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B. Liberman, B.L. Wajchenberg and E.F. Mohrer[1]

SUMMARY

Exchangeable potassium (Ke) was determined in patients with primary and secondary adrenal insufficiency before and after replacement therapy and the induction of a state of hypercortisonism. Ke was also measured in 4 patients with Cushing's syndrome and in 3 of them after therapeutically induced remission. The effect of large doses of prednisone for 7 weeks on Ke in one normal subject was determined. The results suggest that the changes in body potassium content must represent a composite of increases or decreases in body fat and potassium rich lean tissue from non-specific nutritional effects and the specific action of glucocorticoids on the distribution of potassium between intra- and extracellular compartments and or the renal transport of potassium.

There is little information regarding the measurement of exchangeable potassium in states of adrenocortical dysfunction. It was the purpose of this study to investigate in human beings the relationship of adrenal steroids to body potassium composition by the measurement of Ke before and after the administration of prednisone and in conditions of hyper and hypofunction of the adrenal cortex, before and after correction of the abnormal state.

PATIENTS AND METHODS

Eleven healthy normal subjects, 5 males and 6 females, aged 18 to 42 years, were studied to determine the normal values for exchangeable potassium. Eight patients with adrenocortical insufficiency, 5 secondary to pituitary post-partum necrosis (Sheehan's disease) and 3 with Addison's disease were studied. The 5 patients

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with Sheehan's disease were studied after the induction of a state of hypercortisonism with prednisone, 40 mg daily for 3 weeks.

The diagnosis of Sheehan's disease was established from the low urinary gonadotropin excretion, low PBI and ^{131}I uptake and low urinary excretion of hydroxycorticosteroids with a progressive increased excretion of these steroids during intravenous administration of ACTH for two consecutive days. The patients with Addison's disease did not show response to ACTH.

Four patients with Cushing's syndrome, one with bilateral adrenal hyperplasia, of a moderate degree, other with unilateral adenoma, also of a moderate degree and both without clinical detecable muscular wasting or osteoporosis and two patients with adrenal carcinoma, both with a severe form of the disease, were studied. Three of these patients were also studied after treatment of the disease.

One normal subject was also studied after the long-term administration of prednisone, 40 mg/day for 50 days, with 4 studies during steroid treatment.

Exchangeable potassium (Ke) was determined by isotope dilution following the intravenous administration of 0.3 mCi of ^{42}K. The specific activity of potassium was measured in urine after an equilibration time of 24 hours. 4 ml aliquots of two urines samples, obtained after the equilibration time, were used for measurements of radioactivity, each sample of urine was counted in duplicate in a well-type scintillation counter.

Ke was determined dividing the amount of radioactivity remaining in the body after the injection of ^{42}K (i.e., injected radioactivity minus 24-hour excretion of radioactivity in the urine) by the mean of the specific activities of the two urine samples.

$$Ke = \frac{^{42}K \text{ injected} - ^{42}K \text{ excreted (urine)}}{\text{urine } ^{42}K/\text{urine } ^{39}K}$$

^{42}K losses in the stools were not measured as its excretion is negligible (our patients did not have unusual fecal losses).

In many patients each Ke value was based on two isotope dilution studies performed within a 7 to 10 day period, the average value given. The error of a single Ke determination based on 20 paired Ke determinations was ± 5 per cent.

Statistical analysis was made using standard procedures[13], the difference of the means being considered significant at $p < 0.05$ and highly significant at $p > 0.001$.

RESULTS

Table I summarizes the data obtained from the normal subjects. The mean values for total exchangeable potassium were significantly greater in males than in females ($t=4.47$, $p < 0.001$) as well as when expressed in terms of body weight ($t=2.930$, $p<0.05$). These data are in complete agreement with those reviewed by Edelman and Leibman[4] and Moore et al.[10].

The Ke obtained in 5 patients with Sheehan's disease before and after the induction of acute hypercortisonism with prednisone (40 mg daily for 21 days) are given in table II. The patients, before treatment, showed the Ke within the normal female range the results being expressed either as absolute values ($t= 1.521$) or as the Ke/Kg ratio ($t = 1.860$). After steroid treatment, there was a highly significant increase in Ke indicated by its absolute values ($t = 8.745$) but still within the normal range. However, no significant changes were observed in the ratio Ke/Kg

