POTASSIUM REMOVAL AS A FACTOR LIMITING THE CORRECTION OF ACIDOSIS DURING DIALYSIS

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Summary

The diffusional fluxes of urea, potassium and bicarbonate across the dialysis membrane (external balance), and across the cellular membrane (internal balance) were determined in seven patients during haemodialysis using potassium free dialysate and dialysate containing 2.0mEq/L of potassium. The results show an inverse correlation between extraction of potassium and intake of bicarbonate in both external and internal balances. This is probably due to the increase in cell membrane electrical potential resulting from a fall in blood potassium and emphasises the importance of electrical driving forces in diffusional fluxes across cellular membranes.

Introduction

The distribution of potassium (K) between intracellular and extracellular fluid is mainly responsible for the permanent passive electrical potential difference which exists across the cellular membrane, given by the Nernst equation [1]. During dialysis (HD) the fall in extracellular potassium induces an increase in passive electrical potential difference.

The net flux of ions across cell membranes depends not only on the concentration gradient (Fick's first law of diffusion) but also on the electrical gradient (the Nernst-Planck equation) [2].

We have studied indirectly the influence of changes in the electrical gradient on the net flux of hydrogen ions (H\(^+\)) from the cell toward the extracellular space.

Patients and methods

Forty-two studies were conducted on seven clinically stable patients on regular haemodialysis, using a dialysate containing 2.0mEq/L of potassium (KD2) for
three sessions and in the three sessions of another week a potassium free dialysate (Kp0). Dialysate bicarbonate was kept constant at 35mEq/L and the remaining components were equivalent for the two dialysates. A 1.1m² cuprophane membrane dialyser was used. Inlet blood flows were measured by the air bubble time method and ranged from 250 to 300ml/min; countercurrent dialysate flows were reduced to about 300ml/min to emphasise the differences between the solute concentration in the dialysate at inlet and outlet in order to minimise measurement errors.

Samples of blood and dialysate at the dialyser inlet and outlet were drawn hourly and simultaneously during dialysis and glucose, urea, sodium, potassium, chloride, phosphorus, and creatinine were measured by autoanalyser. Blood gases and pH were measured with the IL 613 gas analyser and blood bicarbonate concentration was calculated.

Dialysate pCO₂ was measured with the IL 613 gas analyser; total CO₂ content in the dialysate was measured with the Corning 965 analyser; dialysate bicarbonate concentration was calculated by subtracting the dissolved CO₂ from the total CO₂ content. A CO₂ solubility factor of 0.0301 was used in these calculations.

The diffusional fluxes of urea, potassium and bicarbonate across the dialysis membrane (external balance) and across the cellular membrane (internal balance) were determined.

The external balance was obtained by collecting all effluent dialysate hourly. Only dialyses with a mass balance error for urea of less than five per cent were taken into account.

The internal balance was obtained using the kinetic equation of Sargent and Gotch [3] combined with the usual balance techniques and considering the extracellular volume as equal to 50 per cent of the total body water, measured as the urea distribution volume.

Statistical analysis was performed using Olivetti P6040 programs.

Results

The potassium, bicarbonate and urea concentration gradients between plasma water and dialysate are plotted against the time during dialysis with Kp0 and Kp2 in Figure 1. There were no significant differences in urea gradients. The potassium gradient showed a significant difference at time zero, but there was no significant difference between the slope of the lines. The bicarbonate gradient was not significantly different at time zero but declined at a faster rate during Kp0 dialysis than during Kp2 dialysis.

The external and internal balances for urea, potassium and bicarbonate during the two different dialyses are shown in Figure 2. The whole area represents the total quantity of substances which were removed from the body (urea and potassium) or added to the body (bicarbonate). The lower area indicates the cellular contribution and the remaining area represents the extracellular contribution.

There was no significant difference between Kp0 and Kp2 dialysis as far as the total urea removal is concerned. The amount of urea removed from the
cellular space by dialysis was quantified as 49 per cent of the total removed during both the dialysis procedures.

More potassium was removed from the body with KD0 than with KD2 dialysis (p<0.01). The amount of cellular potassium removed was 60 per cent of the total during KD2 dialysis and 65 per cent during KD0 dialysis (p<0.01).

The amount of bicarbonate leaving the dialysate and entering the body was significantly lower with KD0 than with KD2 dialysis (p<0.01). The amount of bicarbonate which disappeared from the extracellular space was about 76 per cent of bicarbonate received during dialysis from dialysate containing 2.0mEq/L of potassium and was only 44 per cent during dialysis with potassium free

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Figure 2. External and internal balances during \( K_D0 \) and \( K_D2 \) dialyses

Figure 3. Correlation between the loss of potassium and the gain in bicarbonate with \( K_D0 \) and \( K_D2 \) dialyses

\[ y = 482.3 - 2.835x \]

\[ r = -0.756 \quad P < 0.01 \]
dialysate (p<0.01).

Figure 3 shows the highly significant negative correlation between the loss of potassium and the intake of bicarbonate during dialysis (p<0.01). The dashed line links the two average weekly balances for the same patient during the two types of treatment.

Figure 4 represents the correlation between the potassium concentration gradient across the dialysis membrane and the amount of bicarbonate added to the body from the dialysate; the regression line indicates that for every reduction of 1mEq in the potassium gradient between plasma water and dialysate the intake of bicarbonate increased by 50mEq.

\[ n = 14 \]
\[ y = 445.6 - 49.9x \]
\[ r = -0.724 \quad P < 0.01 \]
\[ [K^+]_0 = 2.0 \text{ mEq/l} \]
\[ [K^+]_0 = 0 \quad " \]

Figure 4. Correlation between potassium concentration gradient across the dialysis membrane and the amount of bicarbonate added to the body during K\textsubscript{D0} and K\textsubscript{D2} dialyses.

Discussion

The unequal distribution of the diffusible ions between intracellular fluids and plasma water (Gibbs-Donnan effect) and between plasma water and dialysate during isolate ultrafiltration (sieving coefficient) is induced by a small electrical gradient (1.3mV) as is predictable from the Nernst equation. The predictable increase in potential of 20mV across the cell membrane, due to a 50 per cent reduction of potassium during dialysis, must therefore significantly influence ionic fluxes across cell membranes. This investigation has been performed deliberately using a potassium free dialysate under experimental conditions to emphasise the phenomenon. The reduced disappearance of bicarbonate from the extracellular space indicates a reduced net flux of hydrogen ions from the cell to the extracellular space. A decreased transport of bicarbonate from the
dialysate to the blood occurs and a smaller percentage of bicarbonate is utilised to correct intracellular acidosis. For this reason extracellular alkalosis can occur despite intracellular acidosis.

References

1    Hodgkin AL, Horowicz P. *J Physiol* 1959; 145: 405
2    Shultz SG. *Kidney Int* 1976; 9: 65

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