Plasma potassium in patients with terminal renal failure during and after haemodialysis; relationship with dialytic potassium removal and total body potassium

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Abstract

Background. Chronic haemodialysis (HD) patients may present with severe predialysis hyperkalaemia which is improved by dialytic treatment. However, factors influencing the behaviour of postdialysis plasma potassium (plasma K) are not well known.

Methods. In this prospective study 14 patients (7 female, 7 male) on chronic HD were investigated during a standardized 4-h HD with a 2 m² high-flux dialyser and up to 6 h postdialysis. Dialytic potassium removal was measured by dialysate collection. Total body potassium (TBK) was measured by whole-body counting of $^{40}$K.

Results. Plasma K declined from 5.65 to 3.62 mmol/l on HD. In spite of a total dialytic removal of 107 mmol of potassium plasma K rose to 5.01 mmol/l 6 h postdialysis. TBK, as adjusted for age, was 38.2 and 49.0 mmol/kg BW in female and male patients respectively, i.e. in the normal range. Of a total potassium removal of 107 mmol on HD only 42% originated from the extracellular space. Dialytic potassium removal was best correlated with removal of intracellular potassium but also with extracellular potassium content and with the product of plasma K × TBK. The 6-h postdialysis plasma K was correlated with the predialysis value but not with TBK or dialytic potassium removal.

Conclusion. A rather high dialytic removal of potassium (which is correlated with plasma K × TBK) does not necessarily prevent a rapid postdialysis rebound of plasma K. Therefore patients with marked hyperkalaemia should be monitored closely postdialysis. TBK can be normal in haemodialysis patients who are well nourished.

Key words: hyperkalaemia; plasma potassium; potassium removal on haemodialysis; total body potassium

Introduction

Due to diminished renal potassium excretion as well as impaired extrarenal potassium tolerance [1–3] patients with chronic renal failure are at a high risk of hyperkalaemia. Patients treated by maintenance haemodialysis regularly accumulate potassium during the period between dialyses, and potassium removal is one of the major functions of chronic haemodialysis. Although a postdialysis rebound of the plasma concentrations of several substances has been described [4,5] we have been struck by the rapidity and the degree of the rise of plasma potassium (plasma K) in some haemodialyzed patients in spite of the use of a large surface area dialyser and a dialysis time of 4 h. The purpose of the present study was to investigate this phenomenon systematically and to relate it to potassium removal by dialysis and to total body potassium (TBK).

Subjects and methods

Fourteen patients with terminal renal failure receiving maintenance haemodialysis were studied prospectively. They were selected because they repeatedly, although not regularly, had demonstrated predialysis plasma K levels of ≥5.5 mEq/l. There were seven women and seven men, aged 31–72 years (mean age 51.1 ± 4.0 (SEM) years). Thirteen were Caucasians and one was African. Four patients suffered from non-insulin-dependent diabetes mellitus (two with diabetic nephropathy, one each with analgesic nephropathy and polycystic kidney disease), two more from analgesic nephropathy, two each from chronic glomerulonephritis and chronic interstitial nephritis of unknown origin and one each from insulin-dependent diabetes mellitus with diabetic nephropathy, Alport’s syndrome, malignant nephrosclerosis, chronic pyelonephritis, and bilateral nephrectomy for renal cell carcinoma. Thirteen patients were oliguric (<100 ml/24 h) or anephric, one patient with some residual diuresis collected his urine and excreted 4 mmol of potassium over the entire study period. Some anthropological data are presented in Table 1.

The patients were dialysed three times weekly for 4–5 h using high-flux polysulphone or polyacrylonitrile haemodia-

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and potassium intake. All of them had been repeatedly (The difference between the ECV protein/kg body weight and to moderately restrict sodium ECV converting enzyme inhibitors or potassium binding exchange where ECV of ultrafiltrate during dialysis. The system has been evaluated and bicarbonate concentration was calculated with the volume is a representative portion of the whole volume samples immediately after withdrawal with an AVL-995 calibrated nozzles. From one of these nozzles dialysate is AVL, Graz, Austria). Blood pH and partial pressure of trate is constantly collected in a cylindrical reservoir and Plasma as well as dialysate potassium were measured with the system [6]. With this system the dialysate and ultrafiltration in plasma at the beginning and where Kt

