Low-potassium and glucose-free dialysis maintains urea but enhances potassium removal

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Abstract

Background. The influence of potassium (K) removal on dialysis efficiency as measured by urea elimination is not clear. In this prospective, randomized, crossover study we investigated the magnitude of K removal and its effect on urea (u) elimination during high-flux haemodialysis (HD).

Methods. Twelve stable, non-diabetic HD patients were investigated during three one-week standardized HD periods (1.8 m² high-flux polysulphone dialyser, treatment time 240 min, Qb=300 ml/min, Qd=500 ml/min, dialysate without glucose, bicarbonate 40 mmol/l), using dialysates containing 0 (0K), 1 (1K), and 2 (2K) mmol/l of K. Mass removal of K (Mk) and u (Mu) were measured during the mid-week treatment by partial dialysate collection. Urea reduction rate (URR) and Kt/V were determined.

Results. 0K, 1K and 2K treatments were perfectly comparable. Plasma K (PK) continuously declined reaching stable concentrations after 180 min. While 0K dialysate removed 117.1 mmol, 80.2 and 63.3 mmol (P<0.001) were removed by 1K and 2K baths respectively. Mk was not influenced by Mk (r=0.22) and amounted to 410.1 (0K), 508.6 (1K), and 506.2 (2K) mmol (NS) respectively. Accordingly, urea clearance, URR and Kt/V were constant during 0K, 1K and 2K treatments.

Conclusions. Potassium-free dialysate significantly enhances potassium elimination. Potassium removal has no influence on urea elimination. High potassium removal, when needed, does not impair dialysis efficiency as measured by urea kinetics in high-flux, glucose-free, 40 mmol/l bicarbonate HD.

Keywords: glucose-free dialysis; haemodialysis; low-K dialysis; potassium removal; urea removal

Introduction

Patients suffering from end-stage renal disease (ESRD) are at high risk of hyperkalaemia due to diminished renal potassium excretion as well as impaired extrarenal potassium disposal [1–3]. One of the main goals of dialysis is to remove excess potassium accumulated in the interdialytic period. However, acute lowering of plasma potassium levels by haemodialysis (HD) may have haemodynamic effects and lead to rebound hypertension [4].

On the other hand, we feel that ESRD patients can maintain a normal total body potassium provided they are well dialysed and well nourished, and we are willing to accept a more liberal food intake. In fact, we found a normal total body potassium in patients dialysed three times weekly for 4–5 h using high-flux dialysers with a surface area of 1.8–2.4 m² [5].

The rate of potassium removal from the plasma depends on the gradient between plasma and dialysate potassium concentrations. Potassium-free dialysate improved potassium elimination by 24 and 50%, compared to a dialysate potassium of 1 and 2 mmol/l respectively, during low-flux dialysis [6].

A recent study by Dolson and Adrogue [7] showed that in low-flux, glucose, 30 mmol/l bicarbonate HD, dialysis efficiency evaluated by means of the urea reduction rate and Kt/V was significantly reduced when decreasing dialysate potassium concentration from 3 to 1 mmol/l. The authors explained this effect as a consequence of decreased blood flow to urea-rich tissues, largely skeletal muscles, secondary to vasoconstriction induced by aggressive potassium removal.

The clinical implications of this result have to be seen with scepticism. The dialysis community might be persuaded to favour high potassium concentrations in the dialysate bath and risk hyperkalaemia in order to increase the dose of delivered dialysis measured by urea kinetics. We think that apart from performing an adequate dialysis as measured by urea kinetics, dialysis must focus on removal of acute and chronic uraemic
toxins such as potassium and phosphate, which differ from urea in their distribution and elimination characteristics. Therefore, we undertook a similar study in our dialysis population using treatment modalities typical of our dialysis unit (high-flux, glucose-free, 40 mmol/l bicarbonate HD) and investigated the effect of 0K, 1K, and 2K baths on urea and potassium elimination.

