THE EXCHANGEABLE POTASSIUM CONTENT IN DISEASE STATES [1, 2]

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Although the importance of potassium in physiologic processes has long been recognized, there is a paucity of information concerning its metabolism, particularly in clinical disease states. This scarcity of knowledge has been due primarily to the technical difficulties involved in measurement of soluble cations. The introduction of flame spectrophotometry and the availability of synthetic radioactive isotopes have partially overcome these technical limitations.

Although information is available concerning the external balance of potassium and alterations in the extracellular potassium concentration in various disease states, such data give no direct information concerning the body store of this cation. The injection of radioactive potassium (K^{42}) affords the only direct procedure available for estimating the total potassium content of the body in the intact animal. The specific activity of the urine, after partial equilibration of radioactive potassium with the native atoms, can be used as a measure of the "total exchangeable potassium content" (Ke). Values for normal young males and females have been reported (1, 2). No such studies in individuals with various disease states are yet available.

The purpose of the present study was to determine the extent to which pathologic conditions alter the exchangeable potassium content of hospitalized subjects, and to attempt to correlate alterations in this measurement with the associated metabolic disturbances, and to determine if the administration of "loading" doses of potassium could correct deficiencies detected.

MATERIAL AND METHODS

Subjects

A total of 69 hospitalized subjects—39 males and 30 females—between the ages of 14 and 78 years were studied. The various pathologic states observed are listed in Tables I–IV. All had illnesses of some weeks' duration which justified hospitalization for study and treatment. In addition, six normal individuals, three males and three females, were studied in order to determine the reproducibility of the measurement and the effect of the administration of an oral supplement of potassium.

The subjects were divided into four groups. Group 1 consisted of the normal subjects and the four patients in steady states. Groups 2 and 3, consisting, respectively, of 28 males and 22 females, were used for the original survey study. The individuals in these two groups were selected at random from patients admitted to the medical wards. Studies were performed on any such patient from whom reliable urine collections could be made. Because facilities were limited, it was not possible to include all likely candidates in the study.

Group 4, consisting of nine males and six females, was studied a few months after the study of groups 2 and 3 was completed. In this group serial determinations of Ke were performed in order to study the effect of supplementary potassium chloride. At least two determinations of Ke were performed on each patient in this group.

Plan of the experiment

In the normal subjects in group 1, two determinations of Ke were performed one week apart; the subjects were then given KCl, 3 gm. daily for six days, and the third Ke was measured. The manner in which the other four were studied is described under Comments in Table I.

Single determinations of Ke were done in groups 2 and 3. In group 4, the initial determination of Ke was performed as soon as possible after the patient's admission to the hospital. Upon completion of the initial study, 13 subjects were given oral supplements of potassium chloride, 3 gm. (40 meq.) daily, for a week. Then a second Ke determination was done after which potassium chloride was discontinued. Whenever possible a third determination was performed a week later. Two of the individuals in group 4 were not given supplementary potassium chlo-

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ride; in these cases, the effect of the disease process was observed by serial determinations of Ke.

Experimental procedure

After breakfast, between 8:30 and 9 a.m., each subject was given an intravenous injection of radioactive potassium by calibrated syringe. All urine specimens until 6 a.m. the next morning were collected and pooled, and this collection of urine was measured for the excretion of K^{42}. The specific activity of the urine was determined on two spot samples collected at 7 and 9 a.m. on the day after injection. The subject was asked to empty his bladder completely by voiding. In some instances, a specimen could not be obtained at the exact time, but was obtained as soon thereafter as possible.

No restrictions were placed on activity or on the intake of food, water, or salt. The usual lunch and supper were given on the day of injection. Breakfast the next morning was delayed until after the second spot urine sample was obtained.