Study design

It was our purpose to study the patients under their normal everyday condition. For this reason food was allowed as usual. The measurements were performed after the weekend on the first weekly haemodialysis (i.e. on a Monday or Tuesday). The patients took their normal light breakfast at home. Upon arrival at the dialysis centre two standard haemodialysis fistula needles were placed, and the needle close to the arteriovenous anastomosis was used for blood sampling. A standard 4-h haemodialysis was performed using a polycrylonitrile (PAN) membrane capillary dialyser (Filtral 20, Hospal, France) with a surface area of 2.0 m² and a dialysate with 1 mmol/l of potassium and 40 mmol/l of bicarbonate; 5.5 mmol/l of glucose was added for the five patients with diabetes mellitus. The blood pump was set at 300 ml/min, the dialysate flow was approximately 500 ml/min. The same Fresenius 4006 dialysis monitor (Fresenius Bad Homburg, Germany) was applied for each patient.

To measure potassium removal during dialysis dialysate and ultrafiltrate was collected with a partial dialysate collection system [6]. With this system the dialysate and ultrafiltrate is constantly collected in a cylindrical reservoir and then passes into a chamber from which it flows out through 25 calibrated nozzles. From one of these nozzles dialysate is collected in a canister, the rest is discarded. The collected volume is a representative portion of the whole volume regardless of variations of the dialysate flow and the amount of ultrafiltrate during dialysis. The system has been evaluated by comparing sample/total dialysate ratio and has been found to be very accurate. It was calibrated before each study haemodialysis. Dialysate was collected in four 60-min samples. The collected volume was weighed and expressed in millilitres (assuming 1 g = 1 ml). Potassium concentration was measured in fresh inflow dialysate (1.06±0.01 mmol/l, mean ± SEM) and in the collected dialysate; potassium removal (Kt) was then calculated using the formula

\[
K_t = V_i \cdot f(K_c - K_o)
\]

(where \(V_i = \) collected volume, \(f = \) multiplication factor as obtained by calibration, \(K_c = \) potassium concentration in collected dialysated, \(K_o = \) potassium concentration in fresh dialysate).

Potassium removed by ultrafiltration was calculated for the 4-hourly collection periods using the formula [7]

\[
Q_{uf} = \frac{C_{ub} - C_{uf}}{\ln(C_{ub}/C_{uf})}
\]

(where \(Q_{uf} = \) ultrafiltration rate, \(C_{ub} = \) potassium concentration in plasma at the beginning and \(C_{uf} = \) potassium concentration in plasma at the end of the collection period). Ultrafiltration volume was estimated from the difference between predialysis and postdialysis weights.

Total body water (TBW) was calculated according to Hume and Weyers [9] using body surface area. Extracellular volume (ECV) was assumed to be 1/3 of TBW. Removal of potassium from the extracellular space (ECV) was calculated as follows:

\[
ECV_0 \cdot K_c - ECV_4 \cdot K_4
\]

where ECV0 = ECV before dialysis

\[
ECV_4 = ECV \text{ after dialysis}
\]

(The difference between the ECV0 and ECV4 being the ultrafiltration volume)

Therefore, removal of potassium from the intracellular space can be calculated as follows:

\[
K_t = K_i = (ECV_0 \cdot K_c - ECV_4 \cdot K_4)
\]

where \(K_i = \) potassium originating from the intracellular space

\[
K_{it} = \text{total potassium removed by dialysis}
\]

In the 60 min after haemodialysis the patients were served a standard meal containing 700 mg (17.5 mmol) of potassium. If patients felt hungry during the 6 postdialysis hours they were further allowed a light snack (coffee or tea and biscuits).

Basal levels of blood gas parameters, potassium, sodium, plasma glucose, insulin and osmolality were measured before the start of haemodialysis. Thereafter, blood potassium was measured at 1, 2, 3 and 4 h of dialysis and hourly up to 6 h after dialysis. Blood gas parameters, plasma glucose, insulin, and osmolality were further determined at the end of and 6 h after dialysis.

This study was approved by the ethical committee of Kantonsspital Aarau, Switzerland.