Subjects and methods

Patients

Twelve patients with end-stage renal disease receiving maintenance high-flux HD were studied prospectively. There were six women and six men aged 30–73 (56.2 ± 4.1 mean ± SEM) years. Six patients suffered from chronic glomerulonephritis, three from analgesic nephropathy, and one each from adult polycystic kidney disease, nephroclerosis, and reflux nephropathy, respectively. Dialysis patients and those with heart disease were not considered for this study. Six patients had radiocephalic fistulae, five had brachiocephalic fistulae, and one had a brachiosubclavian PTFE graft. Serum albumin of all patients was above 35 (mean 41.1 ± 2.6 g/l and the mean predialysis haematocrit was 38.0 ± 1.0% at the beginning of the study. Patients were studied under their normal every day condition; for this reason food was allowed as usual. All patients continued to receive their normal medication. One patient received a small dose of the cardioslective β-blocker atenolol (50 mg day) and two received amiodipine (5–10 mg day) for treatment of hypertension, but none was taking digoxin, angiotensin-converting enzyme inhibitors, angiotensin-receptor blockers, or potassium-binding exchange resins. Medications were not modified during the entire study period.

Study design

This was a randomized, cross-over, prospective clinical trial. Patients were investigated during three 1-week standardized high-flux HD periods, using dialysates without potassium (0K), and dialysates containing 1 (1K) and 2 (2K) mmol/l of potassium. They were randomly assigned to the different 1-week study treatments. During the entire study they were treated with high-flux HD using a polysulphone filter (HF80, Fresenius AG, Bad Homburg, Germany). HD was performed thrice weekly, at an effective blood flow rate of 300 ml/min corrected for pre-pump blood pressure and a dialysate flow of approximately 500 ml/min. Treatment time was 240 min. Ultra-pure, bicarbonate-buffered dialysate with Na 140.0, HCO3 40.0, Mg2+ 0.5, and Ca2+ 1.5 mmol/l was obtained by filtration through a 1.8 m² polysulphone filter (Diasafe, Fresenius AG, Bad Homburg, Germany). The dialysate did not contain glucose. The same two dialysis monitors were applied for all patients and filter reuse was not practised. The ultrafiltration rates were prescribed according to the clinical needs and were calculated from the patient's net weight changes.

Potassium and urea kinetic investigations were performed during the mid-week treatment. Potassium and urea removal was measured by partial dialysate and ultrafiltrate collection as described previously [8–10]. The collection system was calibrated before each study. The mean ratio (f = Vd/Vc) of total dialysate volume (Vd) to collected volume (Vc) was 24.9 ± 0.02 (SEM). Dialysate and ultrafiltrate were collected in 4-hourly samples (0–60, 61–120, 121–180 and 181–240 min). Plasma potassium (PK) and blood urea were also measured at the beginning, at hourly intervals, and at the end of the dialysis procedure. Blood samples were drawn from the arteriovenous fistula at the beginning and at the end of HD. Blood sampling at the end of HD was performed 5 min after the completion of treatment to avoid vascular access and cardiopulmonary recirculations following the recommendations of the NKF-DOQI Clinical Practice Guidelines for Haemodialysis Adequacy [11]. During the dialysis procedure, samples were taken from the arterial line.

Efficiency of HD for urea and potassium was determined by clearance (Kd) based on dialysate and ultrafiltrate collection, urea reduction ratio (URR) and Kt/V.

Biochemistry

Potassium was measured using an IMT-Modul on a Dimension RXL (DADE-Behring, Marburg, Germany) in heparin-plasma and dialysate. Coefficients of variation were less than 2%. Urea was measured in heparin-plasma and dialysate according to the urease–glutamate–dehydrogenase technique on the Dimension RXL. Coefficients of variations were less than 2.5%.

Analysis

Total dialysate plus ultrafiltrate volume (Vd, in l) was calculated from the volume (Vc) obtained from partial dialysate and ultrafiltrate collections and from the calibration factor (f) determined at the beginning of each study. Mass of potassium and urine removed (Mk, Mt) was calculated from the mean solute concentration in the collected dialysate and from Vd.