Preparation and administration of radioactive potassium

All shipments of K^{42} were treated in the manner described by Corsa and his co-workers (1). Usable shipments were usually received early Tuesday morning, and most of the injections were made later that morning. Approximately 1.5 meq. of potassium chloride solution, containing 100 microcuries of K^{42}, were injected intravenously. A few injections were given on Wednesday, and these patients received 3 meq. of potassium chloride solution containing 50 microcuries of K^{42}.

Measurement of radioactivity

The activity of the urine specimens was determined with a dipping tube and a scaling circuit. Counts were made to 1% accuracy. All counting rates were at least ten times background, and were usually in the range of 500 to 3,000 per minute. At this range of counting rate, no dead time correction for the Geiger tube was necessary. All determinations were corrected for decay.

Preliminary studies confirmed the observations of Corsa and his associates (1) that the specific activity of potassium in the urine reached an equilibrium by 24 hours. The mean difference in specific activity between the two spot specimens, when expressed as per cent of the mean Ke, was $6.52 \pm 6.65\%$.

TABLE I

Reproducibility of exchangeable potassium measurements and the effect of oral supplements of potassium in normal subjects and in patients in steady states

Subjects	Ke_1		Ke_2		Ke_3	
	(meq.)	(meq./Kg.)	(meq.)	(meq./Kg.)	(meq.)	(meq./Kg.)
Normal Subjects						
Women						
1	2,500	45.9	2,219	40.7	2,280	38.2*
2	1,947	39.3	1,682	34.0	1,679	33.9*
3	2,160	38.9	2,155	38.8	2,358	42.5*
Men						
1	3,641	43.3	3,447	41.0	4,145	49.3*
2	3,537	48.1	3,261	44.3	3,753	51.0*
3	4,064	38.1	4,007	37.5	4,136	38.7*

Patients in Steady States

Patient	Age, sex	Diagnosis	Ke_1		Ke_2		Ke_3		Comments
			(meq.)	(meq./Kg.)	(meq.)	(meq./Kg.)	(meq.)	(meq./Kg.)	
Ba	M 71	Diabetes with peripheral vascular disease	1,544	25.3	1,524	24.9	1,252	20.6*	Diabetes well regulated. Supracondylar amputation between Ke_2 and Ke_3
Ho	M 72	Diabetes with peripheral vascular disease	1,848	34.7	1,750	32.9*	1,894	36.6	Diabetes well regulated
Bo	F 59	Myxedema and diabetes	1,553	21.4	1,576	21.7			Myxedema for several years. Diabetes well regulated. No potassium given
Th	F 38	Ulcerative colitis	1,641	34.7	1,638	34.6	1,811	37.6	Duration 8 years. In relapse during study. Interval of 2 wks. between Ke_2 and Ke_3

All Ke determinations performed at weekly intervals unless otherwise noted.
*Ke determination after administration of oral supplement of KCl, 3 gm. daily, for six days.

TABLE II

Exchangeable potassium content in males with disease states (group 2)

