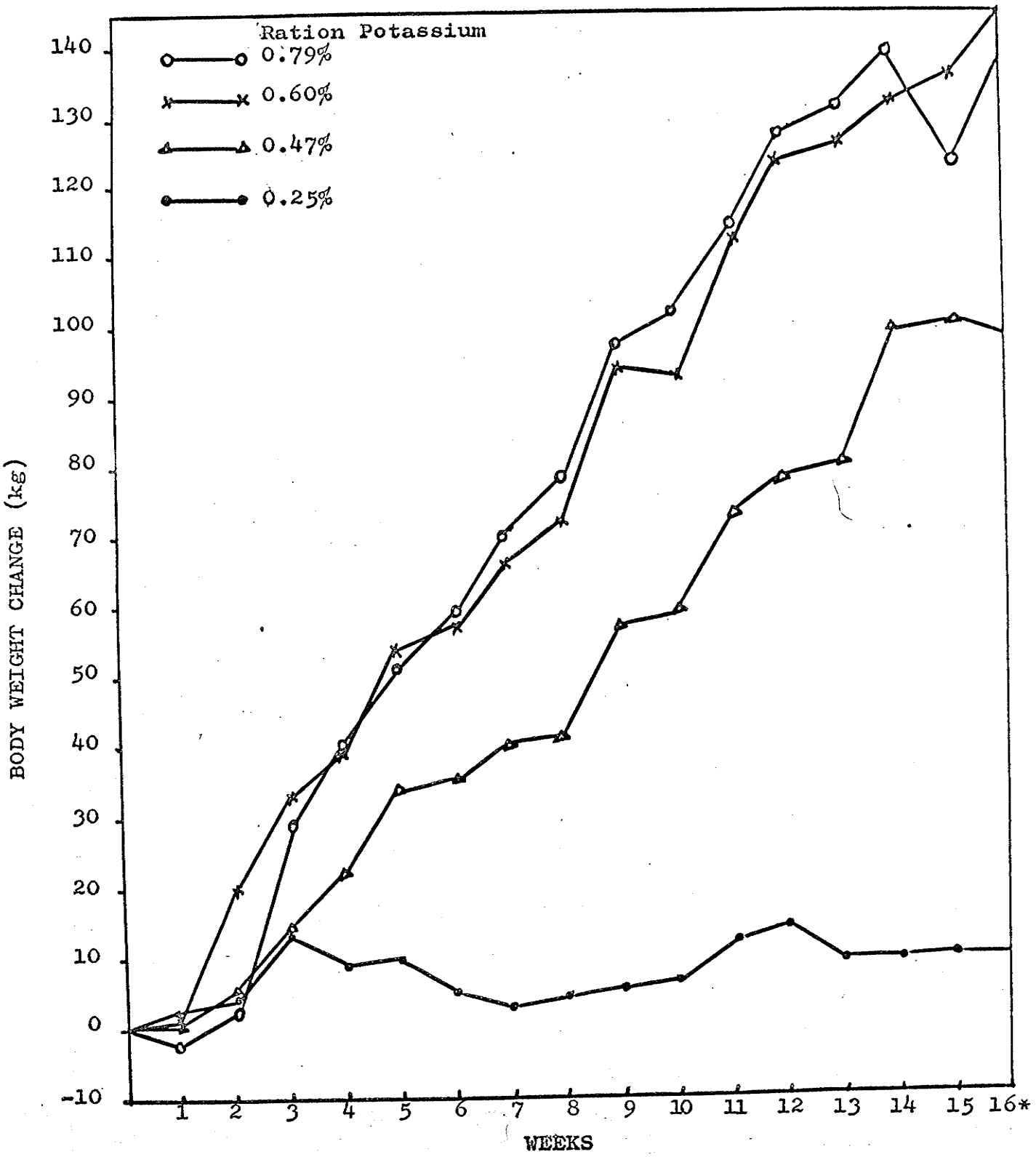

FIGURE 1. Body weight changes in steers fed rations containing various levels of potassium (0.25, 0.47, 0.60 and 0.79%).
* The 16th week was 5 days.

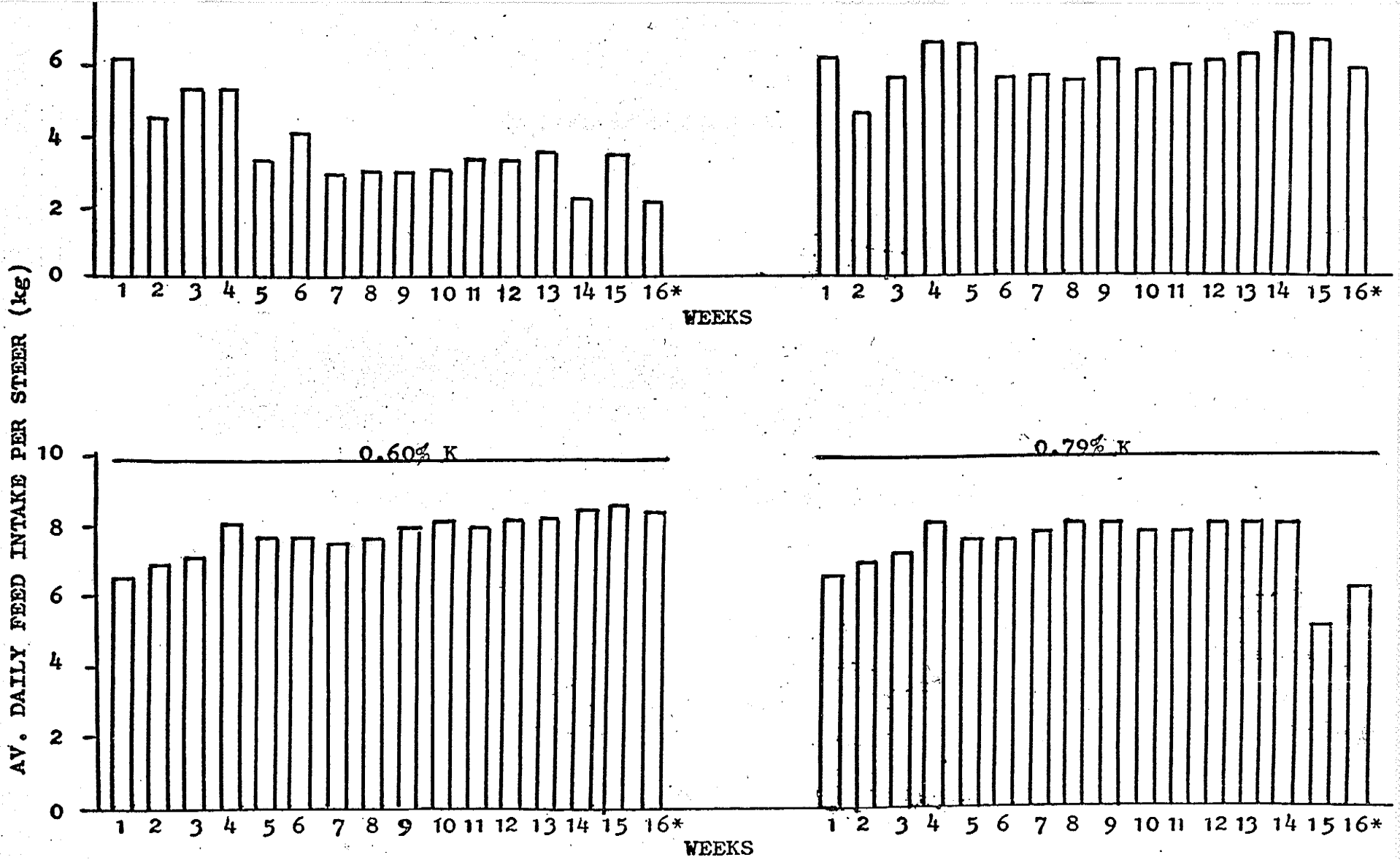

FIGURE 2. Weekly variation in daily feed consumption in steers fed rations containing 0.25, 0.47, 0.60 and 0.79% potassium.

* The 16th week was 5 days.

consumption. The 0.79% potassium ration seemed to have a mild adverse effect on group 4 during the last 12 days of the experiment, which was indicated by a reduction in body weight and feed intake.

The wide variation in total body weight change (58.1 kg gain to 57.2 kg loss) as indicated by the high standard error of group 1 steers tends to indicate that the effect of potassium on appetite varies quite markedly among individual animals (Table 2). Potassium was offered as a percentage of the ration and the intake of the element would partly depend on the amount of feed consumed. Thus in group 1 the initial reduction in feed intake caused by a low ration potassium would further enhance the adverse effect of lack of potassium on appetite with each further reduction in feed intake. The feed required/kg body weight gain was about the same for groups 3 and 4 (6.17 and 6.27, respectively), and slightly less for group 2 (6.98). However, steers in group1, in which case gains were relatively small, showed a value of 42.44 kg feed required /kg of body weight gain. This latter value is not surprising since the steers gained very little and the level of feed consumed would represent approximately the maintenance requirement of these animals. The type of ration used in this experiment may have enhanced the adverse effects of low potassium intake on appetite and growth. The ration was exceptionally high in crude protein (21.56%) in relation to that normally required by steers of this age and weight, and it might be that the requirement of potassium was

increased by this high level of ration protein. Evidence to support this suggestion has been reported by Leach et al. (53). These workers observed an increased need for potassium in chicks when a high-protein or high-energy ration was fed.

The feedlot data suggest that the potassium requirement for rapidly growing steers is greater than 0.47% but either equal to or to less than 0.60% of the air-dry ration. Telle et al. (88) reported nearly optimum growth when lambs were fed a ration containing 0.3% potassium and that growth was greatest at 0.5% potassium. Brink (19) and Campbell(22) observed that the potassium requirement for optimum growth of lambs was greater than 0.3% but not greater than 0.5%. Further, Campbell (22) maintained his lambs in an environment similar to that which existed in this experiment and fed the animals a similar semi-purified,high protein ration. Thus it seems that for the bovine a slightly higher level of ration potassium is required for rapid growth than for the ovine.

Carcass data. The carcass data of groups 3 and 4 were, respectively, (a) hot carcass weights 223.1 and 215.1 kg and (b) Government carcass grades, 4 choice, 2 good; 2 choice, 4 good. These values merely tend to emphasize the fact that the steers in groups 3 and 4 were relatively fat at termination of the trial.

Deficiency symptoms. Potassium deficiency manifested itself in group 1 in the form of partial to near complete

inanition compounded by a peculiar form of pica involving hair pulling and wood eating of boards on fences and in the barn. The latter phenomenon was probably due to a lack of energy intake. The hair coats were rough and staring, giving the animals a haggard appearance. There was, in the early part of the experiment, slight edema around the eyes and some steers had watery nostrils. Some of the steers grew very little, others lost weight and were very emaciated, depressed and reluctant to move around. General weakness near the latter part of the experiment was quite prominant in some animals. Forcing the animals to move quickly or run, resulted in an apparent incoordination of the hind legs with general wobbling of the hindquarters. Campbell (22) reported similar symptoms in lambs that were deficient in potassium. Poor growth and loss of weight have been reported as classic symptoms of potassium deficiency in many species (47, 41). Hughes and Ittner (47) reported general weakness in pigs as a prominant sign of potassium deficiency. Edema, though not a consistant sign, has also been observed in other species (66, 91).

Histological studies revealed no effect of ration potassium on skeletal (gluteal) muscle of steers in groups 1 and 2. The muscle fibres were normal except for few areas of very mild hyaline necrosis which could be expected in a superficial muscle "biopsy" of highly active muscles. Concentrations of potassium and sodium were not measured in these biopsy samples, however, from other published data (Campbell, 22) one might predict that potassium was lowered and sodium

increased in the gluteal muscles of the potassium deficient steers.

Serum electrolyte changes. The changes in serum electrolyte concentrations which occurred as a result of treatment are presented in Table 2 and Figure 3. The effects of potassium intake on serum calcium, phosphorus and sodium were not significant ($P>0.05$), although there was a tendency for calcium and phosphorus levels to increase as potassium intake decreased. However, with a reduction in ration potassium levels, there were significant ($P<0.01$) increases in serum magnesium and chloride levels while serum potassium levels were significantly ($P<0.01$) decreased.

