The faster potassium-lowering effect of high dialysate bicarbonate concentrations in chronic haemodialysis patients

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Abstract

Background. Hyperkalaemia is common in patients with advanced renal disease. In this double-blind, randomized, three-sequence, crossover study, we compared the effect of three dialysate bicarbonate concentrations ([HCO₃⁻/C₀]) on the kinetics of serum potassium (K⁺) reduction during a conventional haemodialysis (HD) session in chronic HD patients.

Methods. We studied eight stable HD patients. The choice of dialysate [HCO₃⁻/C₀] followed a previously assigned treatment protocol and the [HCO₃⁻] used were low bicarbonate (LB; 27 mmol/l), standard bicarbonate (SB; 35 mmol/l) and high bicarbonate (HB; 39 mmol/l). Polysulphone dialysers and automated machines provided blood flow rates of 300 ml/min and dialysis flow rates of 500 ml/min for each HD session. Blood samples were drawn at 0 (baseline), 15, 30, 60 and 240 min from the arterial extracorporeal line to assess blood gases and serum electrolytes. In three of the eight patients, we measured serum K⁺ 1 h post-dialysis as well as K⁺ removal by the dialysis. The same procedures were followed until the completion of the three arms of the study, with a 1 week interval between each experimental arm.

Results. Serum K⁺ decreased from 5.4±0.26 (baseline) to 4.96±0.20, 4.90±0.19, 4.68±0.13 and 4.24±0.15 mmol/l at 15, 30, 60 and 240 min, respectively, with LB; from 5.38±0.21 to 5.01±0.23, 4.70±0.25, 3.8±0.19 mmol/l, respectively, with SB; and from 5.45±0.25 to 4.79±0.17, 4.48±0.17, 3.86±0.16 and 3.34±0.11 mmol/l, respectively, with HB (P<0.05 for high vs standard and low [HCO₃⁻] at 60 and 240 min). The decrease in serum K⁺ correlated with the rise in serum [HCO₃⁻] in all but LB (P<0.05). Potassium rebound was 3.9±10.2%, 5.2±6.6% and 8.9±4.9% for LB, SB and HB dialysates, respectively (P=NS), while total K⁺ removal (mmol/dialysis) was 116.4±21.6 for LB, 73.2±12.8 for SB and 80.9±15.4 for HB (P=NS).

Conclusions. High dialysate [HCO₃⁻] was associated with a faster decrease in serum K⁺. Our results strongly suggest that this reduction was due to the enhanced shifting of K⁺ from the extracellular to the intracellular fluid compartment rather than its removal by dialysis. This finding could have an impact for those patients with life-threatening pre-HD hyperkalaemia.

Keywords: dialysate bicarbonate concentration; haemodialysis; hyperkalaemia

Introduction

Hyperkalaemia is one of the most frequent life-threatening electrolyte disorders in patients with advanced renal disease [1].

The conservative emergency treatment of this condition includes intravenous calcium gluconate, infusion of insulin along with glucose, nebulized β₂ adrenergic agonists, intravenous NaHCO₃ and oral or rectal cation-exchange resins [2].

Although NaHCO₃ is used widely for the treatment of emergency hyperkalaemia, its effect is uncertain and several authors have reported that varying bicarbonate infusion rates failed to lower serum potassium (K⁺) in hyperkalaemic patients with end-stage renal disease (ESRD) [3–5].

Conventional haemodialysis (HD) is highly efficient in removing the excess of body K⁺, decreasing the serum K⁺ concentration ([K⁺]) and, thus, restoring the resting membrane potential, which is vital for normal neuromuscular function and myocardial activity [4].
In severely hyperkalaemic patients, the faster the [K\(^+\)] is lowered, the faster the recovery from related serious conditions, such as cardiac arrhythmia.

Patients with ESRD also have metabolic acidosis, the degree of which depends upon several factors: interdialytic protein intake, protein catabolic rate, the length and frequency of HD sessions and the type and magnitude of the buffer in the dialysate.

The most widely utilized buffer in HD is bicarbonate, which corrects the acidosis that develops during the interdialytic period. The improvement of acidaemia might allow the transfer of K\(^+\) into the cellular compartment; thus, part of the effect of the bicarbonate buffer on serum K\(^+\) could be due to a transcellular shift [6].

To our knowledge, no data exist in the medical literature on the effects on serum K\(^+\) levels of different dialysate bicarbonate concentrations ([HCO\(_3^\)]\(^-\)) administered during dialysis.