Patient	Age (*yrs.*)	Diagnosis	Ke (*meq.*)	Body weight (*Kg.*)	Ke/Wt. (*meq./Kg.*)	Remarks
1	32	Hypertensive cardiovascular disease	3,797	64.5	58.9	Duration 11 yrs. Symptomatic for 6 mos. Clinically well nourished.
2	51	Neurosyphilis	4,016	68.3	58.8	Spinal fluid protein = 150 mg. %. Clinically well nourished.
3	48	Polycystic kidneys with azotemia	3,289	60.8	54.1	Hypertension for 3 yrs. NPN = 166 mg. %. No generalized edema.
4	42	Ulcerative colitis	2,767	53.6	51.6	Duration 6 mos. 34 lbs. weight loss.
5	66	Pneumoconiosis and pulmonary emphysema	2,772	54.1	51.2	Arteriosclerotic heart disease. Slender.
6	37	Pyelonephritis	3,097	61.4	50.4	Minimal symptoms for 6 mos. No recent weight loss or generalized edema. NPN = 65 mg. %.
7	56	Iron-deficiency anemia	2,942	60.0	49.0	Duration 6 mos. Weight loss of 15 lbs.
8	49	Duodenal ulcer, adenocarcinoma of cecum	2,608	54.5	47.9	Clinically emaciated. Weight loss of 20 lbs. in 5 mos. No anemia or hypoproteinemia.
9	25	Convulsive disorder, pyrexia of undetermined origin	2,679	57.5	46.6	Symptomatic 2 wks. Afebrile at time of determination.
10	71	Generalized arteriosclerosis	2,629	56.6	46.4	Chronic blood loss from internal hemorrhoids. Hemoglobin = 10.5 gm.
11	58	Diabetes mellitus	2,088	45.8	45.6	Duration 3 yrs. Poorly controlled 8 mos., with 20 lbs. weight loss.
12	19	Peptic ulcer	2,912	65.1	44.7	Epigastric symptoms for 18 mos. Hematemesis 8 days prior to admission.
13	14	Congenital heart disease	1,358	30.5	44.5	Interventricular septal defect. Cryptorchidism.
14	16	Trichinosis, peptic ulcer	2,478	55.9	44.3	Duration 2.5 wks. Eosinophilia (25%). Positive trichinella skin test.
15	40	Cerebral aneurysm	3,282	79.1	41.5	Symptoms limited to complaint of headaches. Normal otherwise.
16	56	Neurosyphilis	2,370	57.7	41.1	Spinal fluid protein = 102 mg. %.
17	49	Diabetes mellitus	3,383	86.8	39.0	Well regulated. Admitted for therapy of acute upper respiratory infection.
18	58	Chronic cholecystitis with cholelithiasis	2,801	73.0	38.4	Faint icterus. Clinically well nourished.
19	56	Rheumatic and arteriosclerotic heart disease	2,535	69.3	36.6	Digitalized; no edema or weight loss. Auricular fibrillation.
20	41	Progressive peroneal atrophy	1,562	43.4	36.0	Duration 30 yrs.
21	57	Portal cirrhosis	2,053	61.3	33.5	Neurodermatitis disseminata for 8 mos. 28 lbs. weight loss in 2 mos. 45% BSP retention.
22	76	Arteriosclerotic heart disease	2,500	75.2	33.3	Progressive congestive heart failure 14 mos. 20 lbs. weight loss in 1 yr. Pitting edema.
23	78	Adenocarcinoma of head of pancreas	1,329	42.3	31.4	30 lbs. weight loss in 1 mo. prior to admission. Known weight loss for 1 yr. Anorexic.
24	69	Thyrotoxicosis, arteriosclerotic heart disease	1,413	45.5	31.1	Parkinsonism 35 yrs. Duration of thyrotoxicosis, 8 mos. In relapse.
25	56	Arteriosclerotic heart disease, pulmonary embolism	1,437	48.0	29.9	Duration heart disease 15 mos. with 40 lbs. weight loss. Loss of 20 lbs. in 10 days on mercurials and digitalis.
26	42	Metastatic carcinoma	2,350	82.7	28.4	Weight loss 20 lbs. in preceding 4 mos. Persistent fever. Primary site in colon.
27	55	Malnutrition, empyema, pneumothorax	864	32.5	26.6	Hypertensive cardiovascular disease. 75 lbs. weight loss. Expired 10 days after determination.
28	16	Inactive rheumatic heart disease	1,667	67.7	24.6	Acute rheumatic fever 3 yrs. previously. Normal sedimentation rate at time of determination.

Chemical determinations

Total *potassium concentration* in the urine was determined by flame spectrophotometry, using the method of Mosher and his associates (3).

Calculation of exchangeable potassium

The following formula was used to calculate the value for the exchangeable potassium content of the body:

$$Ke = \frac{K_I^{42} - K_o^{42}}{\frac{Ku^{42}}{Ku^{39}}}.$$

Ke = quantity of exchangeable potassium in milliequivalents.

K_I^{42} = quantity of radiopotassium administered (arbitrary units).