Table I - Exchangeable potassium (Ke) in normal subjects

Males					Females					
	Age (years)	Height (cm)	Body weight (kg)	Ke (mEq)	Ke/kg	Patient	Age (years)	Height (cm)	Body weight (kg)	Ke (mEq)	Ke/kg
	18	179	68.6	3574 3637 3606	52.0 53.0 52.5	M.M.	18	146	44.2	2009 1741	45.4 39.4
	22	163	62.2	2936 2700	47.2 43.4	Y.L.	18	158	50.5	1690 1725	33.4 34.1
	28	168	72.7	3560 3998	49.0 55.0	M.F.	19	152	49.0	1949 1639 1986 1518	39.8 33.4 40.5 30.1
	30	173	69.2	2509 2607	36.3 37.6	M.S.	23	164	56.0	2109 2450 2080	37.7 43.7 37.1
	42	168	70.8	3196 2972	45.0 42.0	A.B.	24	164	65.0	2383 2037	36.6 31.3
						D.O.				1680 1740	37.7 39.1
.....				3118	46.0					1912.5	37.2
.....				537.5	6.4					236.8	3.3
fidence interval for single values				3118 ± 1492	46.0 ± 17.6					1912.5 ± 504.8	37.2 ± 8.6

Table II - Exchangeable potassium (Ke) in patients with Sheehan's disease* before and after prednisone treatment (40 mg/day-21 days)

Patient and age (years)	Height (cm)	Weight (kg)		Exchangeable potassium			
				Before prednisone		After prednisone	
		Before prednisone	After prednisone	mEq	mEq/kg	mEq	mEq/kg
S.E., 30 y	154	57.0	57.0	1628	28.6	2103	33.6
H.K., 35 y	150	46.9	50.0	1700	36.2	1873	37.4
M.R., 37 y	154	52.5	56.7	1496	28.5	2303	40.6
W.A., 40 y	141	61.0	61.5	1700	28.0	1802	29.3
O.B., 41 y	158	52.3	59.3	1650	31.6	2108	35.5
Mean				1635	30.6	2038	35.3
S.D.				83.8	3.4	201.4	4.2

* All patients on tri-iodothyronine therapy (50 or 75 mcg/day).

(t = 1.860) with prednisone treatment. Serum potassium, in the normal range before (from 3.7 to 4.3 mEq/1) did not change after steroid therapy.

The results in Addison's disease, before and after replacement therapy, with cortisone 37.5 mg daily, are shown in table III. Before treatment, two of the patients (M.G. and N. D.) who were in excellent clinical condition and with normal serum potassium, had the Ke/Kg ratios within the 95% confidence limits of the normal values in table I. In patient R. M., studied in acute adrenal crisis (serum sodium: 122 mEq/1 and serum potassium: 6.4 mEq/1), the Ke was below the confidence limits for normal absolute values but not when expressed by the Ke/Kg ratio. After one month of cortisone replacement therapy, both the Ke and Ke/Kg were lower than before treatment, particularly in patient N. D. in which

Table III - Exchangeable potassium (Ke) in patients with Addison's disease and after replacement treatment (cortisone 37.5 mg/day-1 month)

Patient, sex and age (years)	Height (cm)	Weight (kg)		Serum potassium (mEq/l)		Exchangeable potassium			
						Before cortisone		After cortisone	
		Before cortisone	After cortisone	Before cortisone	After cortisone	mEq	mEq/kg	mEq	mEq/kg
M.G., ♀, 31y.	162.5	70.1	71.5	4.0	4.0	2484	35.4	2244	31.4
N.D., ♂, 40y.	159.0	64.6	66.4	4.2	4.3	3076	47.7	2459	37.0
R.M., ♂, 42y.	161.0	58.5	58.0 59.1*	6.4	5.8 4.5*	2257	38.6	1919 1876*	33.1 31.7*

* After 5 weeks on cortisone + DOCA 5 mg I.M. every other day, on the last 5 days.

Ke fell from 3076 to 2459 mEq, without significant changes being noticed in serum potassium, except in patient R.M. In that patient a decrease of 338 mEq in Ke was accompanied by a fall in serum K from 6.4 to 5.8 mEq/l, still in hyperkalemic range. At this time the patient's clinical status was not completely improved as he still was weak, anorectic and had postural hypotension. When DOCA was added, without additional salt intake, there was a dramatic return of a sense of well-being, disappearance of anorexia and a rise in blood pressure within 24 hours. Five days after giving DOCA, the patient hat put on 1.1 kg, his serum potassium was 4.5 mEq/l, with pratically no change in the previous values for exchangeable potassium.