The aim of this study was to compare the effects of three different dialysate [HCO\(_3^\)]\(^-\) (27, 35 and 39 mmol/l) on the kinetics of the reduction of [K\(^+\)] during conventional HD in ESRD patients.

Subjects and methods

Design

This is a double-blind (patient and laboratory technician), randomized, three-sequence, crossover study comparing the effect of three different dialysate [HCO\(_3^\)]\(^-\) [low (LB), 27 mmol/l; standard (SB), 35 mmol/l; and high (HB), 39 mmol/l] on the kinetics of the reduction of [K\(^+\)] during a standard HD session in ESRD patients on chronic (thrice weekly) HD. The participants went through one of the three treatment sequences (1: LB, SB, HB; 2: SB, HB, LB; and 3: HB, LB, SB), having been assigned according to a previously designed random-digit table. Three patients received sequence 1, three sequence 2 and two sequence 3.

Inclusion and exclusion criteria

The patients admitted into the study were adults with ESRD who were treated with regular (12 h/week) HD in the Unidad de Nefrología, Hospital Juan A. Fernández, Buenos Aires, Argentina, for at least the preceding 12 months. Subjects had to have a permanent native arteriovenous fistula or a polytetrafluoroethylene prosthetic vascular access allowing a Qb \(\geq 280\) ml/min and be without any relevant associated medical disorder for \(\geq 3\) months prior to the study. Patients with one or more of the following conditions were excluded: diabetes, haemolytic diseases, rhabdomyolysis, hepatic necrosis, neoplasms, dyskalaemic periodic paralysis, chronic obstructive lung disease, haemodynamic instability, hypercalcaemia, intolerance to bicarbonate dialysate, extra-skeletal calcifications, chronic diarrhoea, intestinal by-pass, malabsorptive disorders, enteric fistulas or villous adenoma.

Patients receiving drugs that could modify internal K\(^+\) balance or K\(^+\) excretion (i.e. \(\beta\)-blockers, diuretics, angiotensin-converting enzyme inhibitors, angiotensin-II receptor antagonists, non-steroidal anti-inflammatory drugs, \(\beta\)-agonists, trimethoprim, pentamidine, stool softeners, cathartics or insulin) and those requiring hypertonic dextrose solutions or mannitol infusion during HD were also excluded.

An Institutional Review Board and Ethics Committee (Comisión de Evaluación de Ensayos Clínicos, Departamento de Docencia e Investigación. Hospital Juan A. Fernandez) approved the study protocol, which conforms to the provisions of the World Medical Association’s declaration of Helsinki and the World Medical Assembly of Tokyo (1975). All the participants gave their signed consent prior to enrolment.

Patients and procedures

Upon arrival of a subject for a regular HD session, the vascular access was punctured with two standard HD fistula needles and 1 ml blood was collected anaerobically in heparinized plastic syringes from the arterial side of the extracorporeal line to measure baseline blood gases, [K\(^+\)], [Na\(^+\)], [Cl\(^-\)] and [Ca\(^{2+}\)].

Then, and immediately before starting HD, the dialysate [HCO\(_3^\)]\(^-\) (27, 35 or 39 mmol/l) was selected according to the previously randomly assigned treatment sequence, as described above.

Polysulphone hollow-fibre dialysers (Fresenius F-7, surface area 1.6 m\(^2\); Fresenius) and dialysis machines providing the dialysate automatically (Baxter 1550; Baxter Corp., IL) were used with a Qb of 300 ml/min and a Qd of 500 ml/min in each HD session.

The final HD fluid composition (mmol/l) was Na 140, K 2.0, Mg 0.375, Ca 1.25, acetate 2, Cl 108, 112 or 120 and HCO\(_3^\) 27, 35 or 39. The variation of [HCO\(_3^\)]\(^-\) was obtained by modifying the dialysate’s delivery system and the concentration of Cl\(^-\) varied reciprocally. Discretionary extra-corporeal heparinization was allowed during HD, administered through an automatic pump system.

Sequential blood samples were drawn from the arterial side of the extracorporeal line (as described previously) at 15, 30, 60 and 240 min. In three of the eight subjects, additional blood samples were drawn 1 h after HD to assess potassium rebound.

The same procedures were followed until the completion of the three arms of the study. A washout period of \(\geq 1\) week was allowed between experiments, with the participants receiving their usual dialysis sessions between them.

