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Total body potassium in cor pulmonale

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Howie, A. D., Pack, A. I., Boddy, K., and Mahaffey, Maureen (1976). *Thorax*, **31**, 708–712. **Total body potassium in cor pulmonale.** Total body potassium was measured in 12 patients with cor pulmonale, by determination of potassium-40, a naturally occurring radioisotope. In all subjects the observed value of total body potassium showed no significant depletion when compared with that predicted from height and age. All previous studies in similar groups of patients have been confined to the estimation of exchangeable potassium by the technique of isotope dilution. Results using the latter technique have shown gross potassium depletion. It is suggested that the apparent disparity between total body potassium and exchangeable potassium could be explained by the requirement for a longer equilibration period when using isotope dilution techniques in patients who are in a chronic hypoxaemic state.

Studies of body electrolyte composition in patients suffering from chronic airway obstruction have suggested that gross potassium depletion can occur. These studies have been confined to the measurement of exchangeable potassium (Ke) by the technique of isotope dilution after the administration of potassium-42 (^{42}K), which has a half-life of 12·4 hours (Bauer *et al.*, 1966; Telfer *et al.*, 1968; Schloerb *et al.*, 1970; Campbell *et al.*, 1975; Telfer, Weiner, and Merrill, 1975).

The interpretation of the results obtained by the isotope dilution technique is dependent on equilibration having been reached between the administered radioactive isotope and the native potassium. In the present study, the problem of equilibration was avoided by directly measuring the total body potassium (TBK) with a whole-body counter in a group of patients with cor pulmonale. The results were compared with predicted normal values.

PATIENTS

Twelve subjects, 11 men and one woman, were investigated. All had established irreversible airway obstruction, judged from spirometric testing, and chronic bronchitis according to the Medical Research Council questionnaire on respiratory symptoms (Medical Research Council, 1960). Their mean age was 58·8 (range 49–75) years. All had previously had at least one episode of acute respiratory failure complicated by raised jugular venous pressure and peripheral oedema, but these were in a chronic compensated state of their respiratory disease and free from oedema. Subjects with a blood urea greater than 10 mmol/l were excluded. Ten patients were receiving drug therapy which was discontinued 12 hours before the study. Medications included digoxin, frusemide, Slow-K, spironolactone, beclomethasone, salbutamol, and thyroxine. The patient on beclomethasone had established irreversible airway obstruction with an FEV_1/FVC ratio of 30% before and after bronchodilators. It was, therefore, thought to be justified to include her in our series. The subject on thyroxine was clinically and biochemically euthyroid, and thus there is no reason to suspect that this would alter the results. All patients were active and ambulant and had been on their usual diet and fluid intake before the beginning of the study. The clinical data are shown in Table I.

TEST PROCEDURES

Total body potassium was measured using the Merlin mobile whole-body monitor (Boddy, 1967) with a sodium iodide detector, 29·2 cm diameter by 10·2 cm deep. A shadow-shield surrounded the detector housed in a central turret. The shield is

TABLE I

CLINICAL FEATURES OF PATIENTS INCLUDED IN STUDY

Patient	Age (years)	Height (cm)	Weight (kg)	Sex	Diagnosis	Drug Therapy	Duration of Diuretic Therapy (years)	FVC (litres)	FEV_1 (litres)
1	63	162·6	56·7	M	CB[1]	Digoxin, Frusemide 40 mg	4	1·29	0·59
2	61	163·3	51·3	M	CB	Frusemide 40 mg, Slow-K	4	2·66	1·22
3	62	182·9	81·2	M	CB	Frusemide 40 mg, Slow-K	7	3·40	0·92
4	50	167·6	81·6	M	CB	Frusemide 40 mg, Slow-K	3	2·90	1·39
5	49	179·1	75·8	M	CB	—	—	2·97	0·86
6	49	160·7	64·4	M	CB	—	—	1·82	0·69
7	59	167·6	46·8	M	CB	Frusemide 40 mg, Slow-K	2	1·62	0·63
8	57	139·7	48·4	F	CB	Salbutamol, Beclomethasone	—	1·16	0·36
9	59	161·3	56·0	M	CB	Digoxin, Frusemide 80 mg, Slow-K	5	1·98	0·83
10	64	170·2	51·9	M	CB	Salbutamol	—	3·30	1·29
11	75	170·2	73·0	M	CB	Frusemide 40 mg, Slow-K	1	1·70	0·65
12	58	177·8	80·7	M	CB	Salbutamol, Thyroxine	—	2·57	0·92
Mean	58·8	166·9	64·0						

[1]CB = chronic bronchitis.