K_o^{42} = quantity of radiopotassium excreted in the urine within 21 to 21.5 hours after its intravenous injection.

Ku^{42} = concentration of radiopotassium in the urine samples.

Ku^{42}/Ku^{39} = mean specific activity of two spot specimens.

RESULTS

Group 1 (Table I)

Better reproducibility of Ke was obtained in patients in steady states than in normal young subjects. However, the activity and the diet of the individuals during the study was better controlled in the patients. The mean difference between the two Ke determinations made a week apart without oral supplementation was 125 meq. or 2.0 meq./Kg. Following the administration of an oral supplement, the second Ke was higher by a mean of 212 meq. or 2.2 meq./Kg. In the interpretation of the results in the other groups, an increase of greater than 250 meq. or 5 meq./Kg. was considered of significance, although it is rec-

TABLE III

Exchangeable potassium content in females with disease states (group 3)

Patient	Age (*yrs.*)	Diagnosis	Ke (*meq.*)	Body weight (*Kg.*)	Ke/Wt. (*meq./Kg.*)	Remarks
1	25	Diabetes mellitus	2,201	43.6	50.5	Duration 9 yrs. Poor control. Urinary sugar 3 + on day of determination.
2	55	Diabetes mellitus	2,540	55.9	45.5	Clinically well nourished. Duration 4 yrs. with poor control. Neurogenic bladder.
3	34	Chronic anxiety state	1,811	45.2	40.1	Duration 2 yrs. Weight loss 20 lbs.
4	54	Vascular lesion, basal ganglion	1,805	46.4	38.9	Tremor of right arm, progressive for 3 yrs. Recent involvement of right leg.
5	67	Gastric ulcer	1,368	36.0	38.0	Very slender. Quiescent bronchial asthma.
6	42	Hypertensive cardiovascular disease	1,789	47.5	37.7	No history of weight loss.
7	59	Hypertensive and arteriosclerotic heart disease	2,230	60.9	36.6	Pulmonary fibrosis. Digitoxin, 0.8 mg., given during determination.
8	52	Psychoneurosis	1,498	43.2	34.7	Weight loss and diarrhea for several months. Narcotic addiction.
9	21	Ulcerative colitis, emetine intoxication	1,551	44.8	34.6	Duration colitis, 7 yrs. Emetine therapy 1 mo. previously; subsequently developed generalized weakness and tachycardia.
10	43	Duodenitis, anemia	1,666	51.8	32.2	Iron-deficiency anemia. Asymptomatic intestinal amebiasis.
11	41	Rheumatic heart disease	1,726	54.1	31.9	Mitral stenosis and insufficiency. On digitalis and quinidine.
12	54	Rheumatoid arthritis	1,609	55.9	28.8	Progressive disease for 2 yrs. Bronchial asthma for 30 yrs.
13	30	Exogenous obesity	2,700	95.2	28.4	Typical fat distribution.
14	55	Osteoarthritis	2,031	71.6	28.4	Slightly obese.
15	21	Chronic anxiety state	1,439	50.9	28.3	Emotional immaturity. Clinically simulated thyrotoxicosis. Weight loss of 7 lbs. in 3 wks. before admission.
16	24	Multiple sclerosis	1,380	49.5	27.9	In relapse. Duration of illness, 1 yr. No recent weight loss.
17	65	Generalized urticaria	1,435	53.2	27.0	Urticaria for 2.5 mos., etiology unknown. Postmenopausal osteoporosis.
18	73	Rheumatoid arthritis and cerebrovascular accident	1,221	47.3	25.8	Chronic disease state. Arthritis for several years. Generalized arteriosclerosis.
19	44	Obesity, anemia	1,675	67.5	24.8	Iron-deficiency type. Anticoagulant therapy for retinal vein thrombosis.
20	19	Diabetes mellitus	1,673	72.0	23.2	Duration 2 yrs. Urinary sugar 4+.
21	44	Psychoneurosis	1,307	64.5	20.3	Tension headaches, psychic anorexia, nausea and vomiting. Weight loss of 30 lbs.
	45	Exogenous obesity, hypertensive cardiovascular disease	1,281	68.2	18.8	Mild cerebrovascular accident.

ognized that the retention of administered potassium over a short period may not be definite evidence for a prior depletion.