The changes in serum potassium over the 110-day period were expected, since body intake and output of the cation are of major importance in regulation of serum potassium concentration. Due to an obligatory urinary loss of body potassium, any subnormal intake of the cation would eventually be reflected in the serum level. The reasons for the observed increases in serum levels of calcium and magnesium when the low potassium ration was fed are rather obscure. However, Barker (5) has observed that magnesium and calcium behave in the body in a similar manner, and thus one would probably expect a similar effect of potassium on these cations. On the other hand, Campbell (22) reported a non-significant increase in serum calcium and a decrease in magnesium in lambs when ration potassium was low. In contrast,

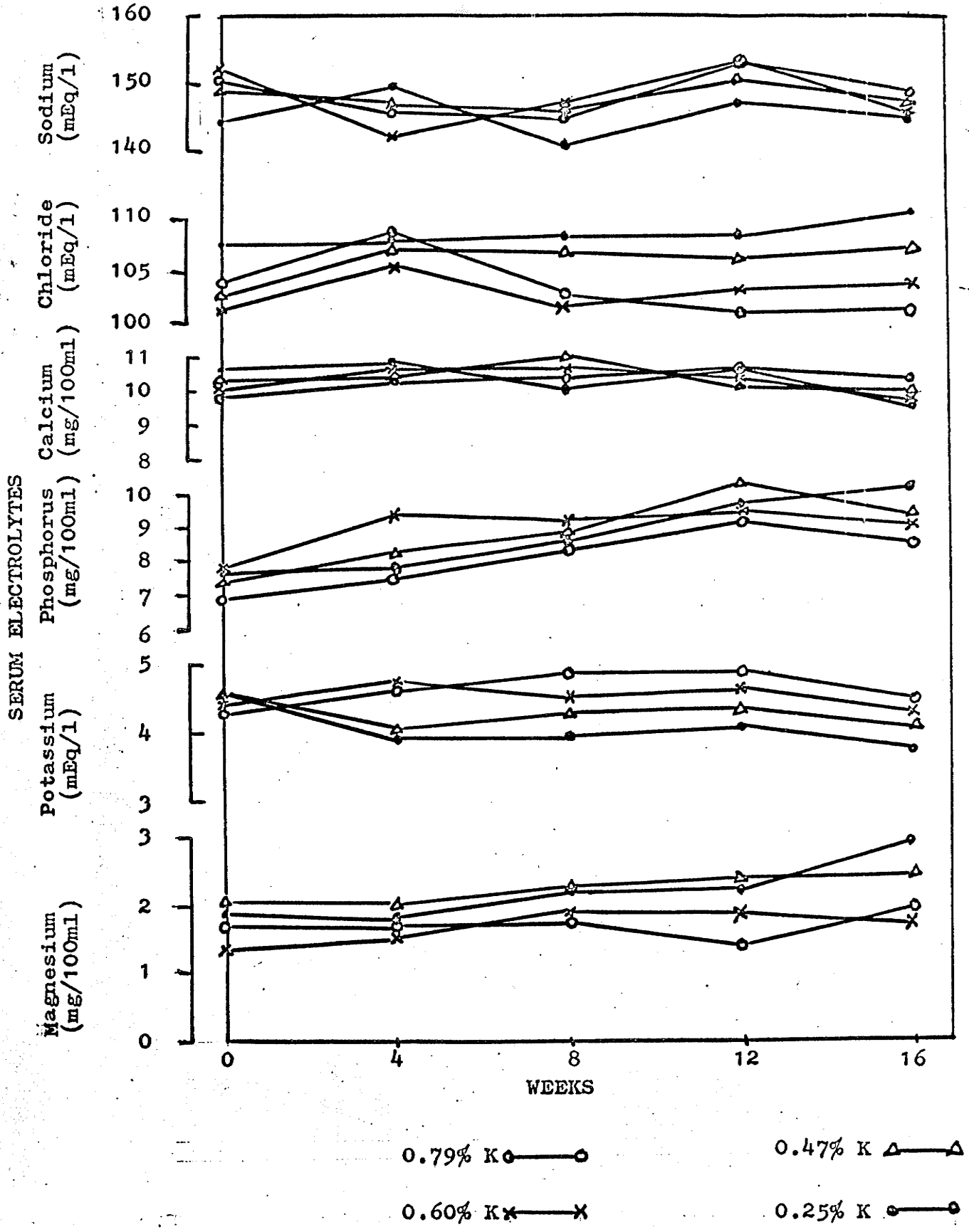

FIGURE 3. The effect of ration potassium on concentrations of serum electrolytes.

Fontenot et al. (36) observed that a high protein (34.4%) and high potassium (4.7%) ration depressed plasma levels of both magnesium and calcium when fed to lambs. Kunkel et al. (52) also reported that feeding a ration containing 5.0% potassium depressed plasma magnesium in ewes. Further, Gaunt as cited by Blood and Henderson (14), observed in humans that a high intake of potassium caused a depression of serum magnesium levels by over-stimulation of aldosterone secretion. However, this mechanism has not been proven to operate in ruminants (14). It appears that a high protein or high potassium ration will either interfere with magnesium absorption or promote secretion of this ion into the gut (36). It has been reported that serum chloride is frequently, but not invariably, depressed when there is a deficit of body potassium (91). Campbell (22), however, reported a trend towards an increase in serum level of chloride in lambs fed decreasing levels of potassium which is in general agreement with the serum chloride data of the present experiment. The observed non-significant ($P > 0.05$) increase in serum phosphorus concentration as ration potassium decreased is not in agreement with the data presented by Campbell (22) and Telle et al. (88). The former noticed a decreasing trend in serum phosphorus and the latter, no effect when potassium intake was low. Since potassium is intimately involved in the metabolism of a number of organic compounds, any severe reduction in potassium intake could possibly impair phosphorylation and

result in an increased plasma circulation of inorganic phosphorus. Further, the resultant cellular acidosis induced by a potassium deficiency (25) would probably promote phosphorolysis of the poorly ionized cellular organic phosphate esters, thereby releasing inorganic phosphate into the circulatory system.

Using body weight change as the criterion to measure the maintenance requirement of potassium for steers it can be seen that steers fed 0.25% ration potassium (group1) showed a slight increase in body weight, and at the same time showed signs of potassium deficiency. Some of these steers were however, so emaciated and weak, and inappetance was so severe that it was apparent a longer duration of dietary potassium deprivation would probably have resulted in some deaths. These observations indicate the inadequacy of this criterion to measure maintenance requirement of potassium. But since some workers have reported the correlation between low serum level of potassium and body potassium deficiency to be rather poor (49, 61, 92) it may be presumed justifiable to use body weight change as a rough measurement for evaluation of maintenance. However, in this study serum potassium values reflected in a general manner the body potassium status of each group of steers in that a low serum potassium was associated with symptoms of potassium deficiency. Thus one cannot regard the level of 0.25% ration

potassium as being adequate for maintenance of group 1 steers. A more reliable method of determining the minimum amount of a given element required to prevent body depletion and also maintain body weight is a metabolism trial. Such data for potassium will be provided in Experiment 111.

Effect of ration potassium upon pH, and sodium and potassium concentrations of rumen fluid and microbial activity of rumen contents. Rumen fluid data are shown in Table 3. The pH of rumen fluid was not significantly ($P > 0.05$) affected by treatment, however, the pH tended to increase as ration levels of potassium increased. The average pH values for the steers in groups 1 to 4 were 6.85, 6.99, 6.87 and 7.22, respectively. Campbell (22) also observed in lambs that as ration levels of K_2CO_3 increased, the pH of the rumen contents increased. Telle et al. (88) measured the pH of rations containing various levels of potassium salts (50% of potassium supplied by $KHCO_3$ and 50% by K_2CO_3) and observed a range in pH from 5.5 to 8.6 when the potassium levels ranged from 0.10 to 0.62%. Thus, it may be inferred from Telle's values that in this experiment the level of potassium in the ration influenced the pH of the rumen fluid. However, it was not possible to ascertain whether potassium per se was the influencing factor or whether it was the carbonate supplied by the K_2CO_3. Further, the alkalinity of ruminant saliva would greatly influence rumen ingesta pH since the samples were collected by oral intubation. Blake et al. (13)

TABLE 3. EFFECT OF RATION POTASSIUM UPON pH, AND SODIUM AND POTASSIUM CONCENTRATIONS OF RUMEN FLUID AND MICROBIAL ACTIVITY OF RUMEN CONTENTS.

Item	Treatment Group*** 1	2	3	4
pH	6.85 ± 0.20	6.99 ± 0.15*	6.87 ± 0.15	7.22 ± 0.18
Microbial activity**	30.6 ±11.15	50.2 ±11.62*	66.5 ± 8.81	73.3 ± 8.30
Sodium mEq/l	107.8 ± 4.3	118.5 ± 8.6	119.5 ± 1.7	122.3 ± 3.6
Potassium mEq/l	21.7 ± 2.3	25.9 ± 2.6	26.9 ± 2.7	25.9 ± 2.1

* Three animals in each treatment.

** Expressed as change in mm Hg/g dry matter/90 minutes.

*** Four animals in each treatment.

Treatment means within any item are not significantly ($P>0.05$) different.

have shown that pH values of rumen fluid are more variable and higher when samples of ingesta are collected orally by intubation than by rumen fistula. This effect is to be expected inasmuch as saliva contamination risk is greater and more variable. This effect may partly explain the lack of significance ($P > 0.05$) in pH values between the four groups of steers. Also the wide range of pH values (6.50 to 7.28) observed among the steers receiving the 0.25% potassium ration was probably due to the varying degree of inanition exhibited within that group. Meiske et al. (58) observed in steers that the pH of rumen fluid rose continuously (from 6.8 to 7.9) as starvation progressed over a period of 48 hours.

The average values for sodium in rumen fluid of steers in groups 1 to 4 were 107.8, 118.5, 119.5 and 122.3 mEq/l and for potassium 21.7, 25.9, 26.9 and 25.9 mEq/l, respectively. There were no significant ($P > 0.05$) differences among treatments for these two ions. It seems that in spite of the varying levels of potassium intake the steers were able to maintain levels of potassium in the rumen fluid within a certain physiological range. It is very probable that endogenous secretions of sodium and potassium were important in maintaining ruminal levels of these ions. Evidence to support this suggestion has been reported by Renkema et al. (73). These workers fed rations to cattle in which potassium levels were constant and sodium levels variable. It was observed that, regardless of the potassium-

sodium intake ratio, these ions were regulated so that the net equivalents of cations and consequently osmotic pressure within the intestinewere relatively unaltered.

The *in vitro* microbial activity data in Table 3 show a considerable but non-significant ($P > 0.05$) difference among treatments. There appeared to be a definite trend towards a decrease in microbial activity as potassium level in the ration was reduced (Figure 4). The average microbial activity values (expressed as change in mm Hg/g dry matter/90 minutes) for groups 1 to 4 were 30.6, 50.2, 65.5 and 73.3, respectively. The lack of a significant effect due to treatment might have been caused by both the small number of animals involved (four in each treatment) and the stomach intubation technique used in obtaining the rumen contents. McBee (56) reported that replicate samples taken from steers by this intubation method might give differences in *in vitro* microbial activity values of over 100%. That potassium can affect *in vitro* digestion has been shown by Hubbert *et al.* (100) where they reported the presence of potassium to be essential for accelerating *in vitro* cellulose digestion by rumen micro-organism. Also the low microbial activity exhibited by the group 1 steers may have been partially due to low feed intake. Meiske *et al.* (58) observed that starvation in steers adversely affected the ability of rumen microorganisms to digest cellulose *in vitro*.

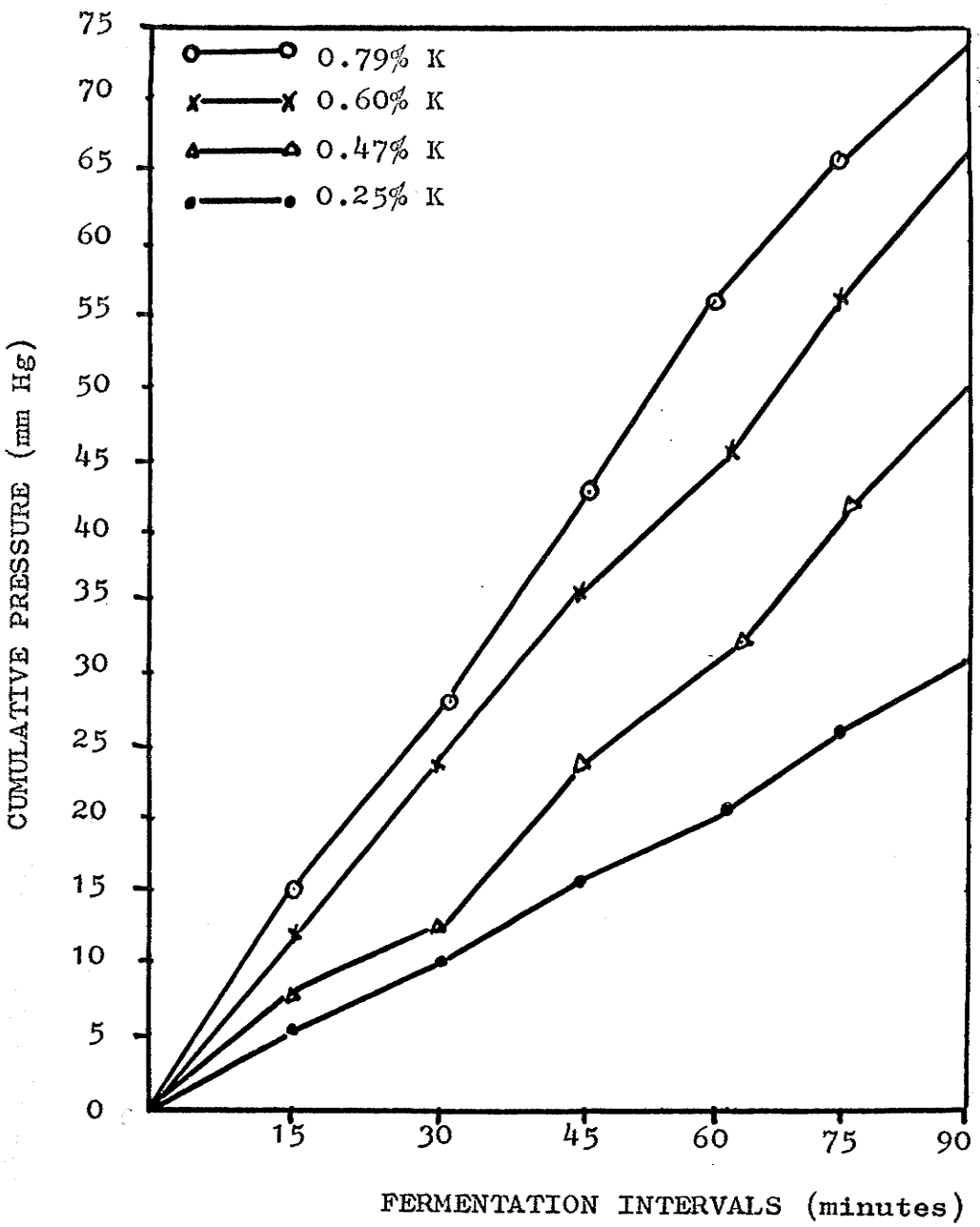

FIGURE 4. Effects of varying levels of potassium (0.25, 0.47, 0.60 and 0.79%) on <u>in vitro</u> microbial activity of rumen contents.

Since the ruminant and its rumen microorganisms show a symbiotic relationship for adequate nutrition, one might expect that major dietary changes which influence animal performance would also be reflected upon the metabolic activities of its microorgansims.

