In chronic renal failure (CRF), intracellular Na+ concentrations have been shown to be increased [1]. This increase has been related to partial inhibition of the sodium pump in red blood cells (RBC) [2, 3]. In patients with CRF, we have shown recently low RBC Na+-K+-ATPase activity, which was totally normalized after successful renal transplantation [1, 4, 5]. Thus, we could speculate on the presence of an endogenous <uremic> toxin which could be responsible for the partial inhibition of RBC Na+-K+-ATPase in CRF. Therefore, we studied the effect of hemodialysis on RBC Na+-K+-ATPase activity, as measured by digoxin-sensitive 86-rubidium uptake, in patients with CRF. 30 patients with various causes of CRF, excluding hypertension, aged 23–72 has been studied. Digoxin-sensitive RBC 86Rb uptake [4–6], blood urea nitrogen, serum creatinine and plasma potassium concentrations were measured prior to and at the termination of one of the twice weekly routine hemodialysis periods. As compared to age-matched healthy volunteers, RBC digoxin-sensitive 86Rb uptake, before dialysis, was decreased in the studied patients (23.77 ± 6.23% vs. 28.65 ± 5.16%; p < 0.05; Student’s unpaired test), which is in agreement with our previous findings [5]. After dialysis values increased significantly to 25.7 ± 6.5% (mean Δ% = 2.4 ± 0.84%; p < 0.01; Student’s paired t test, fig. 1). This increase was not related to changes in body weight (66.3 ± 14.0 kg to 61.2 ± 18.4 kg); blood urea nitrogen (30.7 ± 5.6 mmol/l to 9.5 ± 3.6 mmol/l), serum creatinine (1.031 ± 295 to 428 ± 169 mmol/l). The mechanism of this increase is yet to be demonstrated. Many authors however, postulated that an endogenous toxin with digitalis-like properties could be present in the plasma of patients with renal failure [7, 8].
After
Before
p < 0.01 n = 30 Hemodialysis
Fig. 1. Digoxin-sensitive 86Rb uptake before and after hemodialysis.
Our results do not provide any evidence of the presence of a such substance. But, if present and dialysable, partial clearance of this endogenous toxin could explain the rise of RB 86Rb uptake after hemodialysis in our patients.

References