Dialysate urea clearance (Kd) was calculated from the amount of solute removed and the area under the curve (AUC) of the blood urea concentrations.

Single pool Kt/V (Kt/Vsp) was calculated using the second-generation formula of Daugirdas [12]. Kt/V measures the quantity of clearance normalized to distribution volume received by a patient. Kt/V has an exact physical meaning only with the single-pool constant volume urea kinetic model [13]. The relationship does not appear in more advanced models where volume (V) changes because of ultrafiltration and where urea transfer between peripheral and central body compartments is limited to tissue perfusion. An equilibrated Kt/V can still be calculated either when an equilibrated post-dialysis urea concentration is available or when the magnitude of post-dialysis urea rebound can be predicted. However, the prediction of post-dialysis urea rebound and equilibrated Kt/V becomes unreliable when changes in regional perfusion are expected. Therefore a different approach was used to analyse the effect of dialysate potassium on dose of dialysis.

The single-pool constant volume relationship between Kt/V can be rearranged in terms of mass balances according to the following relationship:

\[ Kt/V = -\ln \left( \frac{M_0 - M_d}{M_0} \right) \] (1)

where M0 and Md refer to the mass of urea at the beginning of dialysis and to the mass of urea removed during dialysis respectively. Therefore Kt/V also measures the amount of solute remaining in the patient at the end of dialysis (M0−Md) relative to the amount of solute (M0) present in the patient at the beginning of the treatment. Using this
definition, mass balance can be used to determine an equilibrated Kt/V (Kt/V) obtained under more realistic variable volume and two-pool conditions. It follows from Eq. 1 that the difference in Kt/V (ΔKt/V) delivered by two different treatment modes A and B is given as

\[ \Delta Kt/V = \ln \left( \frac{M_{o,B} - M_{g,B}}{M_{e,A} - M_{g,A}} \right) \]

The value ΔKt/V as defined here should not be confused with the expression of ΔKt/V\text{spec} which is used to predict the overestimation of dialysis dose by single-pool urea kinetic analysis [14]. ΔKt/V, as defined here can be used to compare the difference in dose of dialysis between treatments when the amount of solute removed during dialysis (M_e) is measured such as by partial dialysate collection. However, it has to be assumed that urea generation rate is not affected by different treatment modes. The initial amount of solute (M_{o,A}, M_{o,B}) was determined from total body water (TBW) [15] corrected for ultrafiltration volume (UFV) and initial plasma urea concentration (c_{b,U}). Systematic errors in the estimation of TBW have a negligible influence on the calculation of ΔKt/V if the effect of two different treatments is measured in the same patient. This approach is independent of two-compartment effects and post-dialysis urea rebound, and does not require an equilibrated post-dialysis urea concentration. This is a major advantage because the study can be performed without reducing effective treatment time or asking the patient to stay for an equilibrated sample.

Statistical analysis

Analysis of variance for repeated measures was used for assessing the statistical significance of differences in M_U and M_K between the treatments. This approach was justified since QQ-plots showed that the distributions of residuals of M_U and M_K were close to normal.

If a given variable showed significant heterogeneity across the three groups of dialysis in the preceding analysis of variance, the differences between any two of the three groups were assessed with two-sided paired t-tests. Applying Bonferroni correction, the resulting P-values were multiplied by 3.

Stepwise multiple linear regression analysis was performed using the blood and plasma levels of urea and potassium respectively, to predict the mass of urea and potassium removed. If the regression model showed a high precision (R^2 ≥ 0.90), analysis of Bland and Altman [16] was performed for determining the strength of association.

For all calculations, Stata software, version 6.0 for Windows 95/98 (Stata Corporation, 77840 Texas) was used. Data are expressed as means ± SEM.

Results

All patients completed the study. The three 1-week periods were perfectly comparable. Predialysis weight, dialysate flow, ultrafiltrate flow, and ultrafiltration volume were not different during 0K, 1K, and 2K treatments. Initial blood urea concentration, total body water and urea mass of the patients were not different during the study HDs as well (Table 1).