The results in Cushing's syndrome are given in table IV. the two young women with moderately severe disease (M.R.S. and M.J.) had normal serum K levels but Ke and Ke/Kg ratios slightly below the 95% confidence limits for normal single adult female values. Since in females, as well as in males, the total exchangeable potassium, as a function of body weight, decreases with advancing age[10] the low values are even more significant. The

Table IV - Exchangeable potassium (Ke) in patients with Cushing's syndrome before and after treatment

Patient, sex and age (aetiology)	Weight (kg)		Serum potassium (mEq/l)		Exchangeable potassium			
	Before treatment	After treatment	Before treatment	After treatment	Before treatment mEq	Before treatment mEq/kg	After treatment mEq	After treatment mEq/kg
M.R.S., ♀, 15 y (bilateral adrenal hyperplasia)	62.7		4.2		1596	25.4		
		61.6		4.1			1916*	31.1
		55.0		4.1			1824**	33.2
		56.1		4.0			1269***	22.6
S.J., ♀, 43 y (left adrenal carcinoma)	61.2		2.6 2.4		1283	20.9		
M.J., ♀, 13 y (left adrenal adenoma)	65.7	62.2	4.0	3.9	1518	23.1	2007****	32.2
F.C., ♂, 12 y (left adrenal carcinoma)	46.7	50.4	2.0 1.6	4.45	1041	22.3	2383+	47.3

* 2 months after bilateral total adrenalectomy on oral prednisone (10 mg/day).

** 3 months after bilateral total adrenalectomy on 50 mg cortisol/day (I.M.) and in "acute adrenal insufficiency".

*** 3 1/2 months after surgery on cortisol (50 mg) and d-aldosterone 1 mg, I.M., daily.

**** 1 month after adrenalectomy and maintained on oral prednisone 2.5 mg/day.

\+ 1 month after left adrenalectomy and maintained on oral prednisone 10 mg/day.

two other patients, with the severe form of the disease and low serum potassium (S.J. and F.C.), showed a lower Ke and Ke/Kg.

The patient with bilateral adrenal hyperplasia (M.R.S.), was studied 2 and 3 months after bilateral total adrenalectomy, at the time that she was deeply pigmented, suggesting either the development of a pituitary tumour (later shown not to be the case) or that the amount of replacement therapy was not sufficient. At both studies, Ke and Ke/Kg increased in relation to the pre-opera

tive values. The 2nd measurement, when the patient was on I. M. cortisol, 50 mg daily, she was hypotensive with the clinical signs of an adrenal crisis. Only after the addition of I. M. d-aldosterone, she was well maintained and 15 days afterwards there was a great decrease in Ke, from 1824 to 1269 mEq, and in the Ke/Kg ratio, from 33.2 to 22.6, still lower that the preoperative value. Afterwards, the patient was maintained on 75 mg daily of oral cortisone, and the body pigmentation was significantly reduced.

Patient M. J., with an adrenal adenoma, studied 1 month after adrenalectomy and maintained on prednisone (2.5 mg/day) showed a significant increase in both Ke and Ke/Kg without change in serum potassium levels.

Of the two patients with adrenal carcinoma who had the lowest exchangeable and serum potassium values, one of them (F.C.) was studied again 1 month after adrenalectomy and kept on prednisone, 10 mg daily. This patient showed a large increase in Ke and Ke/Kg and serum K which more than doubled, coincident with the marked improvement after treatment.

The effects of induced hypercortisonism is shown in table V. Exchangeable potassium, expressed as its absolute value and as the Ke/Kg ratio, was determined repeatedly before and after the treatment of a normal female volunteer, with a pharmacological dose of prednisone for 7 weeks, that caused severe muscle atrophy. Her exchangeable potassium decreased progressively to very low levels without change in serum potassium values. The last determination showed an increase in exchangeable potassium in relation to the previous one, but still greatly reduced in comparison to the mean control value.

In the first two weeks of prednisone treatment a comparison between the change in exchangeable potassium and the external

Table V - Exchangeable potassium (Ke) in a normal subject before and after induction of hypercortisonism with prednisone (40mg/day)

D.O., ♀, 41 years, Height : 160 cm. - Wt : 44.5 kg

Duration of treatment (Weeks)	Weight (kg)	Serum potassium (mEq/l)	Exchangeable potassium	
			mEq	mEq/kg
Control	44.5	4.0	1721	38.0*
2	42.2	3.9	1231	29.2
4	41.1	4.2	666	16.2
5	41.2	4.1	642	15.8
7	41.2	4.0	944	22.5

* Mean of 4 studies (range 36.0 to 40.2)

balance of potassium (diet, urine and fecal potassium measurements) was made. The change in Ke was + 490 mEq and in external balance was - 683 mEq (almost exclusively accounted for by urinary losses), the difference of body measurement from external balance being therefore of + 193 mEq. No further balance studies were performed.