Wound healing. The apparent effect of ration potassium on wound healing is illustrated in Figure 5. The number of days for wound healing was significantly ($P<0.05$) increased when ration potassium was decreased from 0.47 to 0.25%. The average number of days required for wound healing in groups 1 and 2 were 20 and 12, respectively. However, some discretion must be used in interpreting these results since visual appraisal is a very crude method of estimating wound healing, and also there were limited numbers of observations(three steers in each group). It has been reported (23), however, that potassium is essential for protein synthesis and that wound healing is delayed in protein deficient or starved animals (55). The role of potassium in wound healing is, therefore, not clear. Since steers in group 1 were, apart from being potassium deficient, exhibiting various degrees of malnutrition and inanition, potassium deficiency *per se* could have caused a delay in wound healing but delayed healing could also have been the result of a secondary condition indirectly related to potassium deficiency.

Experiment 11

The results of this experiment are presented in Table 4.

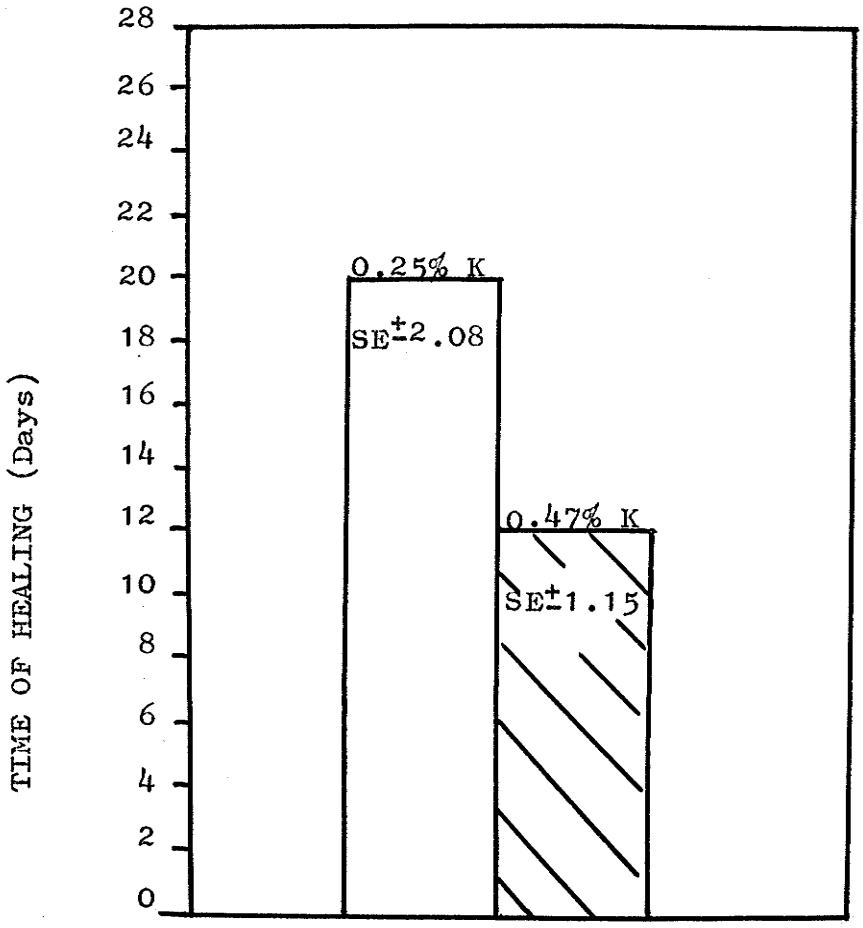

FIGURE 5. The apparent effect of ration potassium (0.25 and 0.47%) on wound healing time. An incision of approximately two inches in length was made into the right gluteal muscle of each animal. Three animals per treatment were used and the experiment was initiated after 112 days on the various rations. There was a significant ($P < 0.05$) treatment effect in length of time required for wound healing.

The average daily feed consumptions for steers (groups 1b and 2b) receiving the intraruminal injection of potassium (50 g) every second day were 4.12 and 4.39 kg, respectively, (Figure 6) compared to values of 2.17 and 3.78 kg for the control steers (groups 1a and 2a, respectively). Also steers in groups 1b and 2b achieved superior weight gains during the 22-day period (Figure 7). Group 1b steers showed a significant ($P < 0.05$) increase in average body weight gain (39.8 kg) over its control (group 1a steers lost an average of 12.4 kg). In addition group 2b steers gained an average of 23.6 kg compared to 9.0 kg by its control steers (group 2a). These values were not significantly ($P > 0.05$) different. Since the steers in groups 2a and 2b, unlike those in groups 1a and 1b, were initially in a higher physiological plane of body potassium it may be that partial potassium deprivation of only 22 days was insufficient to deplete body potassium stores of the control steers (group 2a) and produce the marked symptoms of deficiency observed in the control steers of group 1a. Further, the low potassium accretion by virtue of its effect on appetite probably aggravated tissue catabolism, the extent of which depended on the initial body potassium status of the steers in groups 1a and 2a.

During the subsequent 20-day period when all groups were receiving the same adequate potassium ration (0.60%), there was a general increase in feed consumption by the control animals (groups 1a and 2a) which was approximately double their previous intakes (Figure 6 and Table 4). Groups

TABLE 4. EFFECT OF POTASSIUM ON FEED CONSUMPTION(APPETITE), AND RUMEN AND SERUM CONCENTRATIONS OF SODIUM AND POTASSIUM WHEN STEERS WERE INJECTED INTRARUMINALLY WITH EITHER WATER OR POTASSIUM SOLUTIONS AND THEN FED A RATION CONTAINING 0.6% POTASSIUM.

Group***	Treatment*X	Av. Body wt. (kg)			Av.Daily feed(kg)	Serum electrolytes(mEq/l)		Ruminal electrolytes(mEq/l)	
		Orig.	Final	Change		Na	K	Na	K
PERIOD 1 (22 days depletion)									
1a	r + W	285.1	270.7	-12.4 ±8.9	2.17	136.7 ±3.5	3.50 ±0.57	67.0 ±21.0	43.5 ±14.0
1b	r + K	279.5	319.3	39.8[a] ±10.8	4.12	142.0 ±1.0	4.57[A] ±0.07	96.7 ±10.1	20.3 ±0.4
2a	r + W	351.9	360.9	9.0* ±17.0	3.78	141.0 ±0.9	3.96[a] ±0.02	113.7 ±9.2	21.9 ±2.0
2b	r + K	339.0	362.6	23.6 ±0.8	4.39	142.3 ±1.5	4.55 ±0.20	93.7 ±9.9	24.7 ±0.6
PERIOD 11 (20 days recovery)									
1a	R	270.7	309.6	38.9 ±16.5	5.17	133.7 ±5.8	4.30 ±0.20	84.3 ±31.9	50.9 ±21.6
1b	R	319.3	364.6	45.3 ±3.8	8.54	145.0 ±1.7	4.57 ±0.07	104.7 ±9.3	29.7 ±1.5
2a	R	360.9	423.4	62.5*[a] ±2.1	7.57	147.7 ±1.3	4.52 ±0.03	103.7 ±3.5	24.0 ±2.4
2b	R	354.6	380.0	25.4** ±10.9	7.12	140.3 ±2.7	4.25 ±0.25	93.0 ±10.0	24.3 ±6.3

* Weight gain of group 2a during Period 11 was significantly($P<0.05$) greater than during Period 1.

** Average of the two animals.

A Treatment means within same group number not showing the same superscript letter are significantly ($P<0.01$)different.

a Treatment means within same group number not showing the same superscript letter are significantly ($P<0.05$) different.

*** Groups 1a and 1b were initially in a lower physiological plane of body potassium than groups 2a and 2b.

*X Injections: W, water; K, potassium: Ration potassium: r, 0.25%; R, 0.60%.

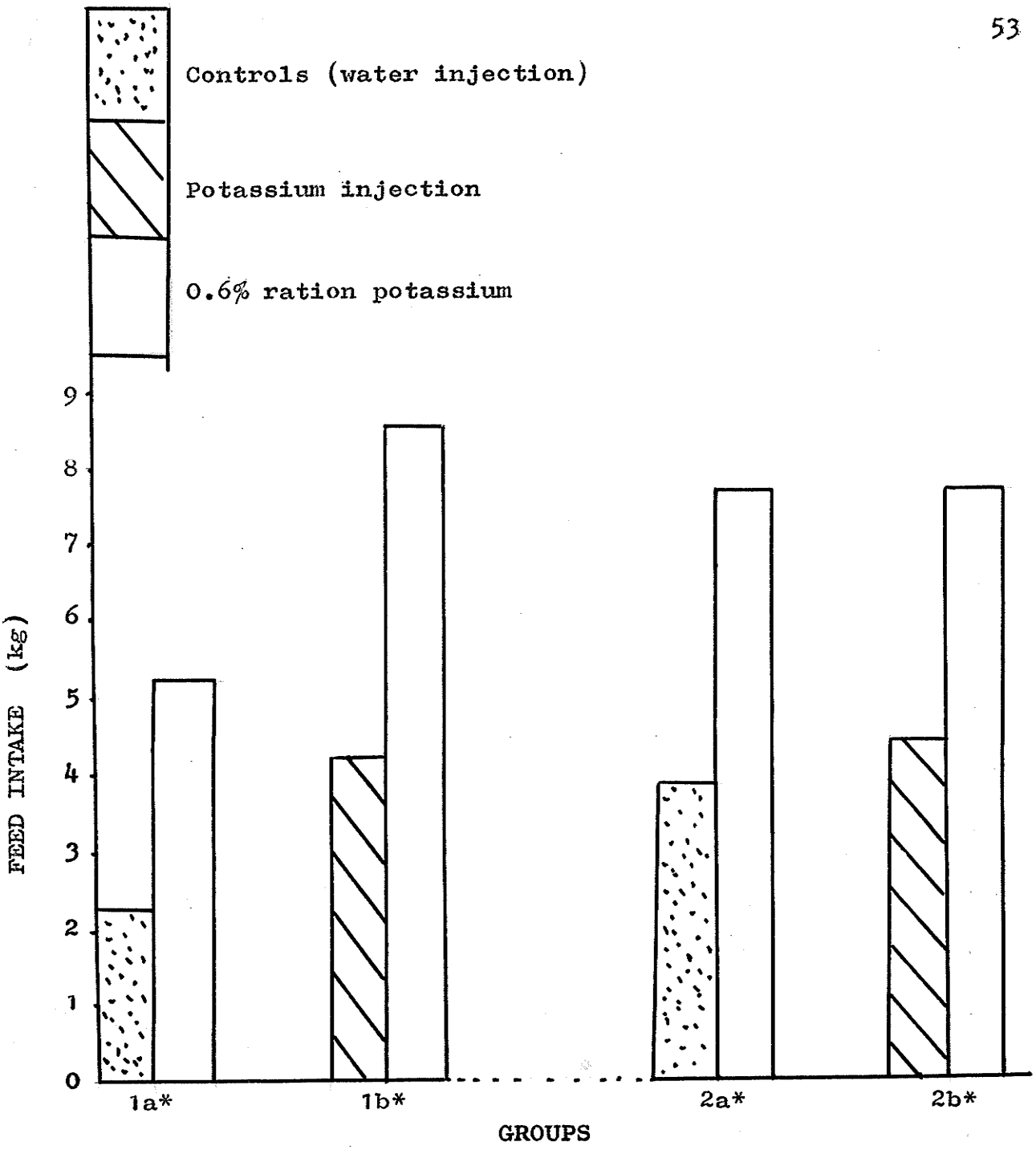

FIGURE 6. The effect of intraruminal injection of either water or potassium solution and 0.6% ration potassium on feed consumption.

* Steers in groups 1a and 1b were initially more depleted in potassium than steers in groups 2a and 2b.

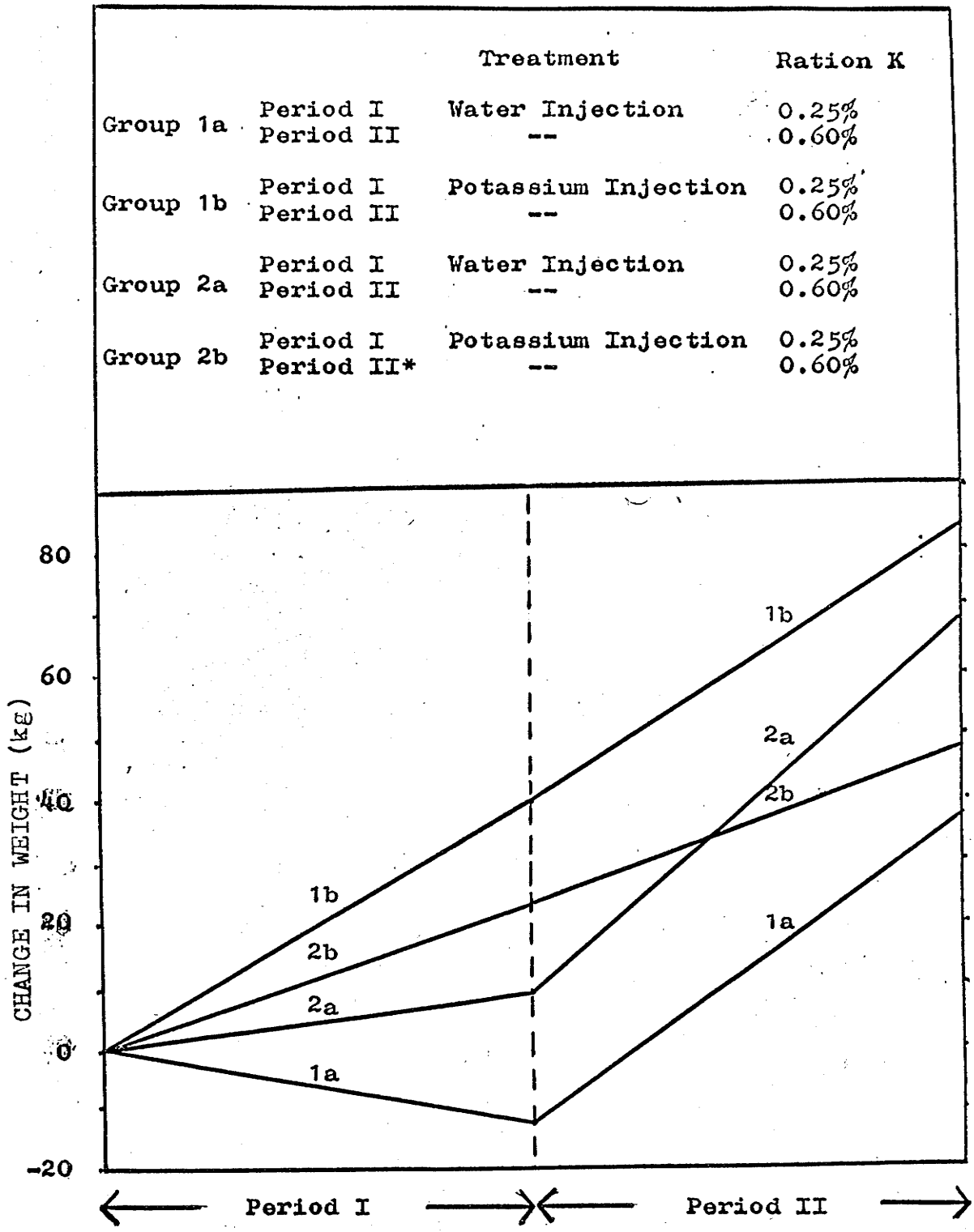

FIGURE 7. Effect of intraruminal injection of either potassium or water and 0.6% ration potassium on weight gain in steers.
* Av. of 2 animals.