Group 2 (Table II)

When the values for Ke/Wt. from this group of male subjects with various disease states were compared with those found in 30 normal young males (in whom the range was 35.6 — 53.6 meq./Kg.), three subjects (Cases 1, 2, and 3) had values higher than those found in the normal males. Eight of the 28 subjects had values lower than 35.6 meq./Kg. The disease states in which these low values were found included inactive rheumatic

TABLE IV

Exchangeable potassium content in subjects studied serially (group 4)

Patient, sex	Age (*yrs.*)	Diagnosis	Ke (*meq.*)	Body weight (*Kg.*)	Ke/Wt. (*meq./Kg.*)	Remarks
Group 4A						
1 M	39	Carcinoma metastatic to the liver	1,566*	55.0	28.5	Onset with weakness and weight loss of 20 lbs. in 5 mos. Confirmed by liver biopsy.
			1,600†	51.0	31.4	
2 F	65	Myxedema	1,586*	75.0	21.1	Duration 8 yrs. Refractory anemia. Inadequate protein intake. Thyroid therapy started 3 days after Ke.*
			1,366†	72.7	18.8	
			1,638‡	72.8	22.5	
3 F	65	Myxedema	1,679§	62.4	27.9	Classic symptoms and signs. Anemia for 4 yrs. Hepatomegaly and ascites. Liver biopsy showed fatty metamorphosis. No supplementary K given.
			1,497	60.9	24.6	
4 M		Fever of unknown origin	1,814§	47.7	38.0	Duration of 30 days. No known weight loss. Febrile during studies.
			1,643	47.7	34.4	
5 F	46	Partial intestinal obstruction	1,235*	45.5	27.1	Due to postoperative adhesions. Symptoms suggestive of peptic ulcer for 8 mos. 10 lbs. loss in 4 mos. Diagnosis confirmed at surgery.
			1,344†	45.5	29.5	
6 F	18	Rheumatic heart disease, active	1,014*	45.9	22.1	Recurrent rheumatic activity for 7 yrs. with valvular heart disease, low grade fever and elevated sedimentation rate.
			941†	43.6	21.6	
Group 4B						
1 M	55	Cirrhosis	2,084*	75.5	27.7	History of alcoholism for 30 yrs. with inadequate diet. Ankle edema, ascites for 9 wks. before Ke.*
			2,550†	68.6	37.2	
			2,423‡	68.2	35.5	
2 M	39	Rheumatic heart disease	1,980*	62.8	31.5	Mitral stenosis and insufficiency, aortic insufficiency. Duration at least 10 yrs. On digitalis.
			2,063†	58.9	35.0	
			2,315‡	59.4	39.0	
3 F	24	Multiple sclerosis	1,380*	49.5	27.9	Duration 3 yrs. Studied during a relapse.
			1,657†	49.5	33.5	
4 M	41	Psychoneurosis, adenoma of thyroid	2,764*	83.2	33.2	Vague complaints. Weakness prominent.
			3,135†	83.2	37.7	
			2,967‡	83.2	35.7	
5 M		Chronic glomerulonephritis	2,555*	79.5	32.1	Duration symptoms 11 yrs. Hypertension 3 yrs. Worse past 3 mos. PSP = 9% in 1 hr. NPN = 87 mg. % with Ke.* NPN increased to 154 mg. % on day of Ke.‡
			3,263†	77.3	42.2	
			2,505‡	74.1	33.8	
6 M		Diabetes mellitus	1,553*	52.5	29.6	Duration of diabetes 5 yrs. with poor regulation. Peptic ulcer symptoms for 2 mos. Developed pyloric obstruction and was placed on nasogastric suction between Ke† and Ke.‡ Metabolic alkalosis with Ke.‡
			1,873†	48.3	38.8	
			1,463‡	47.5	30.8	
Group 4C						
1 F	36	Chronic pulmonary fibrosis	2,928*	52.6	55.7	Chronic bronchitis progressive for 13 yrs., with fibrosis. Prone to hyperventilate. 36 lbs. weight loss in 2 yrs.
			2,157†	52.6	41.0	
			2,425‡	52.6	46.1	
2 M	52	Pneumoconiosis, gastric ulcer	3,472*	57.7	60.2	Progressive cough and dyspnea for 16 yrs. with roentgenologic changes. Diagnosis of ulcer established while hospitalized.
			2,657†	57.7	46.0	
			1,776‡	57.7	30.1	
3 M	41	Ulcerative colitis	3,113*	68.2	45.6	Duration 1 mo. 14 stools daily. 40 lbs. weight loss in 6 mos. Asymptomatic peptic ulcer. With Ke*, NPN = 76 mg. %, with Ke†, NPN = 43 mg. %.
			2,319†	68.8	33.8	
			1,916‡	66.8	28.7	