Analytical procedures

Plasma as well as dialysate potassium were measured with an ion-selective electrode (AVL 987-S Electrolyte analyser, AVL, Graz, Austria). Blood pH and partial pressure of carbon dioxide were determined in anaerobically handled samples immediately after withdrawal with an AVL-995 automatic blood gas analyser system (AVL, Graz, Austria) and bicarbonate concentration was calculated with the

Table 1. Anthropological patient data

<table>
<thead>
<tr>
<th>Pt No.</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Height (cm)</th>
<th>Weight (kg)*</th>
<th>TBW (l)*</th>
<th>ECV (l)*</th>
<th>UF (l)*</th>
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<tbody>
<tr>
<td>1</td>
<td>37</td>
<td>M</td>
<td>169</td>
<td>57.0</td>
<td>35.8</td>
<td>11.9</td>
<td>3.9</td>
</tr>
<tr>
<td>2</td>
<td>72</td>
<td>F</td>
<td>159</td>
<td>58.0</td>
<td>30.2</td>
<td>10.1</td>
<td>3.7</td>
</tr>
<tr>
<td>3</td>
<td>53</td>
<td>F</td>
<td>161</td>
<td>53.0</td>
<td>29.9</td>
<td>10.0</td>
<td>2.9</td>
</tr>
<tr>
<td>4</td>
<td>37</td>
<td>F</td>
<td>160</td>
<td>62.5</td>
<td>31.6</td>
<td>10.5</td>
<td>5.5</td>
</tr>
<tr>
<td>5</td>
<td>45</td>
<td>M</td>
<td>180</td>
<td>104.0</td>
<td>51.9</td>
<td>17.3</td>
<td>4.7</td>
</tr>
<tr>
<td>6</td>
<td>59</td>
<td>M</td>
<td>182</td>
<td>82.0</td>
<td>46.6</td>
<td>15.5</td>
<td>4.4</td>
</tr>
<tr>
<td>7</td>
<td>34</td>
<td>M</td>
<td>174</td>
<td>68.5</td>
<td>40.2</td>
<td>13.4</td>
<td>3.6</td>
</tr>
<tr>
<td>8</td>
<td>31</td>
<td>F</td>
<td>157</td>
<td>52.0</td>
<td>28.4</td>
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<td>3.7</td>
</tr>
<tr>
<td>9</td>
<td>72</td>
<td>F</td>
<td>163</td>
<td>75.5</td>
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<td>11.6</td>
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</tr>
<tr>
<td>10</td>
<td>68</td>
<td>F</td>
<td>155</td>
<td>52.7</td>
<td>27.8</td>
<td>9.3</td>
<td>2.6</td>
</tr>
<tr>
<td>11</td>
<td>62</td>
<td>M</td>
<td>172</td>
<td>88.0</td>
<td>46.8</td>
<td>15.6</td>
<td>3.9</td>
</tr>
<tr>
<td>12</td>
<td>38</td>
<td>M</td>
<td>177</td>
<td>66.0</td>
<td>40.0</td>
<td>13.3</td>
<td>4.1</td>
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<tr>
<td>13</td>
<td>41</td>
<td>M</td>
<td>176</td>
<td>60.0</td>
<td>38.1</td>
<td>12.7</td>
<td>1.1</td>
</tr>
<tr>
<td>14</td>
<td>66</td>
<td>F</td>
<td>159</td>
<td>58.0</td>
<td>30.2</td>
<td>10.1</td>
<td>3.2</td>
</tr>
</tbody>
</table>

*Postdialysis (‘dry weight’); TBW, total body water; ECV, extracellular volume; UF, ultrafiltration volume.

lyers with a surface area of 1.8–2.4 m². Dialysis time and size of dialyser were selected according to body mass. All patients continued to receive their normal medication including phosphate binders (calcium salts) and in some cases benzoazepines at bedtime; six patients received a small dose of the cardiospecific \( \beta \)-blocker atenolol (2 times 25 mg/week to 50 mg/day), and two received amlodipine for treatment of hypertension, but none was taking angiotensin-converting enzyme inhibitors or potassium binding exchange resins. The patients were encouraged to eat at least 1.2 g of protein/kg body weight and to moderately restrict sodium and potassium intake. All of them had been repeatedly instructed about potassium-rich foods.
Henderson–Hasselbalch equation. Plasma glucose, osmolality, and insulin were measured as described previously [9], serum albumin was determined immunochemically.