1b and 2b consumed 5.17 and 7.57 kg daily while the control steers, (groups 1a and 2a), consumed 8.54 and 7.12 kg daily, respectively. The general increases in feed intake paralleled the average changes in weight during the recovery period. The total average changes in body weights during the 20-day period were 38.9, 45.3 and 62.5, 25.4 kg, respectively, for steers in groups 1a, 1b and 2a, 2b. One of the steers in group 2b developed a rumen fistula during the repletion period, which was a result of the injection received during the depletion period. Thereafter this steer exhibited poor appetite and lost variable amounts of its rumen ingesta. This might account for the significant ($P<0.05$) difference in weight gains between groups 2a and 2b. However, during the repletion period the overall rate of weight gain of the control steers was higher than that of the treatment steers. The average values for groups 1a and 2a were 2.0 and 3.1 kg body weight gain daily as compared to daily gains of 2.3 and 1.3 kg for groups 1b and 2b, respectively. The above findings are in general agreement with those of Cannon (23). He observed that protein depleted rats when fed a complete ration effectively recovered their initial loss of weight; but when potassium was omitted from the ration the rats displayed poor appetite and failed to gain weight adequately.

During the 22-day treatment period, the intraruminal injections of potassium and water did not influence the rumen fluid concentrations of either sodium or potassium (Table 4).

The treatments also had no influence on serum sodium levels, but serum potassium of group 1b (4.7 mEq/1) was significantly ($P < 0.05$) greater than that of group 1a (3.50 mEq/1). Similarly the serum potassium level of group 2b (4.55 mEq/1) was significantly ($P < 0.05$) greater than that of group 2a (3.96 mEq/1).

During the 20-day recovery period in which an adequate potassium ration was fed, there was no difference in the concentrations of either sodium or potassium in the rumen fluid or serum. It has been reported that potassium administration to an animal, whose body has been previously depleted of the element, will result in a progressive rise of serum potassium to a normal level and complete tissue repletion (43). Throughout this study serum potassium concentrations, to a certain extent, reflected the potassium status (depletion or repletion) of the animals. In general the results of this experiment also suggest that the extent of ration consumption depended upon adequate quantities of potassium entering the rumen either orally or via intraruminal injection. This suggests that the potassium effect on appetite is probably not mediated via the oral cavity. However, according to the hypothesis advanced by Denton and Sabine (29) it is possible that a change in pH and HCO_3 concentration of the arterial blood perfusing the taste buds is in some way responsible for the effect of low potassium intake on reducing appetite. These workers observed that sheep made sodium deficient by means of

a permanent unilateral parotid fistula showed a selective appetite for sodium salt solutions and rarely ingested any potassium chloride solution when it was included amongst the solutions offered. During the present study the control steers did display a preferential appetite (as measured by feed consumption during two different periods) for the 0.60% potassium ration (repletion period) over the 0.25% potassium ration (depletion period).

Experiment 111

For the purposes of this presentation nutrient balance is defined as total intake minus urinary and fecal excretions of the nutrient in question. Cumulative balance of a nutrient refers to the daily average periodic cumulative balance during six 5-day collection periods. Endogenous secretion of the nutrients in question by way of skin was not considered; consequently the values presented would be apparent balances.

Pre—experimental Period

During the 5-day pre-experimental period, the nine heifers lost an average of 1.9 kg in body weight. The three heifers later to be designated as the high potassium group gained an average of 1.5 kg, while those designated as the medium and low potassium groups lost an average of 2.8 and 0.6 kg, respectively. All the heifers, however, were in apparent positive nitrogen and water balance; the daily averages being 12.97 g nitrogen and 2.4 l of water. The average daily sodium lost by the three medium potassium heifers was 25.93 mEq while the average daily sodium retained by high and low potassium heifers was 1.69 and

2.09 mEq, respectively. The low potassium heifers were in negative potassium balance and a daily loss of 9.07 mEq of potassium occurred. In contrast the high and medium potassium heifers retained 93.52 and 4.90 mEq of potassium daily. The same quantity of feed was consumed by all heifers during this period. One reason for the considerable variability in balances observed above may be the different reaction among animals to the stresses imposed upon them due to catheterization and restriction of movement when retained in the metabolism crates.

Experimental Period

Weight gains and feed consumption. Equivalent feed consumption among the three treatments was maintained throughout the experiment. Each animal consumed daily 2.54 kg of dry matter (mineral-vitamin mixture was not included). The high treatment heifers consumed 15.3 l of water; which was significantly ($P<0.01$) greater than the 9.8 and 10.8 l/day consumed by the medium and low treatment heifers, respectively. Heifers receiving the low treatment lost an average of 1.8 kg body weight compared to increases of 3.9 and 4.9 kg by the medium and high treatment heifers, respectively (Table 5). The differences in weight change were not significant ($P>0.05$). Similarly, Blahd and Bassett (12) observed no change in weight in a human depleted of potassium over a period of 55 days. However, the author admitted that potassium depletion was not severe. In contrast, Campbell (22) fed lambs daily potassium levels

TABLE 5. AVERAGE DAILY INTAKE OF POTASSIUM, SODIUM AND NITROGEN AND AVERAGE DAILY EXCRETION OF POTASSIUM, SODIUM AND NITROGEN IN FECES AND URINE DURING A 5-DAY PRE-EXPERIMENTAL AND A 40-DAY EXPERIMENTAL PERIOD. AVERAGE BODY WEIGHT CHANGES DURING THIS PERIOD ARE ALSO INCLUDED.

Item	Pre-experimental**	Treatment		
		Experimental		
		High	Medium	Low
Potassium intake, mEq/day	439.55	1086.76	439.41	156.59
Urinary potassium, mEq/day	306.26	838.07^{C}	294.76^{B}	105.36^{A}
		77.12*	67.08*	67.26*
Fecal potassium, mEq/day	148.63	101.01^{C}	129.18^{B}	73.36^{A}
		9.29*	29.36*	47.10*
Sodium intake, mEq/day	256.15	256.67	256.04	256.14
Urinary sodium, mEq/day	187.12	143.82	168.67	148.92
		56.03*	60.88*	58.14*
Fecal sodium, mEq/day	76.39	63.14	68.26	69.40
		24.60*	26.66*	27.09*
Nitrogen intake, g/day	96.16	96.16	96.16	96.16
Urinary nitrogen, g/day	64.47	60.91	64.09	62.44
Fecal nitrogen, g/day	18.72	18.26	18.65	18.18
Av. initial wt., kg.		239.1	239.9	240.2
Av. final wt., kg		244.0	243.8	238.4
Av. wt. change, kg		4.9±1.84	3.9±2.73	-1.8±1.81

A,B,C Treatment means within an item, not showing the same superscript letter are significantly ($P<0.01$) different.

* Expressed as percentage of intake.

** Average of nine heifers

of 13.7, 56.1 and 94.4 mEq and observed a significant ($P < 0.01$) loss in weight (4.18 kg) in the lambs receiving the lowest level of potassium over a 30-day period. Campbell also reported a decrease in feed intake by the lambs receiving the lowest level of potassium which may explain the relatively large loss of weight. Data presented by Smith and Meyer (80) emphasized the fact that low dietary potassium will decrease the efficiency of energy utilization in rats. Further, Leach et al. (53) observed that, when chicks were fed diets in which potassium was limited, the grams of gain per gram of potassium consumed was markedly uniform at each level of potassium intake. Thus, the loss of weight observed in the heifers receiving the low level of potassium, though not significant, was probably the result of inadequate dietary potassium.

Potassium. Average daily potassium balance for each 5-day collection period, and cumulative balance during six 5-day collection periods are illustrated in Figure 8. The high treatment heifers displayed a persistent and pronounced positive daily balance. The medium treatment heifers showed a slight positive balance, while the heifers receiving the low level of potassium were, with the exception of the second and third collection periods, persistently in negative balance. Over the experimental period, the high potassium heifers retained an average of 147,67 mEq of potassium daily, which was significantly ($P < 0.01$) greater than a daily retention of 15.47 and a daily loss of 25.13 mEq of potassium by

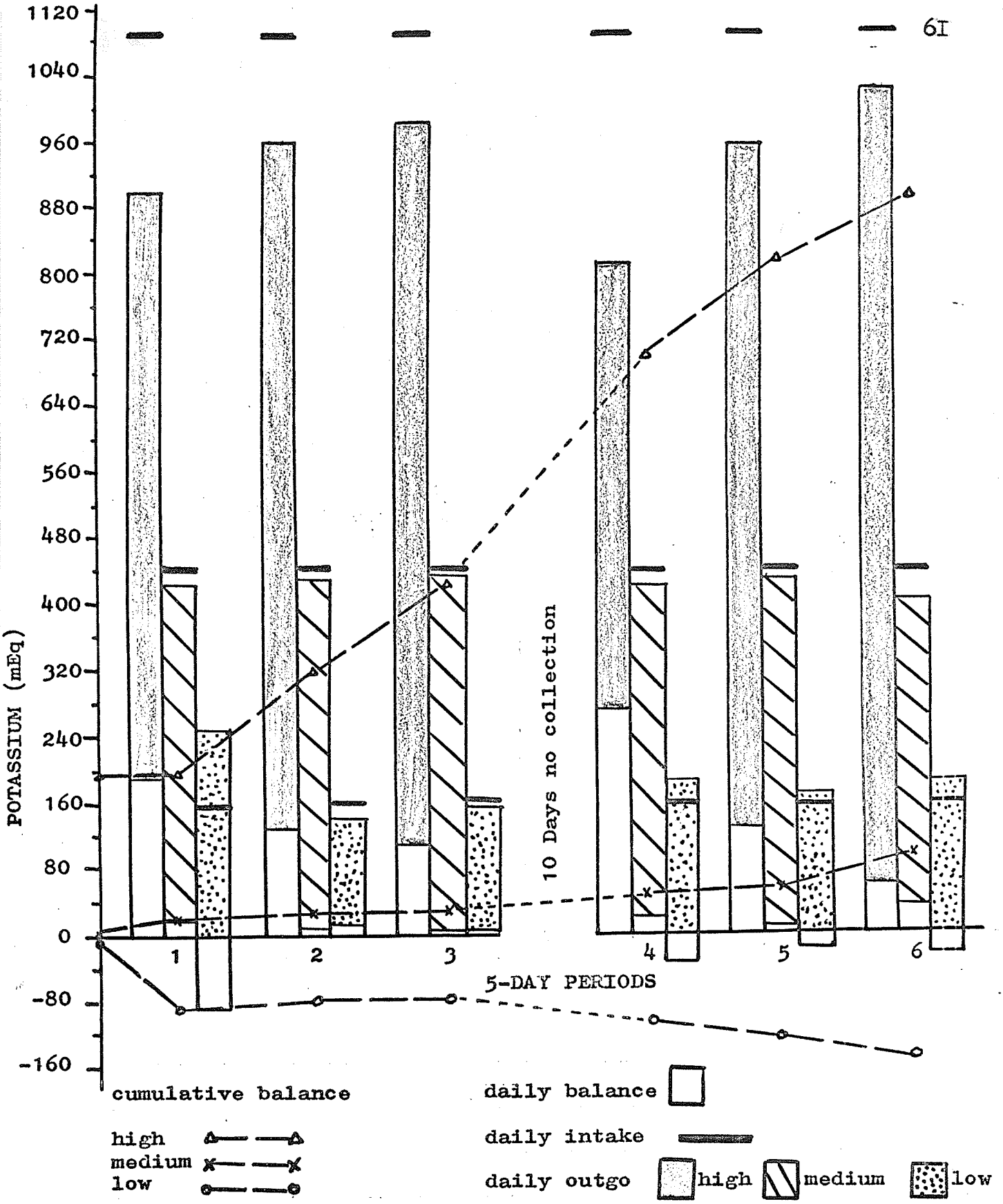

FIGURE 8. The effect of potassium intake upon daily and periodic average cumulative apparent potassium balances of heifers receiving 1086.75 (high), 439.41 (medium) and 156.59 (low) mEq potassium daily.

the medium and low treatment heifers, respectively. The initial large loss of potassium observed when the potassium intake of the low treatment heifers was abruptly changed from the adequate dietary pre-experimental potassium level (439.41 mEq/day) to the low experimental level (156.59 mEq/day) has been observed by other workers (22, 30). The renal mechanisms promoting the excretion of potassium and the conservation of sodium are very active in cattle (2), and when potassium is withdrawn from the diet aldosterone secretion is also lowered. Therefore, due to a negative feedback mechanism, a time lag may be involved before any change in kidney excretion of potassium can be effected through the action of the adrenoglomerulatropic hormone whereby the secretion of aldosterone is decreased. However, the physiological mechanisms initiating and maintaining potassium regulation are not clear (2).