Ke determinations were made at weekly intervals unless otherwise indicated.
* Ke before the administration of KCl.
† Ke after one week of KCl, 3 gm. daily.
‡ Ke one week after discontinuation of KCl.
§ Not given supplementary KCl.

heart disease, malnutrition, carcinoma, arteriosclerotic heart disease, thyrotoxicosis, and cirrhosis.

Group 3 (Table III)

The range of Ke/Wt. in 20 normal young adult females was previously found to be 25.1 to 35.9 meq./Kg. Of the 22 individuals in group 2, seven subjects had values higher than those found in the normal group. Four of the 22 had values below 25.1 meq./Kg. The diagnoses in these four subjects were, respectively: diabetes mellitus, psychoneurosis, obesity and anemia, and exogenous obesity with hypertensive cardiovascular disease.

Group 4 (Table IV)

The results of the study in this group have been divided into three categories on the basis of the type of response to supplementary potassium chloride. Group 4A consists of six individuals who did not show what was considered to be a significant change (± 250 meq.) in Ke when placed on supplementary oral potassium chloride. Group 4B consists of six individuals who showed an increase in Ke at the second or third determination, or with both measurements. Group 4C consists of three individuals in whom the Ke value decreased during the administration of potassium supplements.

Group 4A. No change in the Ke occurred following the administration of potassium supplements in subjects with metastatic carcinoma, myxedema, fever of unknown origin, partial intestinal obstruction, and rheumatic heart disease.

Group 4B. The supplementary feeding of potassium chloride produced an increase in Ke of more than 250 meq. in the following disease states: cirrhosis, rheumatic heart disease, psychoneurosis, multiple sclerosis, chronic glomerulonephritis, and poorly regulated diabetes mellitus.

Group 4C. In these three instances, Ke decreased during the administration of supplementary potassium: chronic pulmonary fibrosis, pneumoconiosis and gastric ulcer, and ulcerative colitis.

COMMENT

It has been recognized in the past that in a few disease states certain abnormalities in potassium metabolism, as evidenced by alterations in the external balance or concentration in the serum, are associated with clinical symptoms and signs. A body deficiency of potassium is known to exist in diabetic acidosis and coma (4), in certain gastrointestinal disorders (5), and in certain postoperative conditions (6). With severe renal damage, retention of potassium may occur. The external balance method, however, gives no direct evidence of any imbalance in the distribution of potassium between the intra- and extracellular fluid compartments, although such an imbalance has been suspected. The difficulty of confirming clinically the suspicion of an intracellular potassium deficiency has been recognized previously. The finding of a low serum potassium concentration is highly suggestive of the presence of body deficiency. However, it is important to realize that a total body deficiency may exist in the presence of a normal serum concentration. All of the serum potassium determinations done on the patients in the present study were within the range of 3.5 to 5.0 meq./Kg.