Total body potassium measurement

Total body potassium (TBK) was measured on a dialysis-free day of the same week, i.e. 1 or 3 days after the dialysis study.

TBK was assessed by measurement of the patients’ K-40 (potassium isotope 40) contents with the whole-body counter (detectors from Quartz & Silice, France) of the University Hospital Basel. The photons from the 1.46 MeV γ-emission line of the radioactive K-40 were detected and the counts analysed with two NaI-detectors of 20 cm diameter and 10 cm thickness in a well-shielded counting area. The two detectors, one from above and one from beneath, scanned the patient horizontally in the course of 24 min. The calibration of the detection system for the absolute determination after haemodialysis was performed by scanning a phantom of known K-40 contents (bottles of K-40 solution). The minimal detectable activity for K-40 amounts to 190 Bq, which corresponds to 6.3 g or 161 mmol of natural K. The statistical error is estimated to 3% and the systematic error to approximately 10%. The total body potassium (all isotopes of K) was subsequently calculated according to the following formula:

$$M(TBK) = \frac{M_K \cdot A \cdot T_h}{\ln(2) \cdot \alpha \cdot N_A},$$

where $M$ (TBK) is the mass of the total body potassium, $M_K$ is the atomic mass of natural potassium, $A$ is the measured K-40 activity, $T_h = 1.28 \cdot 10^9$ years is the half-life of K-40, $N_A = 6.02 \cdot 10^{23}$ is the Avogadro number and $\alpha = 1.17 \cdot 10^{-4}$ is the natural abundance of the K-40.

Total body potassium was measured on a dialysis-free day of the same week, i.e. 1 or 3 days after the dialysis study.

Statistical analysis

Comparison of variation over time was by analysis of variance and also by paired $t$ test. Pearson correlation analysis was used for assessment of relationships between variables. Values are given as mean ± SEM.

Results

Effects of haemodialysis on plasma $K$

Mean predialysis plasma $K$ was 5.65 ± 0.14 mmol/l. During haemodialysis plasma $K$ declined continuously reaching a nadir of 3.57 ± 0.12 mmol/l after 3 h and remaining stable (3.62 ± 0.09 mmol/l) at 4 h (Figure 1).

Total removal of potassium by the dialysis procedure was highest during the 1st hour (34.2 ± 1.7 mmol) and significantly lower during the 4th hour (21.0 ± 1.8 mmol, $P < 0.001$). Total cumulative potassium removal by diffusion and convection amounted to 107.1 ± 6.0 mmol, potassium removal by diffusion alone to 92.8 ± 5.3 mmol (Figure 2); ultrafiltered potassium was 14.3 ± 1.4 mmol, (the mean total ultrafiltration volume was 3.5 ± 0.3 litres).

It was calculated that 44.6 ± 3.0 mmol (= 41.6%) of removed potassium originated from the extracellular space whereas the rest had its provenance in the intracellular space. No significant correlation between predialysis plasma $K$ and cumulative total potassium removal (or potassium removed by diffusion) could be demonstrated. However, total extracellular potassium (i.e. $ECV_d \cdot K_d$) was correlated with total potassium removal ($r = 0.65$, $P < 0.05$). The closest correlation was found between potassium originating from the intracellular space ($K_i$) and total potassium removal ($r = 0.87$, $P < 0.001$).

Plasma potassium after haemodialysis

After haemodialysis plasma potassium rose quite rapidly during the 1st hour (from 3.62 ± 0.09 to 4.33 ± 0.14 mmol/l, $P < 0.001$) and steadily thereafter up to 5.01 ± 0.12 mmol/l 6 h postdialysis (Figure 1). A
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further rise to $5.64 \pm 0.16$ mmol/l ($P < 0.001$) occurred during the following 38 h, i.e. the interval to the next haemodialysis. The postdialysis rise $\Delta K$ was not correlated with TBK, pre- or postdialysis plasma K (or the product of TBK and plasma K) nor with the amount of potassium removed. However, there was a close correlation between the predialysis and the 6-h postdialysis plasma K concentrations ($r = 0.78$, $P < 0.01$). The correlation equation was $K_{6} = K_{0} \cdot 0.658 + 1.294$ (where $K_{0} =$ predialysis and $K_{6} =$ 6-h postdialysis plasma K). Accordingly, this postdialysis rebound of plasma K was particularly impressive in the patient with the highest predialysis potassium of 6.91 mmol/l. Although a total of 140.5 mmol of potassium were removed during haemodialysis and his plasma potassium fell to 4.16 mmol/l postdialysis, there was a rise to 6.12 mmol/l 6 h later (Figure 3).