Potassium removal

Figure 1 illustrates the effect of each dialysate potassium concentration on plasma potassium concentration and on hourly potassium removal. Predialysis plasma potassium concentrations were only slightly lower with 0K and 1K dialysis treatments compared to 2K dialysis treatments; values were 4.4 ± 0.2, 4.5 ± 0.2 and 4.9 ± 0.2 mmol/l (P = 0.02) respectively. During haemodialysis PK declined continuously reaching a nadir at 180 min and remaining constant to the end of the treatments. PK at completion of dialysis was clearly lower with 0K as compared to 1K and 2K, achieving 2.7 ± 0.1, 3.0 ± 0.2 and 3.5 ± 0.1 mmol/l (P < 0.001) respectively. Mass removal of potassium (M_K) was highest during the first 60 min and declined, reaching a constant value during the last 120 min with the exception of the 2K dialysis; in this case M_K from 181 to 240 min was significantly lower than M_K from 121 to 180 min. During the last 60 min potassium removal continued in spite of a constant plasma potassium concentration.

Our analysis shows that the amount of potassium removed is significantly different between the three regimens used. After 240 min of dialysis, potassium-free dialysate is more effective than 1K and 2K dialysate in removing body potassium. Potassium removal was 117.1 ± 10.3 mmol for 0K, 80.2 ± 6.2 mmol for 1K, and 63.3 ± 5.2 mmol for 2K (P < 0.001). Potassium-free dialysate removed 85% more potassium than 2K and 46% more than 1K.

Interestingly, potassium removal could not be predicted by modelling plasma potassium concentrations. Multiple linear regression analyses demonstrated that potassium removal could not be estimated by plasma potassium concentrations (R^2 = 0.18) (Table 2).

Urea removal

Urea removal is summarized in Figure 2. Blood urea and hourly urea removal followed an exponential function, differing clearly from the potassium elimination pattern. In contrast to potassium kinetics, the multiple regression between predicted urea removal modelled from blood concentrations almost perfectly followed the line of identity when compared to measured urea removal (R^2 = 0.98) (Table 3, Figure 3).

Blood urea concentrations at the three study dialyses were identical. Mass removal of urea (M_U) remained constant with 0K, 1K and 2K. M_U amounted to 491.1 ± 45.8 mmol during 0K, to 508.6 ± 48.9 mmol during 1K and to 506.2 ± 50 mmol during 2K (NS). No significant correlation between cumulative potassium removal and total urea removal could be demonstrated (Figure 4). Correspondingly, urea clearance was not influenced by the potassium content in the dialysate. K_dU was 206.1 ± 3.0, 211.1 ± 3.5 and 211.5 ± 2.9 mL/min during 0K, 1K and 2K (NS) respectively.

Dialysis efficiency evaluated by URR and single pool Kt/V was not modified by using 0, 1 or 2 mmol/l of potassium in the dialysate. URR was 75 ± 2% with...
Potassium removal does not impair dialysis efficiency

Table 1. Treatment characteristics

<table>
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<tr>
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<th>0K</th>
<th>1K</th>
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<tr>
<td>Patients</td>
<td>12</td>
<td>12</td>
<td>12</td>
<td>NS</td>
</tr>
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<td>Predialysis weight (kg)</td>
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</tr>
<tr>
<td>Dialysate flow (Qd) (ml/min)</td>
<td>527 ± 8.0</td>
<td>526 ± 6.0</td>
<td>540 ± 9.0</td>
<td>NS</td>
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<td>Ultrafiltrate flow (Qf) (ml/min)</td>
<td>10 ± 0.8</td>
<td>10 ± 0.9</td>
<td>8.0 ± 0.6</td>
<td>NS</td>
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<td>Initial blood urea (cb0) (mmol/l)</td>
<td>20.10 ± 1.40</td>
<td>20.26 ± 1.56</td>
<td>20.11 ± 1.55</td>
<td>NS</td>
</tr>
<tr>
<td>Initial body water (TBW0) (l)</td>
<td>37.08 ± 2.20</td>
<td>37.02 ± 2.18</td>
<td>36.73 ± 2.14</td>
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<tr>
<td>Initial urea mass (M0) (mmol)</td>
<td>768 ± 87</td>
<td>766 ± 82</td>
<td>750 ± 83</td>
<td>NS</td>
</tr>
<tr>
<td>Ultrafiltrate volume (l)</td>
<td>2.3 ± 0.2</td>
<td>2.4 ± 0.2</td>
<td>2.0 ± 0.2</td>
<td>NS</td>
</tr>
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</table>