The availability of radioactive isotopes has provided a method for measuring directly the body content of potassium. Since more than 95% of the body's store of potassium is normally situated within cells, such a measurement of the total body content of potassium should serve as a relatively reliable index to the intracellular store of this cation.

Previous determinations of the exchangeable potassium content in normal male and female subjects have established presumably normal ranges (1, 2). Although it is recognized that such studies to date have been limited to fairly homogeneous groups of young subjects, these values are the only ones available at present for comparison with Ke values obtained in disease states.

The finding, in some diseased males and females, of Ke/Wt. values higher than those obtained in normal subjects suggests that the former group was more muscular than normal subjects. Such a finding suggests that the loss of body fat may elevate the relative fraction of lean tissue. Except in the case of the individual with chronic glomerulonephritis, it is difficult to explain the higher values for Ke/Wt. on the basis of a pathologic state. In severe renal failure, retention of potassium may occur when the intake exceeds the excretory capacity of the kidneys.

The results of the present study seem to indicate that several factors may produce a low Ke/Wt. value:

1. A decrease in body mass secondary to (*a*) a decrease in food intake resulting from anorexia, (*b*) an increase in the catabolic processes, or (*c*) both.

The loss in total tissue mass, in such an event, would parallel the loss of body weight. The concentration of potassium in the remaining tissues, however, would be unchanged, so that no body deficiency of potassium would result. This factor is probably the one principally responsible for the low Ke/Wt. values in the individuals with malnutrition, malignancy, thyrotoxicosis, and chronic infectious diseases or febrile episodes. It has been shown experimentally that during starvation normal rabbits lose body potassium progressively with loss of body weight, but that the concentration of this cation in the remaining tissues is unaltered (7).

2. An increase in the relative fat content of the body, such as occurs with exogenous obesity (Table III, Cases 19 and 22).

3. Expansion of the extracellular fluid compartment as a result of edema or the deposition of myxedema fluid.

Should the low values for Ke/Wt. be due to any or all of the above causes, no decrease in the intracellular concentration of potassium occurs; therefore, no increase in the potassium content of the body would be expected when supplementary potassium is administered. Such was the result obtained in the cases of carcinoma, myxedema, chronic infectious disease, fever of unknown origin, and untreated rheumatic heart disease.

4. Excessive loss of potassium from the body, usually by the gastrointestinal tract, as the result of vomiting, diarrhea, or intestinal suction.

The potassium concentration of the intestinal secretions is several times higher than that of serum. With loss of intestinal contents, a decrease in the intracellular concentration of potassium results. Such a deficit might be corrected simply by supplying potassium orally or parenterally.

5. A cellular metabolic abnormality, which may lead to an intracellular deficiency.

A low value for Ke/Wt. may lead one to suspect such an abnormality. An increase in this value following the administration of supplementary potassium, particularly if such an increase is maintained after the supplement is discontinued, is certainly suggestive of a prior deficiency state. Such an intracellular potassium deficiency appears to be present in poorly regulated diabetes mellitus, and has already been shown to exist in diabetic acidosis and coma. A potassium deficit might be anticipated in cirrhotic patients because of the impairment in carbohydrate metabolism.

In the patients with psychoneurosis and multiple sclerosis, the increase in the Ke values following the administration of potassium supplement suggests the existence of an occult potassium deficiency in these states. Further studies are required to confirm these findings and to explain the mechanism.

SUMMARY

The use of a radioactive isotope of potassium, K^{42}, has made possible the direct measurement of the exchangeable potassium content of the body. Such a determination was performed in 69 hospitalized subjects with various diseases. With few exceptions, the values obtained were either within the range found in normal young subjects, or lower. Although some of these low values could be attributed to the loss of body tissue resulting from chronic illness, the low values in certain disease states suggested the presence of a body deficiency of the potassium ion.

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