Blood gas parameters plasma glucose, insulin and osmolality (see Table 2)

Plasma pH and bicarbonate increased significantly during haemodialysis. Bicarbonate remained stable thereafter, pH declined slightly. Plasma glucose decreased moderately during dialysis, whereas no significant changes in insulin concentrations occurred. Plasma osmolality fell significantly on dialysis and remained stable thereafter.

Total body potassium (TBK)

TBK was lower in female than in male patients ($P < 0.001$). However, factoring total body potassium by dry body weight (BW) decreased ($P < 0.05$), relating it to TBW eliminated the significance. Measured TBK/kgBW was compared with age-adjusted levels as described by Pierson et al. [10] and was found to be close to normal (Table 3).

No significant correlation between total TBK and total cumulative dialytic potassium removal was demonstrated; however, potassium removal by the process of diffusion alone was weakly correlated with TBK ($r = 0.63$, $P < 0.05$). The product of TBK and predialysis plasma K was significantly correlated with total dialysed potassium removal ($r = 0.76$, $P < 0.01$, Figure 4a) as well as with potassium removed by diffusion alone ($r = 0.70$, $P < 0.01$, Figure 4b).

Discussion

Our patients were studied under their usual dietary conditions; fasting was avoided since this has been shown to have an unfavourable effect on hyperkalaemia [11]. The rather high interdialytic weight gain of 3.5 l was due to our liberal dietary prescriptions favouring good nutrition, which has been found to be important to the health of chronic haemodialysis patients [12].

During dialysis a rapid fall in plasma potassium was induced. The total cumulative removal of potassium by haemodialysis was somewhat higher than in previous studies [13–16]; this was undoubtedly due to the use of a large surface area high-flux dialyser and a rather high plasma/dialysate potassium gradient. A glucose-free dialysate (as used in our non-diabetic patients) has also been shown to increase dialytic potassium removal [17]. However, it is unlikely that this contributed much in the present study, since in diabetic patients (whose dialysate contained glucose)

![](image)

Fig. 3. Changes in plasma potassium (mmol/l) during and after haemodialysis in the patient with the highest predialysis plasma potassium.

Table 2. Biochemical parameters during study period

<table>
<thead>
<tr>
<th></th>
<th>Predialysis</th>
<th>Postdialysis</th>
<th>6 h after dialysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pH</td>
<td>7.371 ± 0.010</td>
<td>7.494 ± 0.011*</td>
<td>7.459 ± 0.010b</td>
</tr>
<tr>
<td>Plasma bicarbonate (mmol/l)</td>
<td>21.6 ± 0.5</td>
<td>27.6 ± 0.6*</td>
<td>27.1 ± 0.7*</td>
</tr>
<tr>
<td>Plasma glucose (mmol/l)</td>
<td>7.2 ± 0.6</td>
<td>5.3 ± 0.4*</td>
<td>7.0 ± 0.7</td>
</tr>
<tr>
<td>Plasma insulin (mU/l)</td>
<td>24.3 ± 5.7</td>
<td>11.1 ± 2.4</td>
<td>21.2 ± 4.2</td>
</tr>
<tr>
<td>Plasma osmolality (mOsm/l)</td>
<td>307.2 ± 3.6</td>
<td>285.1 ± 3.3*</td>
<td>291.1 ± 3.7*</td>
</tr>
</tbody>
</table>

* $P < 0.001$ vs predialysis; * $P < 0.02$ vs predialysis; b $P < 0.05$, 6 h postdialysis vs postdialysis.