The treatment effect upon cumulative potassium balance was very marked. The high treatment heifers retained an average of 886.03 mEq of potassium over the six 5-day collection periods. This value was significantly ($P<0.01$) greater than the 92.82 mEq of potassium retained by the medium potassium heifers and the 150.80 mEq of potassium lost by the low potassium heifers. These data suggest that an obligatory loss of potassium occurs when dietary potassium is low. Blahd and Bassett (12) observed a similar obligatory loss in a subject depleted of potassium and stated " that prolonged restriction could lead to severe deficit".

The average daily urinary and fecal excretions of potassium during the balance period are presented in Figure 9. As expected, there was a marked effect of treatment on urinary excretion of potassium. The average daily urinary excretions of potassium for the high, medium and low heifers were 838.07, 294.76 and 105.36 mEq, respectively, during the six 5-day collection periods (Table 5). These were significantly ($P < 0.01$) different. The renal mechanism is designed primarily to prevent hyperkalemia. The very high urinary excretion of potassium by the high potassium group may have been due partly to a diuresis produced by increased hypertonicity of the potassium load in the glomerular filtrate. The preferential exchange of sodium ions for potassium instead of hydrogen ions in the ion exchange mechanism of the distal renal tubules would also favour a high urinary potassium excretion. In contrast the low urinary potassium excretion observed for the low potassium heifers may be the result of a suppressed secretion of potassium with a concomitant increase in hydrogen ion secretion in the distal renal tubules. Further, it has been reported that in dogs and rats the activity of aldosterone is increased or decreased by a high or low potassiun intake, respectively (67). The fact that the medium treatment group, unlike the low potassium group, maintained a slightly positive potassium balance suggests that the daily obligatory urinary loss of potassium for heifers used in this experiment was greater than 105.36 but less than 294.76 mEq.

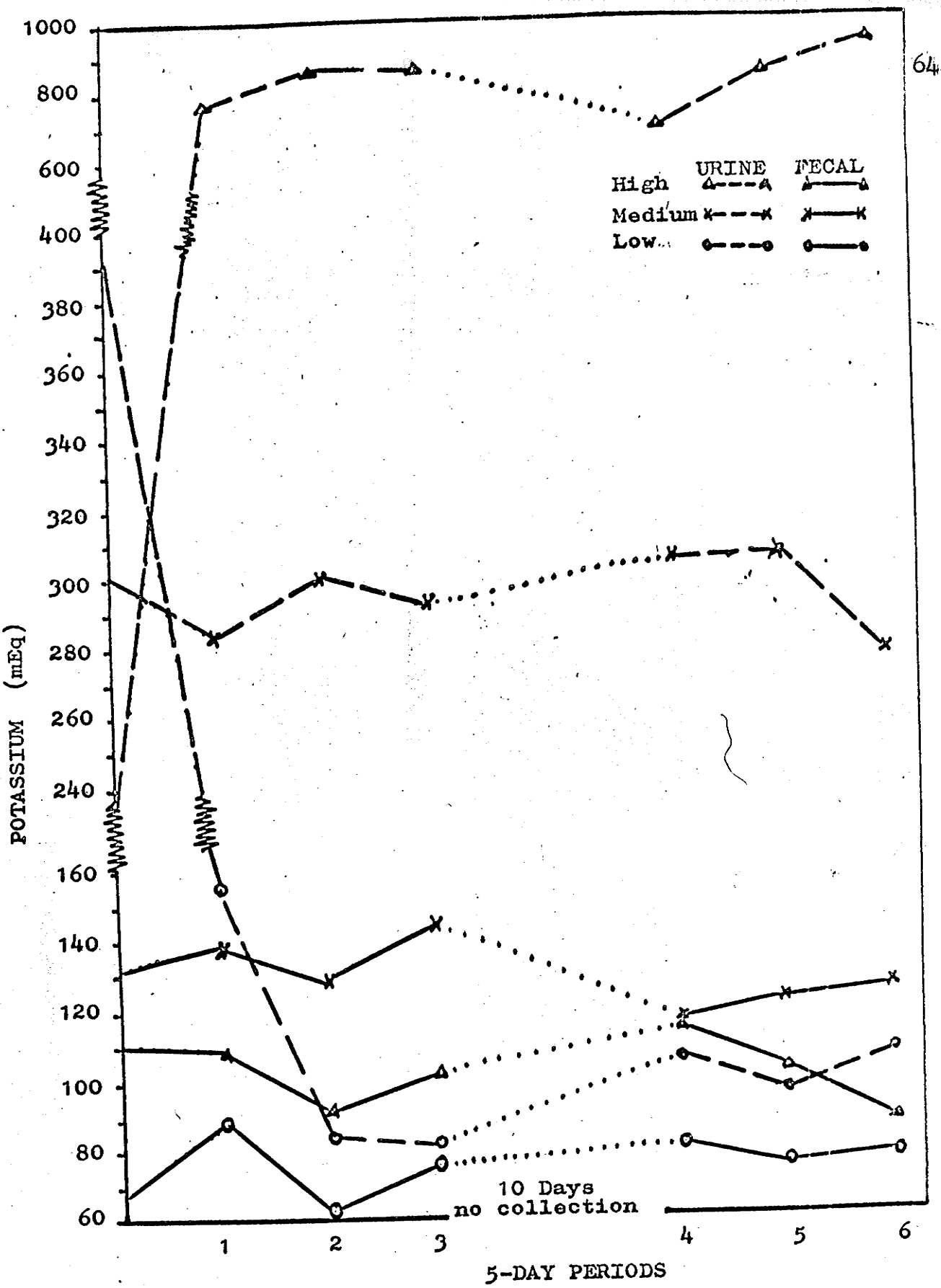

FIGURE 9. The effect of potassium intake upon daily fecal and urinary excretion of potassium of heifers receiving 1086.75 (high), 439.41 (medium) and 156.59 (low) mEq potassium daily.

The effect of treatment on fecal potassium excretion was not of the same magnitude as observed for urinary excretion of the ion (Figure 9). During the balance period, the average daily fecal excretions for the low, medium and high treatment heifers were 73.36, 129.18 and 101.01 mEq, respectively. These values, although significantly ($P<0.01$) different, closely approximated the pre-experimental values which for the low, medium and high potassium heifers, were 67.72, 132.58 and 110.14 mEq/day, respectively. This phenomenon of intestinal regulation of potassium and sodium ions to maintain osmotic pressure within the digestive tract has already been mentioned in the discussion of Experiment 1. However, as was expected, apparent absorption of potassium increased as ration potassium increased.

An attempt was made to evaluate fecal and urinary excretion of potassium as a percentage of intake (Table 5), as was done by Campbell (22). This manner of expression creates an erroneous interpretation of the data. For example, in the present study it would indicate that when ration potassiun is low, absorption of the ion is also low, which probably is not true. The major contributing factor to this misinterpretation is the inclusion of endogenous potassium, arising from saliva and intestinal secretions into the upper part of the digestive tract, in the fecal potassium. Saliva is the most important source of endogenous potassium and sodium entering the digestive tract. For example a 220 kg heifer

would normally secrete about 75 to 100 mEq of potassium daily into the rumen. Since a certain quantity of the endogenous potassium is reabsorbed, fecal potassium would include a large or variable portion of the whole. Thus the digestion coefficient values for either sodium or potassium can be misleading.

Sodium. Data concerning sodium balance are illustrated in Figure 10. The average daily retention of sodium over the experimental period was 49.71, 19.12 and 38.31 mEq for the high, medium and low potassium groups, respectively. The marked negative balance observed in the low potassium heifers during the first collection period was probably due to the sudden drop in potassium intake, and thus the unavailability of potassium in the renal ion exchange mechanisms whereby sodium is reabsorbed and potassium secreted. The rate of exchange by which lumen sodium ions are reabsorbed and potassium ions are secreted is directly dependent on the availability of both ions (76). Additional sodium losses were probably prevented by the replacement of potassium by hydrogen in the renal exchange system (7), and a positive balance was thereafter maintained by the low potassium heifers. A negative sodium balance of 25.93 mEq/day was observed in the medium potassium group during the pre-experimental collection period. This deficit was markedly reduced during the first 5-day experimental period and the group then maintained a persistent positive sodium balance.

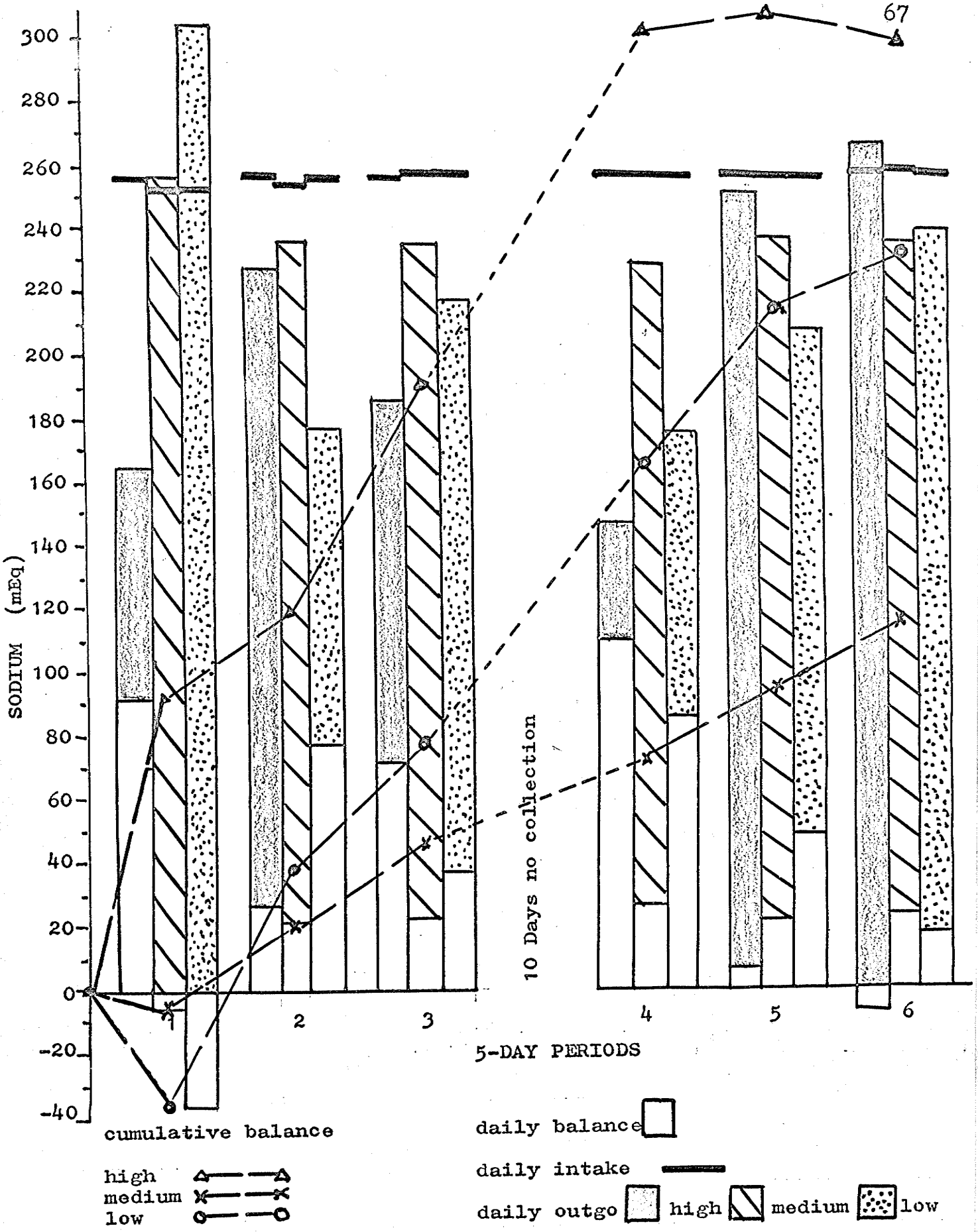

FIGURE 10. The effect of potassium intake upon daily and periodic average cumulative apparent sodium balances of heifers receiving 1086.75 (high), 439.41 (medium) and 156.59 (low) mEq potassium daily.

The high potassium heifers remained in daily positive balance until the last 5-day period when there was a slight loss of 8.62 mEq of sodium daily. Such a loss might have been induced by an increased load of potassium in the golmerular filtrate which is somewhat suggested in Figure 9 by a rise in urinary excretion of potassium. This would produce a temporary diuresis and thus an increase in urinary sodium excretion, which is suggested by the urinary sodium excretion data presented in Figure 11. The cumulative sodium balance values for the high, medium and low potassium heifers were 298.25, 114.74 and 229.87 mEq, respectively. Urinary and fecal excretions of sodium during the 5-day collection periods are illustrated in Figure 11. Potassium treatment did not significantly ($P>0.05$) influence the urinary excretion of sodium (Table 5). However, Campbell's (22) data suggested an inability of the kidney to conserve sodium when lambs were fed low potassium. Daily fecal values for sodium excretion during the experimental period, were approximately the same regardless of treatment. The daily values for the high, medium and low groups were 63.14, 68.26 and 69.40 mEq of sodium, respectively. In contrast, Campbell (22) observed in lambs, that sodium absorption increased as potassium intake decreased. In this experiment, however, ration potassium did not appear to greatly influence the absorptive and excretory mechanisms regulating body sodium.