The three high-flux study HDs were comparable. 0K was dialysis without potassium in the dialysate, 1K was dialysis with 1 mmol/l, and 2K with 2 mmol/l potassium in the dialysate. Treatment time was 240 min and effective blood flow (Qb) was 300 ml/min. Values are means ± SEM.

Table 2. Potassium model

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<th>MS</th>
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<td>1413.67</td>
<td>(5, 30)</td>
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</tr>
<tr>
<td>Residual</td>
<td>33679.02</td>
<td>30</td>
<td>1122.63</td>
<td>P</td>
<td>0.3071</td>
</tr>
<tr>
<td>Total</td>
<td>40747.35</td>
<td>35</td>
<td>1164.21</td>
<td>R²</td>
<td>0.1735</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Potassium removed</th>
<th>Coef.</th>
<th>SE</th>
<th>t</th>
<th>P</th>
<th>95% CI</th>
</tr>
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<tbody>
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<td>cb0</td>
<td>23.34</td>
<td>15.04</td>
<td>1.55</td>
<td>0.13</td>
<td>−7.38−54.07</td>
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<tr>
<td>cb60</td>
<td>4.13</td>
<td>32.88</td>
<td>0.13</td>
<td>0.90</td>
<td>−63.02−71.29</td>
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<tr>
<td>cb120</td>
<td>33.47</td>
<td>41.16</td>
<td>0.81</td>
<td>0.42</td>
<td>−50.59−117.53</td>
</tr>
<tr>
<td>cb180</td>
<td>−39.45</td>
<td>45.20</td>
<td>−0.87</td>
<td>0.39</td>
<td>−131.76−52.86</td>
</tr>
<tr>
<td>cbf</td>
<td>−23.15</td>
<td>42.48</td>
<td>−0.55</td>
<td>0.59</td>
<td>−95.10−48.81</td>
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<tr>
<td>cn5</td>
<td>47.36</td>
<td>42.48</td>
<td>1.11</td>
<td>0.27</td>
<td>−39.39−134.11</td>
</tr>
</tbody>
</table>

Table generated from Stata using potassium removed as an outcome variable and the serum values. cb0, plasma potassium at beginning of HD; cb60, plasma potassium 60 min after initiation of HD; cb120, plasma potassium 120 min after initiation of HD; cb180, plasma potassium 180 min after initiation of HD; cbf, plasma potassium 5 min after end of HD, as predictor variables. The model described here using potassium plasma values does not predict potassium removal. Reduction of factors using likelihood ratio analysis does not show a contribution of single potassium values estimating potassium mass removal.

Fig. 1. Potassium removal. Plasma potassium (PK) concentrations (upper) and potassium mass removed (MK) (lower) during standardized high-flux HD with potassium-free (0K), potassium 1 mmol/l (1K), and potassium 2 mmol/l (2K) dialysates. PK concentrations and MK were measured at 60-min intervals. Values are means ± SEM.

0K, 76 ± 2% with 1K, and 75 ± 2% with 2K (NS). Single-pool Kt/V assessed by blood urea kinetic was 1.73 ± 0.12 for 0K, 1.74 ± 0.10 for 1K, and 1.67 ± 0.10 for 2K (NS). Initial body urea content was not different between treatments (Table 1). The difference in equilibrated Kt/V (ΔKt/Ve) as determined from eq. 2 was 0.08 ± 0.05 and 0.11 ± 0.05 units, and not different from zero when treatments using 1K and 2K dialysate baths were compared to potassium-free treatment modes respectively (one sample sign test, one sample t-test).