Table 3. Total body potassium (TBK) and body potassium per kilogram dry body weight (BW) and per litre total body water (TBW)

<table>
<thead>
<tr>
<th></th>
<th>Female</th>
<th>Male</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total (mmol)</td>
<td>2236 ± 157</td>
<td>3565 ± 147*</td>
</tr>
<tr>
<td>BW (mmol/kg)</td>
<td>38.2 ± 2.5</td>
<td>49.0 ± 3.5*</td>
</tr>
<tr>
<td>TBW (mmol/l)</td>
<td>73.2 ± 4.4</td>
<td>84.0 ± 3.6</td>
</tr>
<tr>
<td>Expected age-adjusted</td>
<td>38.6 ± 1.1</td>
<td>52.6 ± 0.8</td>
</tr>
<tr>
<td>TBK (mmol/kg BW)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Difference female vs male $P < 0.001$; *Difference female vs male $P < 0.05$. 
Plasma potassium during and after haemodialysis

Unfortunately, the amount of potassium of intracellular provenance cannot be measured without collection of dialysate. We therefore looked for parameters that might give some clinical impression about the amount of potassium eliminated under standard dialytic conditions. The product of plasma K and TBK was found to correlate well with dialytic potassium removal. This indicates, that potassium removal will be highest in patients with high plasma K and a large muscular mass (the main contributor to TBK).

In spite of the large amount of >100 mEq of potassium removed by dialysis an impressive rebound of plasma K concentration occurred. This was considerably more marked than described previously by Feig et al. [13]. However, our study differed in many respects from theirs: our patients, who followed a rather liberal diet had definitely higher predialysis plasma K levels, were dialysed with a larger surface area dialysier with higher blood flow rates, and for 4 instead of 3 h. The size of the 6-h postdialysis rebound ΔK was not correlated with pre- or postdialysis plasma K or TBK nor the product of the two. Since blood gas parameters, plasma glucose, insulin and osmolality were virtually unchanged during the 6 hours postdialysis these factors were probably not important in potassium shifts out of cells into the ECF (which comprised only 2.3% of TBK). It seems possible that a defect of the Na-K pump induced by an ouabain-like inhibitor was responsible for a change in cellular potassium uptake in our patients with renal failure.

Potassium removal lay in the average range; in fact two diabetics demonstrated the highest potassium removal. The amount of potassium removed by diffusion alone or by diffusion plus convection did not correlate with baseline plasma K. This is in contradistinction to findings by Allon and Shanklin [16] who described a good correlation between plasma K and cumulative dialysed potassium, which is somewhat surprising since potassium is distributed in at least two compartments [13], and since its dialytic removal is quite variable and dependent on several factors [14]. In our study predialysis plasma K values were clustered in a very narrow range, which may have obscured any relation with dialytic potassium removal. Moreover, our patients were studied in the non-fasting state; this led to a considerable variation of their predialysis plasma insulin levels, which may have masked the effect of predialysis plasma K. However, predialysis total extracellular potassium did correlate with total potassium removal. The best correlation of potassium removal on dialysis was found with the quantity of potassium originating in the intracellular space. These findings indicate that both extra- and intracellular potassium are of importance for the removal of potassium. Unfortunately, the amount of potassium of intracellular provenance cannot be measured without collection of dialysate. We therefore looked for parameters that might give some clinical impression about the amount of potassium eliminated under standard dialytic conditions. The product of plasma K and TBK was found to correlate well with dialytic potassium removal. This indicates, that potassium removal will be highest in patients with high plasma K and a large muscular mass (the main contributor to TBK).

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The TBK of control individuals is dependent on muscle mass, age and sex [10]. For this reason our TBK measurements are reported separately for women and men. We also compared our data with the age-adjusted values of Pierson et al. [10] who showed that TBK values peak by age 20 and then decrease by approximately 20–25% in the elderly because of lower muscle mass. In our patients TBK generally was well maintained compared with the expected normal age-
adjusted range. Previous measurements of TBK in dialysed patients are somewhat controversial: on the one hand Ramirez et al. [20] found normal age-adjusted potassium concentrations per kilogram body weight as we did; on the other hand there are studies describing decreased muscle stores [21,22] that would imply a decrease in TBK, since most of TBK is in muscle mass. We feel that patients with chronic renal failure on haemodialysis can maintain a normal TBK provided they are well dialysed and well nourished.

We are willing to accept relatively large interdialytic weight gains in order to obtain optimal nutritional parameters which is in agreement with a recent publication by Sherman et al. [23]. Accordingly, our patients had a good serum albumin level of 4.35 ± 0.1 g/dl (even in the diluted predialysis state).

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References


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