Blood sample processing

All intradialysis blood samples were drawn from the rubber socket of the arterial extracorporeal line before blood passed through the dialyser and they were submitted to the laboratory immediately for analysis.

In the laboratory, 100 \(\mu\)l whole blood was injected into an automatic analyser (OMNI; AVL Scientific Corp., Roswell, GA). Serum K\(^+\), Na\(^+\), Cl\(^-\), Ca\(^{2+}\), pH, PCO\(_2\) and PO\(_2\) were measured directly, while [HCO\(_3^\)]\(^-\) was calculated using the Henderson–Hasselbach equation, assuming a pK of 6.1 and a solubility coefficient for carbon dioxide of 0.0301. Serum K\(^+\), Na\(^+\), Cl\(^-\) and Ca\(^{2+}\) were measured by ion-selective electrode.
All the samples were processed in duplicate and the final value for each variable was taken as the average of the two measured values.

Potassium rebound (%) was calculated as follows:

\[
\text{Potassium rebound} = \frac{([\text{K}^+]_{\text{post HD}} - [\text{K}^+]_{\text{end of HD}})}{([\text{K}^+]_{\text{end of HD}})} \times 100
\]

Where \([\text{K}^+]_{\text{post-HD}}\) and \([\text{K}^+]_{\text{end of HD}}\) denote \([\text{K}^+]_1\)h after HD and at 240 min of HD, respectively.

Dialysate processing

In the three patients in whom \(\text{K}^+\) rebound was studied, we assessed the removal of \(\text{K}^+\) by dialysis. For this, the dialysate was collected in a cylindrical reservoir. Four sequential samples (0–15, 15–30, 30–60 and 60–240 min) were taken.

Each sample, and the total of the four samples were weighed, and a relationship of 1 g = 1 ml was assumed.

The concentration of potassium was measured in the inflow and collected dialysate and \(\text{K}^+\) removal was calculated as:

\[
\text{K}^+_R = [\text{K}^+]_{\text{ID}} \times J_{\text{ID}} - [\text{K}^+]_{\text{OD}} \times J_{\text{OD}}
\]

where \(\text{K}^+_R\) stands for potassium removal, \([\text{K}^+]_{\text{ID}}\) and \([\text{K}^+]_{\text{OD}}\) for potassium concentrations in the inflow and collected dialysate, respectively, and \(J_{\text{ID}}\) and \(J_{\text{OD}}\) are the volumes of the inflow and collected dialysates, respectively. Dialysate \([\text{K}^+]\) was measured with an ion-selective electrode.

Adverse experiences

Any adverse experience during this trial was documented.

Statistical analysis

All values are expressed as means ± SEM. The Shapiro–Wilk test was performed in order to determine if the data were normally distributed. One-way and repeated measures analysis of variance (ANOVA) in both factors (time and treatments) were used, with a posteriori Student–Newman–Keuls analyses when appropriate. Simple linear regressions and correlations were also used to examine the relationship between the change (\(\Delta\)) in \([\text{HCO}_3^-]\) and \(\Delta[\text{K}^+]\). All the tests were two-tailed and \(P\)-values of <0.05 were accepted as statistically significant.

Results

We studied eight anuric ESRD patients (46 ± 4.9 years old), with a dry weight of 70.3 ± 8.0 kg, on chronic HD for 66 ± 13.5 months. During the 3 months preceding their enrolment, their haematocrits were stable (29.5 ± 2.7%), their interdialytic weight gain varied from 1.8 to 5.5 kg (3.26 ± 0.43 kg), their calcium × phosphorus product ranged from 33.6 to 83.7 mg²/dl² (59.2 ± 6.5 mg²/dl²) and all of them were normotensive without medications.

Effect of HD on plasma \([\text{K}^+]\)

Baseline \([\text{K}^+]\) was similar under the three treatment conditions (5.40 ± 0.26, 5.38 ± 0.21 and 5.45 ± 0.25 mmol/l for the LB, SB and HB dialysates, respectively). The changes of \([\text{K}^+]\) along the 4 h HD with the three different dialysate \([\text{HCO}_3^-]\) are shown in Figure 1. Plasma \([\text{K}^+]\) decreased significantly (\(P < 0.05\)) at as early as 15 min for each of the three treatment schedules, but the \([\text{K}^+]\) values recorded at 60 and 240 min were statistically significantly lower with the 39 mmol/l bicarbonate dialysate than with the 27 and 35 mmol/l ones (\(P < 0.05\)).