Nitrogen. The average 5-day and cumulative balance data

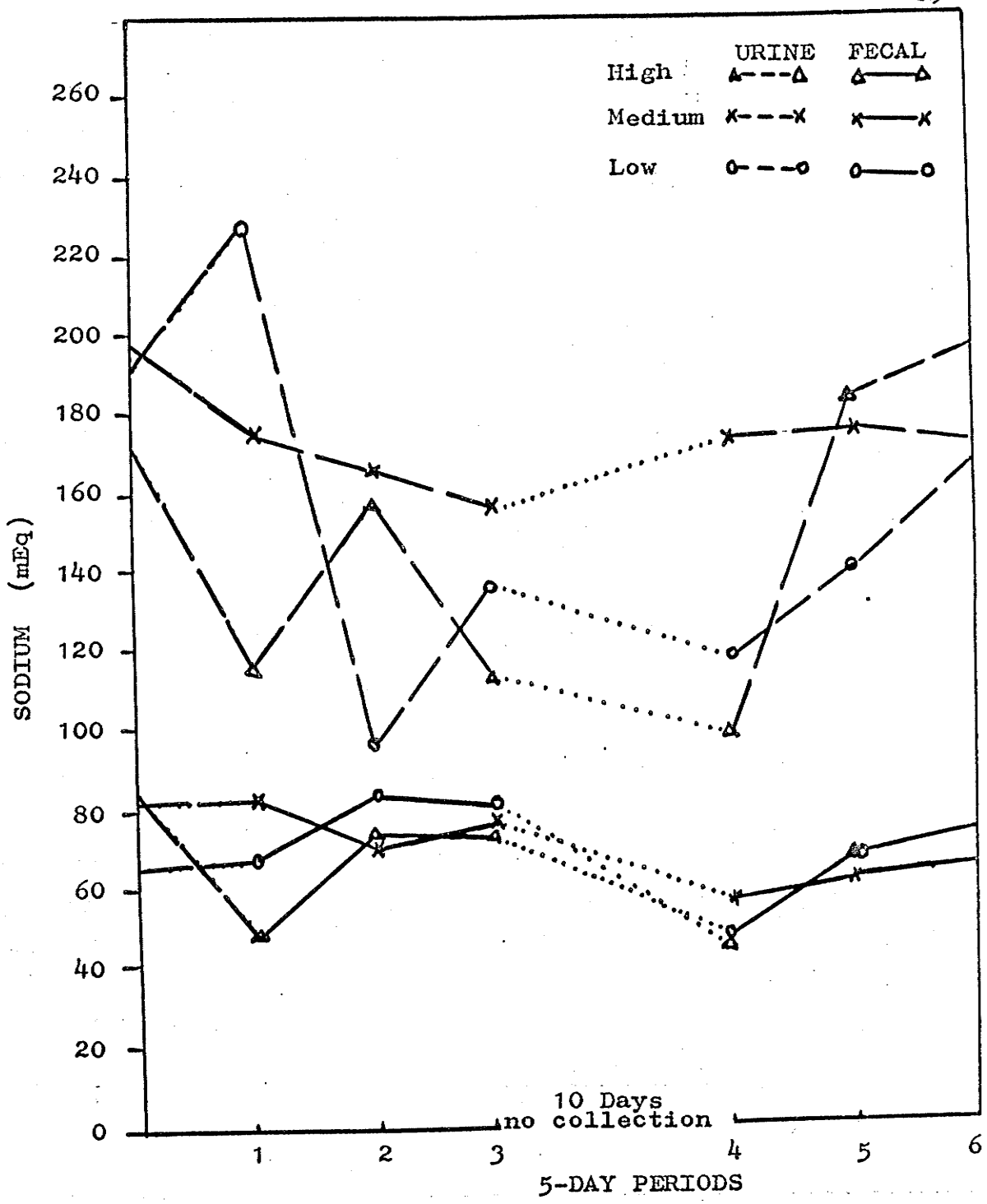

FIGURE 11. The effect of potassium intake upon daily fecal and urinary excretion of sodium of heifers receiving 1086.75 (high), 439.41 (medium) and 156.59 (low) mEq potassium daily.

are illustrated in Figure 12. Each treatment group remained in persistent daily positive nitrogen balance. The average daily retention of nitrogen during the experimental period for the heifers receiving the high, medium and low levels of potassium were 17.00, 13.43 and 15.54 g, respectively. The values were not significantly ($P>0.05$) different. However, during the pre-experimental period and the first three 5-day collection periods, the heifers receiving the low potassium ration appeared to retain more nitrogen than heifers receiving the medium potassium ration. Thus, it seems that when dietary potassium is not drastically lowered, there is no immediate apparent effect of this ion on nitrogen balance. Blahd and Bassett (12) also observed no change in nitrogen balance in man, when potassium depletion was induced by dietary means. In contrast, Campbell's (22) data suggested a marked reduction ($P<0.05$) in nitrogen retention by lambs receiving a low level (13.7 mEq/day) of dietary potassium. However, the lambs exhibited varying degrees of inanition which was probably the primary factor affecting nitrogen retention. Cannon (23) also demonstrated that nitrogen balance was less positive in potassium depleted than in control rats. Both these workers (22, 23) maintained their animals in a much more severe state of potassium depletion than were the heifers receiving the low potassium ration.

The cumulative data show the average retention of nitrogen for the high, medium and low potassium groups to be 101.98, 80.55 and 93.21 g, respectively. During the last

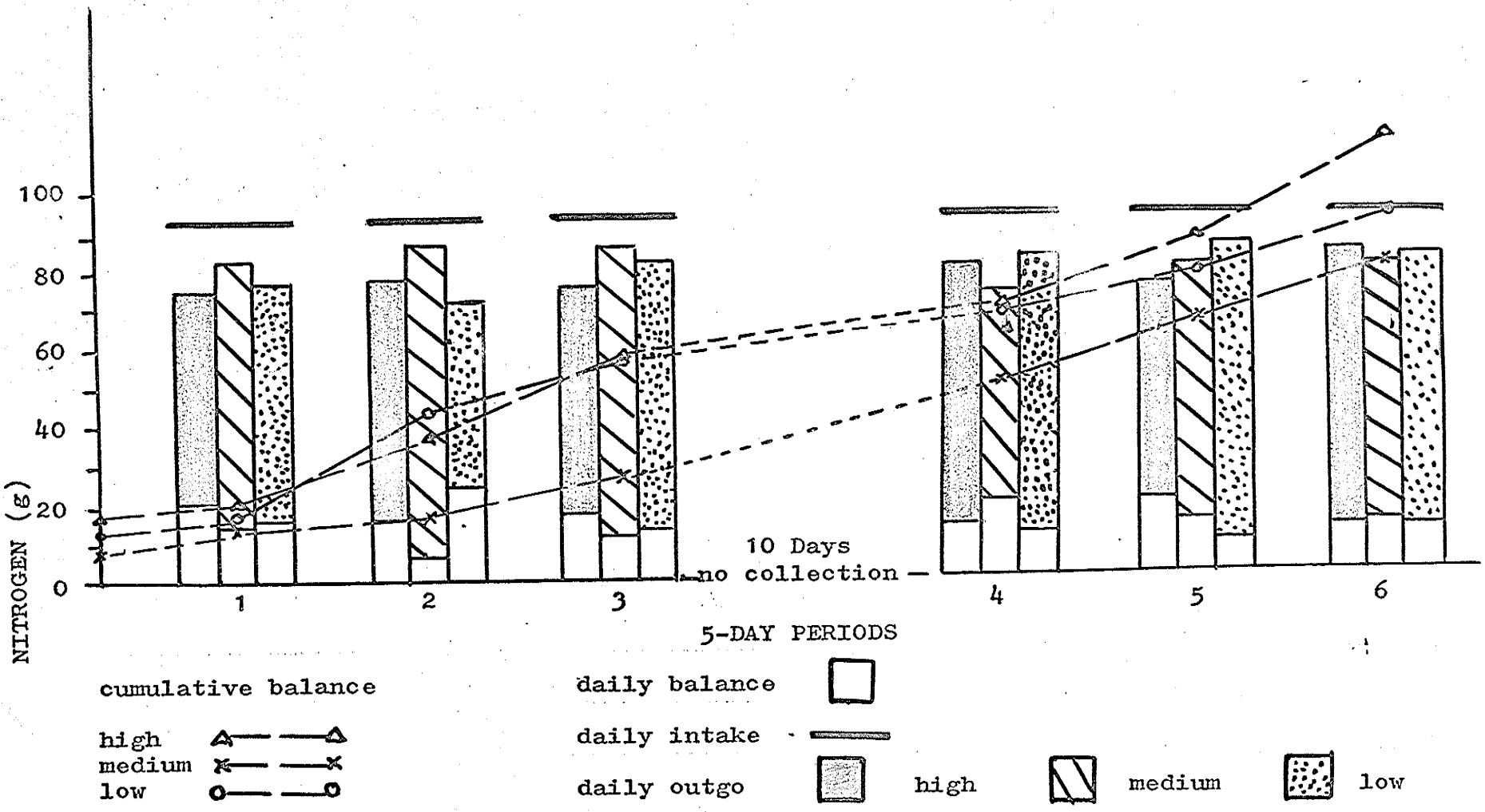

FIGURE 12. The effect of potassium intake upon daily and periodic average cumulative apparent nitrogen balances of heifers receiving 1086.75 (high), 439.41 (medium) and 156.59 (low) mEq potassium daily.

4 collection periods the rate of increase in cumulative nitrogen balance for the high and medium potassium heifers was slightly greater than that of heifers receiving the low potassium ration. Thus one might postulate that, over a longer experimental period, the magnitude of nitrogen retention for the low potassium group would eventually be lower than that of the heifers receiving the high and medium potassium rations.

The average daily urinary and fecal excretion of nitrogen during the experimental period suggest no effect of treatment on the absorptive or excretory mechanisms (Table 5). Welt et al. (91) reported that it is not known whether the reduced positivity of nitrogen balance observed in chronic potassium depletion is the result of decreased absorption of nitrogen from the gastro-intestinal tract or increased urinary excretion. The results of this experiment give no indication as to mechanisms affecting nitrogen balance.

Urinary ammonia and urea excretion. The effects of ration potassium on urinary excretion of ammonia and urea are presented in Table 6. Urine ammonia excretion increased as ration potassium decreased. The average daily urine ammonia excretions during the last 5-day period were 0.47, 3.74 and 7.11 g for the high, medium and low potassium heifers, respectively. These values were significantly ($P<0.01$) different. The phenomenon of increased excretion of ammonia in potassium depletion has been previously reported (91). The cause has been ascribed to increased renal glutaminase (26,91) and carbonic anhydrase activity(91), thereby favouring hydrogen-

TABLE 6. EFFECT OF RATION POTASSIUM ON DAILY WATER INTAKE, NITROGEN BALANCE, URINE AMMONIA AND UREA EXCRETION DURING PRE-EXPERIMENTAL AND THE LAST 5 DAYS OF THE EXPERIMENTAL PERIOD.

Treatment	Group	Water Intake (l)	NITROGEN				UREA-N EXCRETION(g)		NH_3-N EXCRETION (g)	
			Intake (g)	Fecal (g)	Urine (g)	Apparent Retention (g)	Total N to urea-N Excretion Ratio	Daily	Total N to NH_3-N Ratio	Daily
Pre-experimental Period										
Medium	I	13.46	96.16	18.28	60.66	17.22	1.10	66.88 ±19.72	0.056	3.40 ±0.35
Medium	II	9.63	96.16	18.90	67.61	9.65	0.97	65.60 ±17.24	0.062	4.17 ±0.20
Medium	III	9.64	96.16	18.97	65.14	12.05	0.97	63.05 ±19.47	0.058	3.78 ±0.59
Experimental Period										
High	I	16.13	96.16	18.40	65.32	12.44	1.31	85.71 ±20.52	0.007	0.47^{A} ±0.15
Medium	II	11.54	96.16	18.11	62.77	15.28	1.01	63.13 ± 4.46	0.060	3.74^{B} ±0.43
Low	III	12.65	96.16	17.96	64.38	13.82	0.81	51.83 ± 4.60	0.110	7.11^{C} ±0.51

A,B,C Treatment means within an item not showing the same superscript letter are significantly ($P<0.01$) different.

sodium ion exchange in the distal renal tubules. In contrast, urea excretion was not significantly ($P>0.05$) influenced by ration potassium. However, there was a tendency for urea values to increase as ration potassium increased (Table 6).

Water. The data illustrated in Figure 13 indicate that apparent water balances, both daily and cumulative, were persistently positive. The average daily quantities of water consumed during the six 5-day periods were 15.3, 9.8 and 10.8 l for the high, medium and low potassium heifers, respectively. Heifers receiving the high potassium ration consumed significantly ($P<0.01$) more water than the other two groups, also the volume of urine excreted by the high potassium group was significantly ($P<0.01$) greater than that of the other treatment groups. The daily water consumption values were 10.7, 5.4 and 6.7 l for the high, medium and low potassium heifers, respectively. In spite of the difference in water intake the daily water retention values were 2.2, 2.2 and 2.1 l for the high, medium and low potassium groups, respectively, and they were not significantly ($P>0.05$) different. The polydipsia observed in heifers receiving the high potassium ration may have been secondary to the polyuria induced by diuretic action of the potassium ions. However, the kidney regulated urine excretion in a compensatory manner to maintain water retention. Many workers, on the other hand, as indicated by Welt et al. (91), have associated polydipsia and polyuria with potassium depletion. In contrast Campbell (22) observed no effect of ration potassium on water consumption in lambs. Bressani and Braham (18) reported in dogs that nitrogen retention, and

