SODIUM–LITHIUM COUNTERTRANSPORT IN RED CELLS

To the Editor: Woods et al. (May 26 issue)[1] provide evidence for a decrease in the rate of sodium–lithium countertransport in red cells after hemodialysis, and suggest that this effect is related to the removal of a plasma factor. They did not observe a difference in ouabain-dependent sodium efflux during hemodialysis, but this conclusion is based on data in only two patients. This latter observation disagrees with previous data, which demonstrated a decrease in the sodium–potassium pump activity in patients with chronic renal failure.[2]

We have recently studied the sodium exchange in the red cells of 11 patients receiving hemodialysis (six men and five women, mean age, 42±14 years). Using a slightly modified version of the method described by Garay et al.,[3] we determined the ouabain-dependent sodium efflux and the furosemide-sensitive sodium–potassium cotransport in the red cells before and after hemodialysis performed in a classical way using a CF 1511 Travenol hollow-fiber dialyzer or hemofiltration using a Gambro hemofilter FH 303. In four patients treated by hemofiltration, the ultrafiltrate removed was exactly

Table 1. Effects of Hemodialysis on the Sodium–Potassium Pump.

	BEFORE HEMODIALYSIS	AFTER HEMODIALYSIS
Intracellular Na (μmol/liter of cells)	10±3	9.1±2.0
Intracellular K (μmol/liter of cells)	97.8±10	97.2±14
Cell volume (% initial volume)	85±11	84±7
k_{Na} (hr^{-1})	0.024±0.008	0.020±0.008
k_K (hr^{-1})	0.010±0.003	0.011±0.004
Ouabain-sensitive Na efflux (μmol/liter of cells × hr)	3505±891 *	4993±1378 *
Na cotransport	261±183	362±259
K cotransport	249±194	371±246

*P of difference <0.001 by paired t-test. Other differences were not significant.

compensated by the perfusion of saline solution. The intracellular sodium and potassium concentrations were also measured. The main findings are summarized in Table 1.

Like Woods et al., we did not observe any difference in cell volume, sodium and potassium intracellular concentrations, or sodium and potassium permeability coefficients. But the ouabain-dependent sodium efflux was significantly increased after hemodialysis, with a strong correlation to the amount of fluid removed during the hemodialysis sessions ($r = 0.87$, $P<0.01$). Similar data were obtained after hemofiltration; however, when the amount of fluid removed was carefully compensated for by saline infusion, the effect disappeared. Moreover, when red cells obtained after hemodialysis were incubated in plasma from the same patient, collected before the session, a significant inhibition of sodium ouabain-sensitive efflux was observed (4.993±1.378 to 3.728±741 mmol per liter of cells). These data suggest that modifications in the rate of ouabain-sensitive sodium efflux observed after hemodialysis may be related to a volume-dependent plasma factor and not only to a decrease in the concentration of a dialyzable factor; this factor may be similar to the "natriuretic digoxin-like factor" postulated by de Wardener and MacGregor[4] as playing a part in primary hypertension. It is also possible that the effect observed by Woods et al. on sodium–lithium countertransport is linked to improvement of the sodium pump activity.

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