The values of serum pH and \([\text{HCO}_3^-]\) during each treatment are shown in Table 1. Plasma \([\text{HCO}_3^-]\) increased faster with the high-bicarbonate dialysate (\(P < 0.05\)).

![Fig. 1. Serum potassium concentration measurements (means±SEM) in ESRD patients during conventional 4h HD sessions with three different bicarbonate concentration dialysates. High (filled circles), standard (open squares) and low (filled triangles) bicarbonate concentration dialysate. *\(P < 0.05\) for 39 mmol/l \([\text{HCO}_3^-]\) vs 27 and 35 mmol/l.](image-url)
The decrease in $[K^+]$ ($\Delta[K^+]$) correlated inversely with the increase in $[HCO_3^-]$ ($\Delta[HCO_3^-]$) with HB ($\Delta[K^+] = -0.73 - 0.11 \times \Delta[HCO_3^-]; r = -0.4, P < 0.05$) as well as with SB ($\Delta[K^+] = -0.37 - 0.19 \times \Delta[HCO_3^-]; r = -0.50, P < 0.05$), but not with LB ($\Delta[K^+] = -0.7 + 0.12 \times \Delta[HCO_3^-]; r = 0.17, P = NS$) (Figure 2).

Ionic calcium levels remained within the normal range throughout the study with each of the three treatment arms (1.05±0.05, 1.13±0.06 and 0.98±0.06 mmol/l for LB, SB and HB dialysis, respectively, at baseline and 1.2±0.03, 1.08±0.04 and 1.02±0.07 mmol/l for LB, SB and HB, respectively, at the end of dialysis).

Data on blood pressure are provided in Figure 3. Both systolic and diastolic pressures decreased during the dialysis sessions with the three treatments. This reduction was statistically significant but not clinically. Similarly, there were no significant changes in heart rate under the three experimental conditions.

No adverse events were recorded at all, severe alkalenaia was not evident after HD with HB and no cardiac arrhythmia was documented clinically.

Plasma $[K^+]$ after dialysis

In three out of the eight patients, plasma $K^+$ was also measured 1 h after HD. Plasma $K^+$ rose slightly from 3.5±0.25 to 3.8±0.23 mmol/l (HB), from 3.67±0.22 to 3.83±0.12 mmol/l (SB) and from 3.8±0.25 to 3.9±0.17 mmol/l (LB) 1 h after the end of HD; therefore, $K^+$ rebound (%) was 8.9±4.9 (HB), 5.2±6.6 (SB) and 3.9±10.2 (LB). This rebound was slight and statistically not significant when the three treatment schedules were compared. This absence of statistically significant differences may well be due to the small number of patients in whom potassium rebound was studied.

Potassium removal by HD

In the same three patients in whom $K^+$ rebound was assessed, we also measured $K^+$ removal by dialysis. Although no statistically significant differences were found, the total amount of $K^+$ removed by HD was higher during treatment with LB than with SB or HB.
(116.4 ± 21.6, 73.2 ± 12.8 and 80.9 ± 15.4 mmol/dialysis, respectively).

Cumulative $K^+$ removal is displayed in Figure 4. Potassium removal with the 27 mmol/l $[\text{HCO}_3^-]$ dialysate was associated with the lowest $K^+$ rebound (3.9 ± 10.2%; $P = \text{NS}$).

**Discussion**

Hyperkalaemia is one of the most frequent life-threatening electrolyte disturbances in patients suffering from advanced renal failure, and HD is the most effective approach to reduce $[K^+]$ in them [4].

This study demonstrates that HD with high concentrations of dialysate bicarbonate caused a faster decrease in serum $K^+$ than dialysates with lower bicarbonate content.

The effects of different compositions of dialysates on $[K^+]$ (besides those related to $K^+$ or glucose components) as well as the behaviour of $K^+$ removal with different dialysis modalities (i.e. peritoneal dialysis, standard HD, pre- or post-dilutional haemofiltration or variable-$K^+$ HD) have not been reported yet and, to our knowledge, this is the first trial that assessed the impact of using different dialysate $[\text{HCO}_3^-]$ on plasma $[K^+]$ during a standard HD session [7–10].

Agroyannis et al. [11] studied pH and $[K^+]$ changes in eight ESRD patients undergoing 32 HD sessions with a dialysate containing 39 mmol/l $\text{HCO}_3^-$ and concluded that $K^+$ was not significantly affected. In that study, only one $\text{HCO}_3^-$ concentration was used and blood samples were taken only pre- and post-HD.