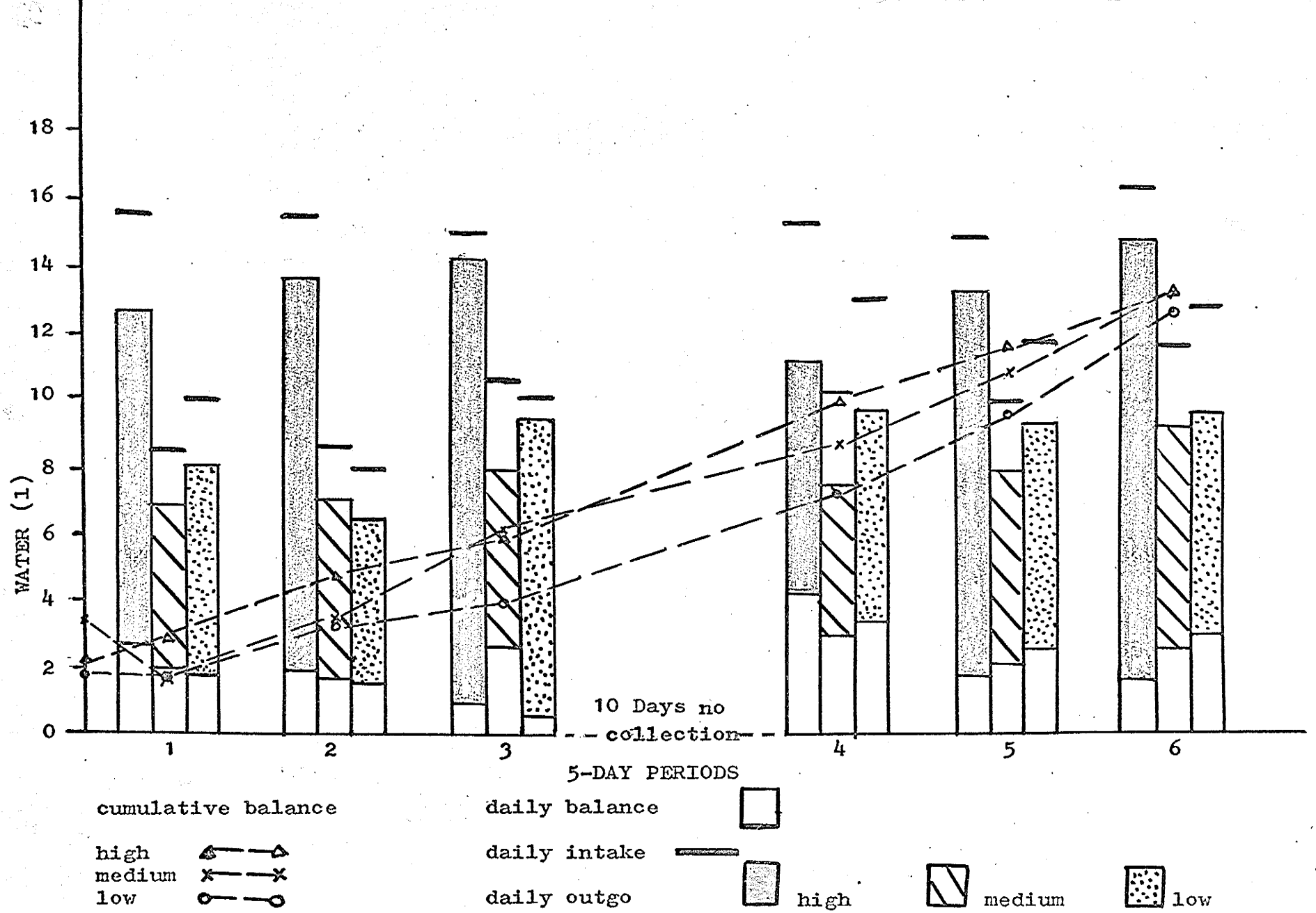

FIGURE 13. The effect of potassium intake upon daily and periodic average cumulative apparent water balances of heifers receiving 1086.75 (high), 439.41 (medium) and 156.59 (low) mEq potassium daily.

therefore urinary ammonia and urea excretions, was influenced by water intake. The data presented in Table 6 suggest the contrary.

Ration digestibility. Ration potassium had no significant ($P > 0.05$) effect on apparent digestion coefficients for energy, dry matter, crude fibre, nitrogen and ether extract (Table 7). Campbell (22) also observed no differences in ration digestibility when lambs were fed a basal ration similar to that used in the present study and containing various levels of potassium. In ruminants, the microorganisms in the rumen have an effect upon digestibility. In addition to aiding digestion through the breakdown of carbohydrates, protein and fats, these microbes are also responsible for the syntheses of amino acids, fatty acids and certain vitamins. Meiske et al. (58) have shown that inanition adversely affected rumen microorganisms and thus decreased in vitro cellulose digestion. Further, Hubbert et al. (46) demonstrated an in vitro need for potassium by rumen microorganisms. However, as little as 3.9 mEq of potassium/l satisfied minimum in vitro requirements for the rumen microorganisms. Since in Experiment 111 feed intake was equal for all groups, and results obtained in Experiments 1 and 11 indicate no effect of potassium treatment on potassium concentration of rumen fluid, one may thus assume that microbial digestion of this ration was not influenced by treatment. This would partly, if not completely, account for the lack of differences in ration digestibility among groups.

TABLE 7. EFFECT OF RATION POTASSIUM UPON APPARENT ENERGY, DRY MATTER, ETHER EXTRACT, CRUDE FIBRE AND NITROGEN DIGESTIBILITY.

Item	Treatment		
	High	Medium	Low*
Energy, %	68.2 ± 1.35	68.4 ± 1.15	64.7 ± 1.39
Dry matter, %	64.2 ± 1.78	64.0 ± 1.63	64.7 ± 1.39
Nitrogen, %	80.9 ± 1.93	81.2 ± 0.71	81.4 ± 0.18
Crude fibre, %	35.7 ± 2.97	36.2 ± 4.50	36.2 ± 7.79
Ether extract, %	90.7 ± 0.20	90.3 ± 0.50	90.2 ± 1.51

* Average of two animals.

Treatment means within any item are not significantly ($P>0.05$) different.

Serum Electrolytes. The effect of potassium intake upon serum electrolytes are presented in Table 8. The serum potassium concentration (4.09 mEq/1) of the low potassium heifers was significantly ($P < 0.05$) lower than those of the medium (4.35) and high (4.54) potassium heifers. However, all the potassium levels were within what is considered a normal range. Intracellular potassium reserves may have prevented a more drastic decrease in serum potassium levels of the low potassium heifers (96). It has been suggested that, when potassium loss due to deprivation is unaccompanied by excessive sodium intake, there is little tendency for hypokalemia even though catabolism is actively taking place (62). However, in all three of the present experiments sodium intake did not seem to influence the effect of ration potassium on serum potassium levels. In contrast to serum potassium, the concentration of phosphorus for the low treatment heifers (10.14 mg/100 ml) was significantly ($P < 0.05$) higher than the values of 7.85 and 8.28 observed for the medium and high treatment heifers, respectively. This effect of low potassium intake on serum phosphorus is in agreement with similar data from Experiment 1. No significant ($P > 0.05$) differences were found in the serum concentrations of sodium, chloride, calcium and magnesium. With the exception of the phosphorus and magnesium, the serum electrolyte changes are similar to those reported by Campbell (22). This worker observed in lambs a concomitant decrease

TABLE 8. EFFECT OF RATION POTASSIUM UPON VARIOUS SERUM ELECTROLYTES.

Item	Treatment		
	High	Medium	Low*
Serum potassium, mEq/l	$4.54^{a} \pm 0.09$	$4.35^{ab} \pm 0.08$	$4.09^{b} \pm 0.04$
Serum sodium, mEq/l	148.00 ± 2.60	143.00 ± 3.06	148.00 ± 1.29
Serum chloride, mEq/l	106.56 ± 1.03	105.34 ± 0.52	107.82 ± 1.29
Serum calcium, mg/100ml	9.12 ± 0.17	9.18 ± 0.35	9.45 ± 0.00
Serum magnesium, mg/100ml	2.08 ± 0.06	2.56 ± 0.22	2.45 ± 0.16
Serum phosphorus mg/100ml	$8.28^{a} \pm 0.37$	$7.85^{a} \pm 0.33$	10.14 ± 0.14

* Average of two animals

a,b Treatment means within an item not showing the same superscript letter are significantly ($P < 0.05$) different.

in serum phosphorus and magnesium levels when ration potassium was low.

The data of this experiment suggest a daily potassium intake of 439.41 mEq to be adequate for maintenance of yearling heifers (average weight 239.9 kg). The dietary potassium level of 156.59 mEq/day was inadequate. In conclusion it is of interest to add that the results of this experiment support the argument presented in Experiment 1 that 0.25% potassium ration (169.26 mEq of potassium daily) received by group 1 steers was below the potassium maintenance requirement.

Raw data and a list of mean squares illustrating the statistical analyses are presented in the appendix.

SUMMARY

A 110-day feeding trial, a 42-day appetite trial and a 40-day metabolism trial were conducted to study the role of potassium in bovine nutrition. Yearling Hereford steers were used in the first two trials and yearling Hereford heifers in the last trial. A semi-purified ration to which various levels of potassium (K_2CO_3) were added was used in each trial. The levels of potassium fed during the feeding trial were 0.25, 0.47, 0.60 and 0.79% of the air-dry ration. During the depletion period (22 days) of the appetite trial 0.25% potassium ration was fed in conjunction with intraruminal injections of either deionized water (100 ml) or potassium solution (50 g/100 ml deionized water). The level of potassium in the ration was changed to 0.60% during the repletion period (20 days). The daily potassium intakes during the metabolism trial were 1086.75 (high), 439.41 (medium) and 156.59 mEq (low).

Data collected indicate the following:

(1) The potassium requirement for rapidly growing yearling steers is greater than 0.47% but equal to or slightly less than 0.60% of the air-dry ration.

(2) A ration level of 0.25% (169.26 mEq/day) potassium is below the potassium maintenance requirement and steers receiving that level show typical symptoms of potassium deficiency. However a daily potassium intake of 439.41 mEq (1.83 mEq/kg body weight) is adequate for maintenance of yearling heifers averaging 239.9 kg in body weight.

(3) Ration potassium greatly influences growth. Rations containing 0.47, 0.60 and 0.79% potassium supported significantly greater body weight gains than the ration containing 0.25%.

(4) Potassium has a marked effect on appetite. Daily feed consumption of steers decreased as level of potassium in the ration was decreased below 0.60%. The effect of potassium on appetite does not appear to be mediated solely via the oral cavity. Steers receiving an inadequate potassium ration in conjunction with intraruminal injections of potassium (unlike the control steers) maintained normal serum potassium levels, displayed superior appetite and gained significantly more in body weight.

(5) Potassium intake has an effect on serum electrolytes. Serum potassium levels of the steers were significantly decreased while serum magnesium and chloride levels were significantly increased when dietary potassium was low. Serum calcium, phosphorus and sodium levels were not significantly influenced by treatment. There was a tendency, however, for serum calcium and phosphorus levels to increase as ration potassium decreased. Serum electrolyte values of the heifers were similarly affected by potasium intake except that levels of serum phosphorus increased significantly, and serum magnesium non-significantly as potassium intake decreased. Serum levels of chloride were not affected by treatment.

(6) The number of days for wound healing (gluteal incision) is significantly increased when ration potassium is decreased from 0.47 to 0.25%.

(7) The pH and *in vitro* microbial activity of rumen ingesta increased non-significantly as ration potassium increased.

(8) Potassium and sodium levels of rumen fluid were not significantly affected by ration potassium.

(9) Apparent potassium balance is markedly influenced by ration potassium. The high potassium heifers retained an average of 147.67 mEq of potassium daily which, is significantly greater than a retention of 15.47 and a loss of 25.13 mEq/day by the medium and low treatment heifers, respectively.

(10) Fecal and urinary potassium excretion increases significantly as ration potassium increases. However, the fecal potassium values during the experiment closely approximated the pre-experimental values. The apparent absorption of potassium increases as ration potassium increases.

(11) The positivity of apparent sodium balance is not significantly affected by ration potassium. Similarly ration potassium does not significantly influence the urinary and fecal excretions of sodium.

(12) Heifers receiving the low potassium ration lost an average of 1.8 kg body weight compared to increases of 3.9 and 4.7 kg by the medium and high potassium heifers over a 40-day period. However, the positivity of apparent nitrogen balance was not affected by potassium treatment. The excretions of urine ammonia increase significantly and urea non-significantly as ration potassium decreases.

(13) Potassium has no significant effect on apparent water balance. However, heifers receiving the high potassium ration consumed significantly more water than the medium and low potassium heifers. Similarly, the volume of urine excreted by the high potassium heifers was significantly greater than that of medium and low potassium heifers.

(14) Levels of potassium in the ration have no significant effect on apparent digestibility of ration components.

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APPENDIX

Experiment 1

TABLE 9. MEAN SQUARES FOR WEIGHT GAINS, SERUM ELECTROLYTES, RUMEN FLUID pH, MICROBIAL ACTIVITY AND SODIUM AND POTASSIUM CONCENTRATION AND WOUND HEALING

ITEM		
Source of variation	Treatment	Error
Degrees of freedom	3	20
	(Mean squares)	
Weight gains	23295.21**	906.49
Potassium	0.45**	0.03
Sodium	10.93	11.31
Calcium	0.59	0.27
Magnesium	0.51**	0.04
Phosphorus	2.43	1.36
Chloride	74.58**	8.36
Microbial activity	1400.52	400.66b
Rumen fluid pH	0.11	0.11b
Rumen fluid sodium	0.16	0.13c
Rumen fluid potassium	0.02	0.02c
Wound healing	96.00*a	8.50d

* $(p < 0.05)$
** $(p < 0.01)$
a Treatment degrees of freedom 1
b Error degrees of freedom 11
c Error degrees of freedom 12
d Error degrees of freedom 4

Experiment 11

TABLE 10. MEAN SQUARES FOR THE VARIOUS MEASUREMENTS MADE IN EXPERIMENT 11.

ITEM		
Source of variation	Treatment	Error
Degrees of freedom	3	8
Part 1		
Serum potassium	0.79**	0.06
Serum sodium	20.56	12.42
Rumen fluid potassium	0.34	0.16
Rumen fluid sodium	1.12	0.55
Part 11		
Serum potassium	0.08	0.08
Serum sodium	98.31	36.25
Rumen fluid potassium	0.49	0.39
Rumen fluid sodium	0.28	0.86
Change in weight within subgroups.		
Part 1		
Group 1	4188.30*a	268.96b
Group 11	288.29a	608.74b
Part 11		
Group 1	60.61a	430.35b
Group 11	1650.21**a	66.24b
Change in weight within same subgroups.		
Parts 1 and 11		
Group 1a	3947.54a	526.65b
Group 1b	44.55a	198.41b
Group 2a	4174.90*a	439.63b
Group 2b	74.39a	143.65c

* $(p < 0.05)$
** $(p < 0.01)$
a Treatment degree of freedom 1
b Error degree of freedom 4
c Error degree of freedom 2

Experiment 111

TABLE 11. MEAN SQUARES FOR THE VARIOUS MEASUREMENTS MADE IN EXPERIMENT 111.