10 cm thick lead bricks with a total weight of less than 8000 kg. The patient, lying supine on a motorized couch, passed beneath the detector and was scanned from head to feet. The output was taken to a TMC 400-channel pulse height analyser. After printout of the accumulated data, the patient adopted the prone position, the direction of travel of the couch was reversed, and the patient was scanned from feet to head. The data were again recorded, and mean values were obtained for the two positions. The sensitivity obtained was comparable with that of the conventional steel or lead room monitors. The subject counting rate in the potassium-40 photopeak was expressed as millimoles of potassium without the administration of a radioactive isotope using the procedure described by Boddy *et al.* (1971). The estimated coefficient of variation of this procedure was shown to be 3·9% for a subject having 3600 mmol of potassium.

Expected total body potassium for each subject was estimated from height and age, and age, height, and weight using the regression equations derived for a healthy population (Boddy *et al.*, 1972a). The equations are:

(a) Total body potassium based on height and age:

Males: mmol potassium =

(53·02 × height in cm) − (9·74 × age in years) − 5305

Females: mmol potassium =

(33·63 × height in cm) − (7·73 × age in years) − 2727

(b) Total body potassium based on height, weight, and age:

Males: mmol potassium =

(23·96 × weight in kg) + (35·15 × height in cm) − (12·09 × age in years) − 3762

Females: mmol potassium =

(14·76 × weight in kg) + (22·07 × height in cm) − (9·05 × age in years) − 1669

The standard deviation from regression for height and age are 9·3% for males and 8·8% for females, and for height, weight, and age are 7·3% for males and 7·8% for females.

The measured total body potassium in each patient was compared with the predicted values using a modification of the zM test to establish statistical significance, or otherwise, of the difference.

Blood for blood gas and plasma electrolyte estimation was obtained from an indwelling cannula inserted into the brachial artery. Each patient was rested in the sitting position for at least 15 minutes. Two arterial samples were taken at 30-minute intervals to ensure that stable results had been obtained. Informed consent was obtained from all patients in the study.

RESULTS

Blood gas measurements (Table II) revealed hypoxaemia in all the patients ($Pao_2 < 9·8$ kPa) with a mean Pao_2 of 7·43 kPa. The arterial carbon dioxide tension ($Paco_2$) was frequently increased, but in all patients the plasma potassium was within normal limits. The values obtained for total body potassium, as measured by the whole-body monitor and those calculated from height and age, and height, weight, and age, are shown in Table III along with the relevant anthropo-

TABLE II

MEASUREMENTS OF ARTERIAL BLOOD GASES AND ELECTROLYTES

Patient	pH	Pa_{O_2} (kPa)	Pa_{CO_2} (kPa)	Plasma Electrolytes (mmoles/l)				
				K	Na	Cl	HCO_3	Urea
1	7·39	6·74	7·12	4·2	140	100	31	3·9
2	7·48	9·02	5·92	3·9	141	102	—	6·5
3	7·41	9·19	6·13	3·8	140	102	28	6·2
4	7·38	6·82	6·90	4·5	142	100	23	4·0
5	7·42	7·97	4·91	4·0	139	101	26	4·6
6	7·34	5·15	7·00	4·3	142	102	30	5·7
7	7·37	5·80	9·31	3·6	139	92	34	6·2
8	7·36	6·72	8·41	4·2	143	99	29	4·2
9	7·43	5·56	6·44	3·8	140	93	36	5·9
10	7·40	9·76	6·07	4·0	141	105	27	5·8
11	7·49	6·98	5·72	3·8	140	98	26	8·9
12	7·40	9·47	5·60	3·6	142	100	28	6·1

TABLE III

MEASUREMENT OF TOTAL BODY POTASSIUM IN 12 PATIENTS WITH CHRONIC COR PULMONALE

Patient	Observed TBK (mmol)	Predicted TBK (mmol) (height, age)	P[1]	Predicted TBK (mmol) (height, weight, age)	P[1]
1	2920	2701	0·53	2550	0·18
2	2527	2760	0·50	2471	0·84
3	3241	3788	0·14	3865	0·04
4	3732	3095	0·06	3481	0·36
5	3614	3714	0·77	3757	0·60
6	3024	2736	0·40	2834	0·49
7	2487	3005	0·13	2537	0·86
8	2245	1529	0·005	1611	0·005
9	3063	2673	0·26	2537	0·06
10	2726	3095	0·29	2691	0·90
11	3079	2987	0·80	3064	0·96
12	3918	3558	0·31	3721	0·48
Mean	3048	2970		2927	

TBK = total body potassium; Predicted values for TBK are calculated from the data of Boddy *et al* (1972a).
[1]P based on a modification of the zM test (see text).

metric data. In each case the observed total body potassium was not significantly less than the predicted value calculated from the height and age, and in only one subject (no. 3) was the observed level significantly less ($P<0·05$) than the value predicted from height, weight, and age. In addition, in one patient (no. 8) the observed total body potassium was significantly greater than predicted, which suggests that the sample of patients studied was comparable with the normal population.