The main aim of our work was to study the kinetics of $K^+$ and changes of acid–base balance using different dialysate $[\text{HCO}_3^-]$. 

![Fig. 3. Blood pressures (systolic and diastolic) during standard HD in ESRD patients with high (filled squares), standard (filled diamonds) or low (open triangles) bicarbonate dialysates. Repeated measures ANOVA: NS. Error bars denote SEM.](image1)

![Fig. 4. Cumulative potassium removal (mmol) during HD with 27 (grey bars), 35 (black bars) or 39 mmol/l (open bars) $[\text{HCO}_3^-]$ dialysates in three individual ESRD patients. The treatments were not significantly different statistically. Error bars denote SEM.](image2)
Plasma \([\mathbf{K}^+]\) decreased significantly 15 min after the start of HD with each of the three treatments, the decrease being more pronounced with the high [\(\text{HCO}_3^-\)] dialysate. After 60 min of HD, a statistically significantly lower \([\mathbf{K}^+]\) was observed during treatment with the 39 mmol/l dialysate [\(\text{HCO}_3^-\)] treatment. These lower values were maintained until the end of the session.

Transmembrane \(\mathbf{K}^+\) gradient is larger at the start of HD than it is during the process.

Although it is widely used for the treatment of emergency hyperkalaemia, the effect of Na\(\text{HCO}_3\) is uncertain and several authors have reported that varying the rate of bicarbonate infusion failed to lower \([\mathbf{K}^+]\) in hyperkalaemic ESRD patients [3–5].

Neither isotonic nor hypertonic 60 min pre-dialysis bicarbonate infusions to HD-dependent ESRD patients decreased \([\mathbf{K}^+]\), despite a steady increase in plasma [\(\text{HCO}_3^-\)] and pH [4]. Similarly, only during the 240–360 min of dialysis did a 6 h pre-dialysis bicarbonate infusion induce a significant decline in plasma \([\mathbf{K}^+]\) in nine out of 12 patients with terminal renal failure on chronic HD [5].

In another study, Gutierrez et al. [3] evaluated the potassium-lowering effect of hypertonic vs isotonic Na\(\text{HCO}_3\) infused pre-dialysis for 3 h in patients with ESRD on chronic maintenance HD. At 180 min, plasma \(\text{HCO}_3^-\) increased by an average of 3 mmol/l and \([\mathbf{K}^+]\) decreased by 0.35 mmol/l. They concluded that intravenous bicarbonate is not useful for the emergency treatment of hyperkalaemia in patients on maintenance HD.

HD corrects acid–base imbalance and is one of the most effective means of removing \(\mathbf{K}^+\) from the body [4,12–14]. Depending on the dialyser membrane and the rate of blood flow, 70–90 mmol of \(\mathbf{K}^+\) may be extracted per HD session [15]. The blood-to-dialysate \([\mathbf{K}^+]\) gradient determines the magnitude of \(\mathbf{K}^+\) removal: the higher the serum \([\mathbf{K}^+]\) and the lower the bath \([\mathbf{K}^+]\), the greater the rate of \(\mathbf{K}^+\) transfer [8,16]. Potassium-free dialysate significantly enhances \(\mathbf{K}^+\) elimination [10], while pre-treatment with \(\beta_2\) agonists or glucose-containing dialysate significantly decreases \(\mathbf{K}^+\) removal [17,18].

The combined effect of HD and a high buffer load, such as was induced in our study during HB dialysis, resulted in a rapid drop-off of \([\mathbf{K}^+]\). To clarify whether or not the rapid decline in \([\mathbf{K}^+]\) was due to an increased transfer of \(\mathbf{K}^+\) through the dialysis membrane or due to intracellular uptake, or a combination of these two mechanisms, we studied \(\mathbf{K}^+\) removal and \(\mathbf{K}^+\) rebound in three of the eight patients in our cohort. The magnitude of \(\mathbf{K}^+\) removal we observed lay in the range already reported by others and it is close to the finding of Blumberg et al. [19], who in 14 HD-dependent ESRD patients reported an average total \(\mathbf{K}^+\) removal of 107 ± 6.0 mmol during a standard 4 h HD session with 40 mmol/l bicarbonate in the dialysate. This larger \(\mathbf{K}^+\) removal (than ours during HB dialysis) is probably due to the lower \([\mathbf{K}^+]\) of the dialysate (1 mmol/l) used in their study.