Discussion

In this manuscript we report results of a randomized, prospective study showing that dialysis using low-potassium dialysate baths did not affect the removal of urea in a group of stable, non-diabetic HD patients without heart disease treated with high-flux dialysers. This result is in striking contrast to an earlier report by Dolson et al. [7] who observed that a 1K dialysate bath significantly reduced dialysis efficiency as measured by urea kinetics when compared to a 3K dialysate bath. It was not our aim to challenge the results reported by Dolson and Adrogue but to show that in a usual setting at our unit, using low-potassium dialysate did not affect dialysis efficiency as measured by urea kinetic analysis.

Is there an explanation for these findings?
Fig. 2. Urea removal. Blood urea (upper) and urea mass removed ($M_U$) (lower) during 0K, 1K, and 2K dialysis. Note the overlap of the three blood urea curves. $M_U$ measured in 60-min intervals were comparable during dialyses with different potassium regimen. Values are means ± SEM.

Table 3. Urea model

<table>
<thead>
<tr>
<th>Source</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>Number of obs</th>
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<tr>
<td>Model</td>
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<td>2</td>
<td>452,981.11</td>
<td>F (2, 33)</td>
<td>784.97</td>
</tr>
<tr>
<td>Residual</td>
<td>191,043.23</td>
<td>33</td>
<td>577.07</td>
<td>P</td>
<td>0.0000</td>
</tr>
<tr>
<td>Total</td>
<td>925,005.46</td>
<td>35</td>
<td>26,428.72</td>
<td>$R^2$</td>
<td>0.9794</td>
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</tbody>
</table>

Urea removed

<table>
<thead>
<tr>
<th>Coef.</th>
<th>SE</th>
<th>$t$</th>
<th>$P$</th>
<th>95% CI</th>
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<tr>
<td>cb60</td>
<td>22.79</td>
<td>5.94</td>
<td>3.84</td>
<td>0.001</td>
</tr>
<tr>
<td>cb120</td>
<td>23.38</td>
<td>6.94</td>
<td>3.37</td>
<td>0.002</td>
</tr>
<tr>
<td>cons</td>
<td>26.27</td>
<td>16.62</td>
<td>1.58</td>
<td>0.123</td>
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</table>

Table generated from Stata using potassium removed as an outcome variable and the serum values. cb60, blood urea 60 min after initiation of HD; cb120, blood urea 120 min after initiation of HD. The model described here using values of blood urea does predict urea removal precisely. Urea removal is therefore highly predicted using blood urea values of the first and second hour after initiation of HD.

Dialysate composition was different with regard to glucose and bicarbonate concentrations. Whereas dialysate in the study of Dolson contained glucose at a concentration of 200 mg/100 ml, glucose-free dialysis was delivered in our study. Thus, a major factor contributing to the distribution of potassium between extracellular and intracellular compartments was different in these studies. With the delivery of glucose and the stimulation of insulin secretion, potassium is even more sequestered into the intracellular compartment [17]. In addition, dialysate bicarbonate was 40 mmol/l in our study compared to 30 mmol/l in the study reported by Dolson et al. High dialysate bicarbonate concentration may contribute to intracellular potassium sequestration [18].

The current explanation for the reduction in dialysis efficiency as measured by urea kinetics using low-potassium dialysate is based on the regional blood flow model [19]. The delayed removal of solute from
the body is mainly due to the low perfusion of the locomotor system, which contains almost 80% of total body urea. Since urea transport can be assumed as flow limited, any increase in muscle blood flow will increase total body clearance and dialysis efficiency. And any impairment in muscle blood flow will lead to enhanced sequestration and delayed removal of urea from this organ system. In the study of Dolson et al., the increase in dialysis efficiency as measured by urea kinetics using a 3K dialysate bath was attributed to the vasodilatatory effect of potassium and to an increased muscle blood flow. However, with the use of 11 mmol/l glucose in the dialysate, and with the stimulation of insulin secretion it is likely that blood flow was also elevated in this study. Muscle blood flow is higher with glucose administration and insulin secretion [20]. In our study one would expect a difference in urea removal between 0K and 2K baths with glucose-free dialysate as well. This was not the case. With absence of insulin, muscle blood flow will be low at the beginning of dialysis and the effect of hypokalaemic vasoconstriction should be assumed to be minimal. The exact mechanism to explain the discrepancy in urea removal between the results reported by Dolson and our study remains speculative. It is very probable that dialysate glucose plays a key role in this question, especially when glucose concentration in dialysate is high (11 mmol/l). Studies to clarify the importance of dialysate glucose remain to be done in future.