DISCUSSION

As noted in previous publications 4,10 there is a wide range of normal values for Ke and Ke/Kg (table I) to a great extent related to individual differences in the electrolyte-free adipose tissue of the body. This factor of relative body contents of adipose tissue and lean body mass must be kept in mind in interpreting the values of exchangeable body potassium per Kg in patients with adrenocortical dysfunction. On the other hand, it is conceivable that any increase in body potassium in adrenocortical

insufficiency (Sheehan's and Addison's disease), to be expected from the experimental data [3,11], tends to be masked by its change in opposite direction which characterizes many chronic disease states: that is, body potassium stores would tend to fall as muscle mass decreases and concomitantly the extracellular space tends to expand absolutely and body sodium content increases[9]. This is probably the situation in the patients with Sheehan's disease kept on triiodothyronine replacement treatment whose control values for Ke and Ke/Kg are in the lower limits of the confidence interval for single normal values. In both Addisonian patients, in good health, there was normal pre-treatment values for Ke and Ke/Kg (patients M. G. and N.D., table III). Similar results were obtained by Brown et al.[2], in two patients with Addison's disease. In the patient R. M. in acute adrenal insufficiency we found, similar to Arons et al.[1] in their acutely ill patient the Ke/Kg was well within normal limits.

It is felt that the lower Ke and Ke/Kg values after 1 month of replacement therapy in patients M. G. and N. D. (table III) were mainly a reflection of the renal losses of potassium due the steroid treatment as we have seen in Addison's disease potassium balance becoming positive during cortisone withdrawal and subsequently negative on steroid resumption, until the patient enters potassium equilibrium after several days of therapy, without significant changes in serum potassium levels (unpublished data).

In patient R. M., examined during an crisis, the serum potassium concentration, abnormally high before treatment, fell but not to normal during the initial treatment period with cortisone. However, when mineralocorticoid deficit was corrected producing a normal serum potassium without a significant change in Ke (table III), the fall in serum potassium levels was probably the result of internal redistribution of potassium between fluid compartments

of the body and/or hemodilution following the increase of plasma volume due to sodium retention; the latter is suggested by a serum sodium concentration of 118.5 mEq/l at the time when the potassium level became normal and the patient had gained 1.1 kg since DOCA was started. The evidence is insufficient to show which of these factors were predominant in the fall of serum potassium.

The obese and protein depleted Cushing's syndrome patients had low levels of Ke and Ke/Kg (table IV) particularly in those with the greatest reduction of lean tissue manifested as muscular wasting and osteoporosis, developed most rapidly in malignant disease with decrease in serum potassium (patients S. J. and F. C., table IV).

In those patients with hypercortisonism (Cushing's syndrome) studied after therapeutically induced remission, there was an increase in Ke, as previously reported by Ernest[5], without a change in serum potassium in those cases normal values before treatment. In patient F. C., with low serum potassium, the increase in Ke was associated with normal concentration in the serum.

The effect of large doses of prednisone, on a short-term basis, in patients with Sheehan's disease and for a longer period in a normal female subject, caused the opposite results: a decrease in Ke and Ke/Kg in the normal subject (table V) with a decrease in body weight and a significant increase in Ke in all patients with Sheehan's disease with a gain in body weight in half of them (table II). The increase in Ke is interpreted as a reflection of an increase in body cell mass and indicating an improvement of the disease with the subsequent increase in food intake and body weight. In animals[8], the amount of dietary calories and protein can modify the nitrogen balance during cortisone treatment and that an initial negative balance may improve during pro

longed treatment. Kyle et al. [6] have shown that dietary intake are of decisive importance for the body composition in patients with Cushing's syndrome. Finally, Lindholm[7] has shown that patients with bronchial asthma under synthetic cortisol analogues treatment for more than two years, in a daily dose corresponding to 25-72 mg of cortisone, did not show a decrease in Ke and some showed increase in of body composition during which their diet was adequate due to improvement of their asthma.

The increase in Ke and Ke/Kg in all patients with Sheehan's disease after the treatment with prednisone, suggests the patient's gain in potassium-rich lean body mass as the Ke/Kg ratio change was not significant.

The opposite effect observed in the normal control, was certainly related to her low dietary intake with loss of body weight. In this patient the discrepancy between potassium balance and exchangeable potassium in the first 2 weeks of steroid treatment, suggests that previously non-exchangeable depots of potassium may have become exchangeable during the period of hormone administration. However, the difference observed between the balance and Ke was not sufficiently large to rule out inherent errors of determining the balance. Arons et al.[1] have also noticed instances of significant discrepancies between potassium balance and body potassium measurements and have postulated similar explanations.