The tendency for potassium removal by HD to be lower with standard and high [\(\text{HCO}_3^-\)] is already visible after 1 h (Figure 4) and it could be due to increased intracellular uptake. The rapid correction of low serum bicarbonate and blood pH would result in rapid alkalinization and in \(\mathbf{K}^+\) entering into the intracellular fluid compartment, decreasing the driving force of the gradient across the dialyser membrane and, thereby, reducing overall \(\mathbf{K}^+\) removal.

The intracellular sequestration of \(\mathbf{K}^+\) could explain why we did not observe a significant \(\mathbf{K}^+\) rebound soon after dialysis—in disagreement with data reported by other authors. Blumberg et al. [19] prospectively studied the removal of \(\mathbf{K}^+\) as well as \(\mathbf{K}^+\) rebound in 14 ESRD patients during a standard 4 h HD session. Potassium concentration declined continuously during the first 3 h and then remained stable until the end of the procedure. Despite the removal of an average of 107.1 ± 6.0 mmol \(\mathbf{K}^+\), \([\mathbf{K}^+]\) rose rapidly and steadily after HD (0.7 and 1.4 mmol/l during the first and sixth hours, respectively).

This rebound is more marked after manoeuvres to move \(\mathbf{K}^+\) through the cellular membrane (e.g. \(\beta_2\) adrenergic stimulation, insulin, glucose, etc.) and it reflects the retrieval of this ion from a previously augmented intracellular pool [20].

Kaloceritis et al. [21] studied post-dialysis \(\mathbf{K}^+\) rebound in 29 anuric ESRD HD patients and found a statistically significant increase in \([\mathbf{K}^+]\) 30 min after a standard HD.

In our study, blood samples to assess \(\mathbf{K}^+\) rebound were obtained in three of the eight studied ESRD patients 1 h after the end of HD and showed negligible increases of \([\mathbf{K}^+]\) after HD. It is possible that a \(\mathbf{K}^+\) rebound might have been observed in blood samples drawn later than 1 h after the end of HD.

The possibility that cell excitability may be influenced by changes in the resting membrane potential due to rapid variations in plasma \([\mathbf{K}^+]\) during HD is a matter of great concern. In a multicentre, prospective, randomized crossover trial, Redaelli et al. [22] studied the effect of \(\mathbf{K}^+\) removal in 42 chronic HD patients who had premature ventricular complexes during dialysis. With ECG Holter monitoring they demonstrated that decreasing dialysate \([\mathbf{K}^+]\) and a constant intra-HD \(\mathbf{K}^+\) gradient significantly reduced the arrhythmogenic effect of standard HD, in which dialysate \([\mathbf{K}^+]\) remains stable while the plasma–dialysate \(\mathbf{K}^+\) gradient is constantly reduced.

Similarly, Hou and colleagues [8] demonstrated that the use of a potassium-free dialysate is safe for most of the stable individuals with ESRD, although an increase in the frequency and severity of ventricular ectopy associated with profound hypokalaemia was noted in an occasional patient.

Although we did not perform Holter monitoring in our study, none of our patients developed clinically evident arrhythmia; analogously, we did not detect any remarkable changes in blood pressure or ionic calcium in our patients.
Even though the baseline $[K^{+}]$ in our cohort was not as high as levels seen in emergency hyperkalaemia, and even considering that our results may only be valid for patients with ESRD, our findings could have remarkable implications for those patients with dangerously high pre-HD $[K^{+}]$, who may be at high risk of developing life-threatening medical conditions. Hyperkalaemic patients with acute renal failure could benefit the most from the approach suggested by our findings. The mechanisms of $K^{+}$ tolerance are not developed in them and high serum $[K^{+}]$, otherwise generally inconsequential in individuals with ESRD, may have adverse effects in those with recently developed nephron drop-out. Under such circumstances, mainly in those patients with oliguria, acidemia and fluid overload, HD should not be delayed and HD with high dialysate $[\text{HCO}_3^{-}]$ seems to be the treatment of choice.

In conclusion, our data clearly show that HB provides a fast and statistically significant decrease in $[K^{+}]$ in HD-dependent ESRD patients. Our data also strongly suggest that this reduction is due to the enhanced shift of $K^{+}$ into the intracellular fluid compartment rather than to the increased removal of $K^{+}$ by dialysis. The safety of this HB dialysis, however, should be a matter of further research.

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