Ultrafiltration volumes were similar in both studies; however, ultrafiltration rates were 30% lower in this study because of the longer treatment times (4 vs 3 h). Thus intravascular volumes were probably higher, avoiding a critical reduction of the regional perfusion in this study.

One of the goals of chronic HD is the removal of potassium that has accumulated in the body in the interval between two dialyses. The present study confirms the behaviour of potassium removal described by others [21]. Plasma potassium concentration rapidly decreased during the first 60 min and stabilized during the last 60 min of dialysis. Plasma potassium reached a steady state during the last hour of dialysis, while potassium continued to emerge in the dialysate. Therefore it can be assumed that potassium removal rate was equal to the intra- to extracellular mass transfer rate. Although the rate of potassium removal depends on the difference between plasma and dialysate potassium concentrations, the amount of potassium removed could not be predicted by the change in intradialytic plasma potassium concentrations using a statistical model.

In another study by our group [5], the best correlation of potassium removal on dialysis was found with the quantity of potassium originating in the intracellular space. In contrast to potassium, urea blood concentrations during dialysis are predictive for the cumulative removal.

As expected, potassium removal was significantly greater with potassium-free dialysate than with 1 or 2 mmol/l dialysate potassium. On a clinical basis, potassium-free dialysate is very effective for stable, non-diabetic patients treated for a duration of 4 h. Potassium removal during 0K was 117 mmol, compared to 78.5 mmol as published by others [6]. The difference can be explained by the lack of glucose dialysate, which tends to increase potassium removal [22]. We are willing to accept a more liberal food intake without strong potassium restriction in order to obtain optimal nutritional parameters. Accordingly, our patients had a albumin level of 41.1 ±1.2 g/l in the haemodiluted predialysis state. Potassium-free dialysis appears as an alternative for stable dialysis patients without potassium dietary restriction and at risk of severe hyperkalaemia. Careful cardiological evaluation is recommended for those patients, since low potassium bath concentration may predispose patients on digoxin therapy, or with left ventricular hypertrophy, to ventricular arrhythmias [23].

This study was designed to evaluate the influence of dialysate potassium on urea removal and dose of dialysis. Both URR and single-pool Kt/V revealed no influence of dialysate potassium on dose of delivered dialysis as measured by urea kinetics. The difference in equilibrated Kt/V between treatments analysed according to Eq. 2 was not significantly different from zero (H0=0). However, there was a trend for increased dose of dialysis measured by urea removal when increasing dialysate potassium. The analysis according to Eq. 2 is based on the assumption that urea generation rate was independent of dialysate potassium concentration. This assumption is supported by the small contribution of urea generation rate to urea mass balance during dialysis. Only large variations in urea generation rate are expected to invalidate the calculation according to Eq. 2. Patients served as their own controls so that any possible difference in dialysis dose (AKt/V) should have produced a difference in solute removal. Even though the trends were in support of the result reported by Dolson et al., the changes were far from significant. Therefore, the hypothesis that low-potassium dialysate baths reduce dialysis efficiency as measured by urea kinetics is not supported by our data. As mentioned above, glucose and its indirect effect on regional blood flow distribution could play a key role in this question.

In conclusion, the present study demonstrates that potassium removal has no measurable influence on urea removal during high-flux, glucose-free, 40 mmol/l bicarbonate dialysis. In this setting, high potassium removal, when needed, does not significantly impair dialysis efficiency based on urea kinetics.

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