If the eight patients in respiratory failure with a Pa_{O_2} of less than 8 kPa are considered separately from the whole group, the observed total body potassium is still not significantly less than that predicted from height and age.

DISCUSSION

In this study we have shown that the total body potassium in all subjects was not significantly less than that predicted from height and age. This finding was surprising as in all previous reports where exchangeable potassium had been measured in similar groups of patients it was found to be significantly less than predicted normal values (Bauer *et al.*, 1966; Telfer *et al.*, 1968; Schloerb *et al.*, 1970; Campbell *et al.*, 1975; Telfer *et al.*, 1975). Our patients were clinically similar to those included in these other series and had similar blood gas abnormalities (Table IV).

The equilibration period used in these studies was 24 and 48 hours. Furthermore, as a result of demonstrating low exchangeable potassium in patients with chronic respiratory failure, Schloerb *et al.* (1970) proceeded to correct the apparent potassium deficiency with potassium supplements, while admitting that there was no evidence of functional improvement. It is, therefore, of considerable clinical importance to examine the relevance of low exchangeable potassium in patients with chronic airways obstruction, especially in the light of our results.

The difference between the results obtained for total body potassium and exchangeable potassium

TABLE IV

COMPARISON OF ARTERIAL BLOOD GASES IN PATIENTS STUDIED BY DIFFERENT AUTHORS

Authors	PaO_2 (kPa)			$PaCO_2$ (kPa)			pH		
	Mean		SD	Mean		SD	Mean		SD
Schloerb *et al* (1970)	8·57	±	2·11	6·89	±	2·03	7·42	±	0·03
Telfer *et al* (1975)	7·18	±	1·33	6·65	±	1·46	7·40	±	0·05
Campbell *et al* (1975)	6·80	±	1·10	7·40	±	1·10	Not stated		
Present study	7·43	±	1·60	6·63	±	1·23	7·41	±	0·01

in cor pulmonale could be explained by the need for a longer period of equilibration when studying exchangeable potassium by the isotope dilution technique. This explanation is supported by the work of Telfer *et al.* (1975). The value for exchangeable potassium which they obtained at 48 hours was 20% greater than that at 24 hours but still significantly less than the predicted normal values. This finding is analogous to that of Boddy *et al.* (1972b) in patients with chronic renal failure. They compared total body potassium determined by whole-body monitoring with exchangeable potassium using ^{43}K which has a half-life of 22 hours, in the same group of patients. Though total body potassium was within the normal range, the value for exchangeable potassium at 64 hours was 2·3% greater than that at 48 hours and 25·2% greater than that at 24 hours but still 14% less than the predicted normal value.

It is possible that the exchangeable potassium is a smaller fraction of the total body potassium in patients with cor pulmonale than in healthy subjects. Studies in normal subjects have suggested that exchangeable potassium is 92% to 97% of total body potassium (Talso *et al.*, 1960; Remenchik and Miller, 1962; Surveyor and Hughes, 1968), whereas in patients with chronic renal failure, for example, exchangeable potassium at 24 hours was 60·7% of the total body potassium, and at 64 hours was 85·9%.

It is unlikely that diuretics or other drug therapy affected our results. Dargie *et al.* (1974) showed that patients with essential hypertension and normal renal function, who had been on 40 mg frusemide daily with or without potassium supplementation, showed no evidence of depletion of total body potassium.

Other workers have suggested that the low values obtained for exchangeable potassium could be explained by changes in body weight. Campbell *et al.* (1975) observed body weight and exchangeable potassium in patients with cor pulmonale after treatment of the acute illness when the oedema had disappeared but while the patients were still on oxygen therapy. Six patients were then re-examined two to three months later during convalescence. They concluded that the low values of exchangeable potassium obtained could be explained by loss of tissue mass. However, this does not explain why exchangeable potassium remained reduced in convalescence even though in most instances weight gain was almost complete. In two patients in particular there was an increase in dry body weight in convalescence but a further reduction in exchangeable potassium from the value obtained during the initial assessment. If low exchangeable potassium in patients with cor pulmonale is to be explained mainly on the basis of tissue loss, then it is surprising that in all our patients the total body potassium showed no significant depletion when compared with normal values predicted from height and age.

The results obtained for total body potassium and exchangeable potassium in patients with cor pulmonale are conflicting. It may be that a longer equilibration period is required or that the exchangeable potassium fraction is smaller in patients with chronic respiratory disease, and that these are the main reasons for the disparity between total body potassium measured in the whole-body monitor and exchangeable potassium measured by the isotope dilution technique.

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