ITEM			
Source of variation Degrees of freedom	Treatments 2	Periods 5	Error 10
Potassium balance	48987.48**	1348.38	2599.92
Sodium balance	1433.52	1697.07	1363.96
Nitrogen balance	674.09**	2392.71**	21.87
Water balance	0.02	1.58	0.54
Urinary potassium	867912.22	1578.09	3051.75
Urinary sodium	1032.91	1221.78	1179.72
Urinary nitrogen	15.18	3.31	19.62
Urinary water	45.45**	1.76*	0.51
Fecal potassium	4191.14**	142.86	74.14
Fecal sodium	66.69	264.58*	69.67
Fecal nitrogen	0.38	1.09	1.34
Fecal water	0.62**	0.01	0.03
Water consumption	50.32	3.09	1.10
Urinary urea	765.52	-	537.47
Urinary nitrogen	23.90	-	0.35
Energy digestibility	9.84	-	4.56
Dry matter digestibility	0.31	-	7.77
Nitrogen digestibility	0.16	-	5.08
Crude fibre digestibility	0.25	-	71.22
Ether Extract digestibility	0.22	-	1.26
Av. weight change	29.46	-	14.32
Serum potassium	0.12	-	0.02
Serum sodium	21.67	-	18.13

Serum chloride	3.75	-	2.26
Serum magnesium	0.19	-	0.07
Serum calcium	0.07	-	0.26
Serum phosphorus	3.36*	-	0.30

* $(p<0.05)$
** $(p<0.01)$

Experiment 1

TABLE 12. MICROBIAL ACTIVITY AND pH OF RUMEN CONTENTS AND POTASSIUM AND SODIUM CONCENTRATION OF RUMEN FLUID.

Group	Steer No.	Treat-ment	Microbial Activity**	pH	Potassium*	Sodium*
1	5W	.25%	19.23	7.28	20.0	110
	18W	"	63.89	7.08	24.5	95
	22W	"	17.13	6.50	35.8	123
	24W	"	21.99	6.53	16.3	103
11	6W	.47%	46.15	6.61	28.8	98
	7W	"	68.91	7.16	18.8	118
	8W	"	18.96	7.30	30.8	140
	12R	"	66.91	6.87	25.0	118
111	1W	.60%	68.72	6.75	33.3	123
	11W	"	80.56	6.86	28.3	115
	20W	"	50.27	7.00	25.8	120
	16W	"	-	-	20.0	120
1V	6R	.79%	96.77	6.68	32.0	120
	10R	"	59.00	7.46	25.0	128
	16R	"	64.93	7.39	22.0	113
	21R	"	72.22	7.35	24.5	128

* Expressed as mEq/1 rumen fluid.

** Expressed as mm Hg/g dry matter/90 minutes.

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TABLE 13. DAILY PERIODIC FEED CONSUMPTION, POTASSIUM INTAKE, FECAL POTASSIUM, URINARY POTASSIUM, SODIUM INTAKE, FECAL SODIUM, URINARY SODIUM, NITROGEN INTAKE, FECAL NITROGEN, URINARY NITROGEN, WATER INTAKE, FECAL WATER AND URINARY WATER OF HEIFERS ON THE LOW POTASSIUM RATION

Heifer No.	c	Period 1⁰	2⁰	3⁰	4⁰	5⁰	6⁰
		Feed Consumption (kg)					
1R	2.54	2.54	2.54	2.54	2.54	2.54	2.54
13R	2.54	2.54	2.54	2.54	2.54	2.54	2.54
11R	2.54	2.54	2.54				
		** Potassium intake (mEq)					
1R	439.48	105.20	104.54	104.69	105.19	105.01	105.27
13R	439.33	104.59	104.50	104.47	104.69	104.59	104.60
11R	439.34	103.93	103.91				
		Fecal Potassium (mEq)					
1R	57.94	42.34	58.83	50.81	53.65	56.83	59.88
13R	113.89	145.78	86.16	98.27	107.78	92.26	92.97
11R	31.34	80.39	42.24				
		Urinary Potassium (mEq)					
1R	520.13	196.50	105.72	94.65	124.70	102.34	137.10
13R	345.26	137.85	69.85	67.44	87.29	92.37	76.72
11R	276.81	135.04	77.58				
		** Sodium intake (mEq)					
1R	256.10	256.53	256.02	256.14	256.52	256.38	256.58
13R	255.98	256.06	255.99	255.97	256.18	256.06	256.07
11R	255.99	255.55	255.54				
		Fecal Sodium (mEq)					
1R	36.64	66.96	46.91	43.57	45.12	57.17	44.18
13R	124.07	56.28	157.58	118.74	46.95	77.15	99.65
11R	31.34	80.39	42.24				

TABLE 13 (continued)

Urinary Sodium (mEq)							
1R	112.03	354.96	99.68	156.17	146.97	155.73	195.86
13R	296.26	133.26	99.78	115.99	109.11	125.96	137.00
11R	161.25	188.74	89.90				
Nitrogen intake (g)							
1R	96.16	96.16	96.16	96.16	96.16	96.16	96.16
13R	96.16	96.16	96.16	96.16	96.16	96.16	96.16
11R	96.16	96.16	96.16				
Fecal Nitrogen (g)							
1R	16.14	14.03	17.15	16.51	17.81	18.20	18.08
13R	23.08	19.65	16.40	19.88	21.50	21.28	17.74
11R	17.68	18.55	14.80				
Urinary Nitrogen (g)							
1R	58.41	67.10	57.39	66.26	57.90	58.73	64.63
13R	73.06	65.71	59.37	62.58	68.59	73.33	64.12
11R	63.96	52.17	48.03				
Water intake (l)							
1R	10.39	14.72	9.60	10.80	14.60	13.20	15.20
13R	9.20	10.00	9.28	9.10	11.20	10.00	10.10
11R	9.33	4.93	4.79				
Fecal Water (l)							
1R	2.25	2.53	2.31	2.10	2.12	2.07	2.22
13R	2.07	2.33	2.11	1.86	1.94	1.83	1.85
11R	1.68	2.05	1.33				
Urinary Water (l)							
1R	8.00	9.73	6.04	9.47	8.91	8.90	9.79
13R	4.46	4.60	4.99	5.40	6.24	5.60	5.48
11R	5.38	3.07	2.46				

c 5-day pre-experimental period
0 5-day experimental period
* Expressed as kg air-dry ration
** Amount of the element fed and inherent in the drinking water.

Experiment 111

TABLE 14. DAILY PERIODIC FEED CONSUMPTION, POTASSIUM INTAKE, FECAL POTASSIUM, URINARY POTASSIUM, SODIUM INTAKE, FECAL SODIUM, URINARY SODIUM, NITROGEN INTAKE, FECAL NITROGEN, URINARY NITROGEN, WATER INTAKE, FECAL WATER AND URINARY WATER OF HEIFERS ON THE MEDIUM POTASSIUM RATION

Heifer No.	Period c	1°	2°	3°	4°	5°	6°
			* Feed Consumption (kg)				
4W	2.54	2.54	2.54	2.54	2.54	2.54	2.54
12B	2.54	2.54	2.54	2.54	2.54	2.54	2.54
22B	2.54	2.54	2.54	2.54	2.54	2.54	2.54
			**Potassium Intake (mEq)				
4W	439.46	439.61	439.33	439.78	439.91	439.79	440.00
12B	439.38	438.99	439.14	439.20	439.35	439.08	439.54
22B	439.30	439.17	439.22		439.08	439.34	439.36
			Fecal Potassium (mEq)				
4W	135.91	144.34	117.81	115.87	137.82	106.07	125.42
12B	153.50	142.91	132.84	170.35	142.08	120.27	121.60
22B	108.34	131.89	139.16		67.56	140.97	125.05
			Urinary Potassium (mEq)				
4W	220.74	262.04	295.46	301.02	258.64	320.29	280.00
12B	289.29	278.19	305.92	283.82	339.47	356.08	335.84
22B	395.44	310.98	298.95		319.63	247.49	239.49
			** Sodium Intake (mEq)				
4W	256.09	256.20	255.98	256.33	256.43	256.34	256.50
12B	256.02	255.72	255.84	255.88	256.00	255.79	256.14
22B	255.96	255.86	255.90		255.79	255.99	256.01
			Fecal Sodium (mEq)				
4W	75.37	91.42	59.90	59.95	75.77	46.96	77.18
12B	103.61	80.17	75.05	94.66	56.97	65.92	51.68
22B	66.98	74.03	74.39		36.43	71.53	59.40

TABLE 14 (continued)

			Urinary Sodium (mEq)				
4W	144.33	141.10	144.37	156.69	144.53	136.66	168.96
12B	203.46	151.74	162.76	155.42	173.93	195.65	223.90
22B	252.08	230.80	185.68		197.30	189.48	120.79
			Nitrogen Intake (g)				
4W	96.16	96.16	96.16	96.16	96.16	96.16	96.16
12B	96.16	96.16	96.16	96.16	96.16	96.16	96.16
22B	96.16	96.16	96.16	96.16	96.16	96.16	96.16
			Fecal Nitrogen (g)				
4W	15.85	17.03	16.69	15.16	17.73	15.71	19.13
12B	20.51	20.95	24.24	25.76	18.46	19.47	16.81
22B	20.35	18.85	16.23		14.84	19.75	18.39
			Urinary Nitrogen (g)				
4W	62.49	62.49	69.16	65.15	54.77	64.06	57.93
12B	72.16	59.68	71.73	64.33	67.06	72.00	71.65
22B	68.18	72.89	70.94		54.45	51.82	58.73
			Water Intake (l)				
4W	10.27	11.42	9.20	12.70	13.70	12.80	14.40
12B	9.60	6.60	7.78	8.20	9.36	7.30	10.84
22B	9.02	8.00	8.40		7.30	9.32	9.48
			Fecal Water (l)				
4W	1.89	2.24	2.04	2.13	1.99	1.94	2.26
12B	2.33	2.08	2.63	2.81	2.62	2.76	2.70
22B	2.34	1.94	1.81		1.72	2.30	2.17
			Urinary Water (l)				
4W	3.40	6.72	6.72	8.25	7.61	8.54	9.66
12B	3.18	2.53	3.01	2.70	4.19	3.91	6.40
22B	5.73	4.86	4.76		3.95	3.87	4.17

c 5-day pre-experimental period
o 5-day experimental period
* Expressed as kg air-dry ration
** Amount of the element fed and inherent in the drinking water.

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TABLE 15. DAILY PERIODIC FEED CONSUMPTION, POTASSIUM INTAKE, FECAL POTASSIUM, URINARY POTASSIUM, SODIUM INTAKE, FECAL SODIUM, URINARY SODIUM, NITROGEN INTAKE, FECAL NITROGEN, URINARY NITROGEN, WATER INTAKE, FECAL WATER AND URINARY WATER OF HEIFERS ON THE HIGH POTASSIUM RATION

Heifer No. c	Period 1°	2°	3°	4°	5°	6°
			* Feed Consumption (kg)			
3R	2.54	2.54	2.54	2.54	2.54	2.54
2W	2.54	2.54	2.54	2.54	2.54	2.54
1W	2.54	2.54	2.54	2.54	2.54	2.54
			** Potassium Intake (mEq)			
3R	439.80	1035.05	1034.90	1034.74	1035.21	1035.05	1035.21
2W	439.74	1034.85	1034.90	1034.85	1034.43	1034.69	1034.79
1W	440.09	1034.74	1034.81		1034.90	1034.59	1034.90
			Fecal Potassium (mEq)			
3R	84.81	79.97	59.98	92.02	121.36	69.76	80.16
2W	117.63	119.73	98.02	111.92	122.44	129.65	96.34
1W	127.97	128.74	115.86		98.45	104.29	87.48
			Urinary Potassium (mEq)			
3R	102.60	873.91	927.71	912.95	691.54	829.19	953.19
2W	281.31	746.81	718.34	842.42	504.54	885.63	898.13
1W	324.75	737.03	955.96		914.76	844.73	970.67
			** Sodium Intake (mEq)			
3R	256.35	256.74	256.62	256.50	256.86	256.74	256.86
2W	256.30	256.58	256.62	256.58	256.26	256.46	256.54
1W	256.57	256.50	256.55		256.62	256.38	256.62
			Fecal Sodium (mEq)			
3R	102.43	69.47	85.11	76.98	63.96	112.62	128.32
2W	101.41	53.04	98.98	67.10	27.19	33.23	25.61
1W	45.66	23.16	29.97		52.82	59.48	57.44

TABLE 15 (continued)

Urinary Sodium (mEq)							
3R	82.08	178.02	123.34	112.71	92.21	132.67	148.13
2W	245.24	116.06	202.20	113.81	75.87	255.94	233.62
1W	187.34	54.64	147.07		127.51	159.01	202.74
Nitrogen Intake (g)							
3R	96.16	96.16	96.16	96.16	96.16	96.16	96.16
2W	96.16	96.16	96.16	96.16	96.16	96.16	96.16
1W	96.16	96.16	96.16	96.16	96.16	96.16	96.16
Fecal Nitrogen (g)							
3R	15.94	16.16	13.87	17.43	22.48	16.64	22.09
2W	20.15	22.38	18.99	19.12	17.30	22.14	16.25
1W	18.74	16.78	17.60		17.06	17.14	16.85
Urinary Nitrogen (g)							
3R	49.25	59.34	72.93	59.74	69.67	49.09	61.83
2W	61.31	53.49	55.34	59.20	57.66	56.88	59.18
1W	71.42	57.98	60.93		62.09	66.58	74.95
Water Intake (l)							
3R	12.86	16.80	15.60	14.40	18.00	16.80	18.00
2W	12.40	15.20	15.60	15.20	11.97	14.00	14.80
1W	15.11	14.40	14.90		15.60	13.20	15.60
Fecal Water (l)							
3R	2.64	2.38	2.91	2.94	3.04	2.60	2.76
2W	2.12	2.38	2.57	2.57	2.52	2.65	2.79
1W	2.24	2.36	3.11		2.37	2.73	2.72
Urinary Water (l)							
3R	8.21	10.79	10.73	11.27	10.25	13.27	12.88
2W	7.21	10.09	10.64	11.38	7.59	8.13	10.38
1W	11.71	9.83	10.51		11.09	9.94	12.29

c 5-day pre-experimental period
o 5-day experimental period
* Expressed as kg air-dry ration
** Amount of the element fed and inherent in the drinking water.