In favor of this concept is the study reported by Rundo and Sagild[12] that the value for body potassium obtained by isotope dilution represented 85% of the total body potassium estimated by total body gamma counting. However, Talso et al.[14] obtained a better relationship between the value for exchangeable potassium and the value for ^{40}K reported by Rundo and Sagild[12], the Ke representing 97% of the total body potassium.

Therefore, the data obtained from these studies suggest that the resultant changes in body potassium content must represent a composite of increases or decreases in body fat and potassium rich muscle tissue non-specific nutritional effects, as well as any specific action of adrenal steroids on internal distribution of potassium between intra-and extra cellular compartments and/or on the renal transport of the cation.

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RESUMO

O potássio total permutável (Ke), utilizando-se ^{42}K, foi determinado em pacientes a) com insuficiência suprarenal primária e secundária, antes e após o tratamento; b) em que se induziu hipercortisonismo; c) com síndrome de Cushing antes e após a remissão terapêutica; d) o efeito de altas doses de prednisona, administrada por 7 semanas a um indivíduo normal, foi também avaliado. O Ke e/ou o Ke/Kg é normal em todos pacientes com insuficiência suprarenal não tratados. O Ke diminuiu nestes pacientes após tratamento com a cortisona. Nos pacientes em crise, a adição de DOCA corrigiu o potássio sérico, sem alterar o Ke, o que sugere uma redistribuição interna de potássio e/ou uma hemodiluição.

O hipercortisonismo secundário à prednisona (40 mg/dia por 21 dias) é diferente nos pacientes com moléstia de Sheehan daquêle induzido em uma paciente normal; enquanto há um aumento significativo de pêso nos pacientes com hipopituitarismo, há uma diminuição de pêso e de Ke na paciente normal. O aumento de Ke parece refletir um aumento na massa celular, rica em potássio, devido a ingestão alimentar, e não um efeito específico dos corticoesteróides sôbre o metabolismo de potássio.

O Ke na síndrome de Cushing é diminuído, sobretudo nos pacientes com carcinoma da suprarenal e hipocalemia. Após o tratamento o Ke aumenta.

As alterações do potássio corpóreo refletem flutuações do tecido adiposo e do tecido rico em potássio, sob a ação de fatôres nutricionais não específicos e também sob a ação do efeito específico de glicocorticoides sôbre a distribuição do potássio entre extra e o intracelular e/ou o transporte renal dêste catíon.

RÉSUMÉ

Le potassium échangeable (Ke) est déterminé par le ^{42}K chez des malades en insuffisance surrénale primitive ou secondaire, avant et après traitement, ou après l'induction d'un hypercorticisme. Le Ke est aussi mesuré dans 4 maladies de Cushing, et, dans trois de ces cas, après traitement. L'effet de fortes doses de prednisone, données pendant sept semaines, sur le Ke chez un sujet normal, est aussi évalué. Le Ke et/ou le Ke/Kg est normal chez tous les insuffisants surrénaux non traités. Le Ke a diminué chez ces malades après traitement par la cortisone. Pour les malades en crise, l'addition de DOCA a corrigé le potassium sérique, sans changer le Ke, ce qui suggère une redistribution interne du potassium et/ou une hémodilution.

L'hypercorticisme secondaire à la prednisone (40 mg/jour pendant 21 jours) est différent chez les malades avec nécrose hypophysaire du post-partum (maladie de Sheehan) de ce qu'il est chez une femme normale: il y a accroissement significatif du Ke, avec prise de poids chez la moitié des malades présentant un hypopituitarisme, alors que le poids et le Ke diminuent chez le sujet normal. L'accroissement du Ke semble être le reflet de l'augmentation de la masse cellulaire riche en potassium, elle-même due à l'ingestion d'aliments, et non un effet spécifique des stéroides surrénaux sur le métabolisme du potassium.

Le Ke, dans les maladies de Cushing, est diminué surtout dans deux cas avec carcinome surrénalien et hypocalcémie. Après traitement, le Ke s'élève. Ces modifications du potassium corporel doivent être le reflet des fluctuations des tissus graisseux et des tissus riches en potassium sous l'effet de facteurs nutritionnels non spécifiques, et aussi de l'effet spécifique des glucocorticoides sur la distribution du potassium entre les secteurs intra et extra cellulaires et/ou le transport rénal de ce cation.

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