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Use of serum hemoglobin (Hb) versus serum albumin (Alb) concentrations in estimating blood volume decrease during hemodialysis. *D.J.W. van Kraaij, M.M.J. Schuurmans, I.H. Go, R.W.M.M. Jansen, W.H.L. Hoefnagels, Department of Internal Medicine, Canisius-Wilhelmina Hospital; Department of Geriatric Medicine, University Hospital Nijmegen, Nijmegen, the Netherlands.* Appropriate assessment of circulating blood volume is critical in the management of patients on hemodialysis (HD). Increments in both serum albumin (Alb) and hemoglobin (Hb) concentrations during HD have been used previously to estimate circulating blood volume changes and predict hypotensive episodes. We compared pre- and postdialytic serum concentrations of Hb and Alb, and their mutual correlation in 15 HD patients on five separate occasions. Changes in concentrations are expressed as percentage of predialysis values.

	% (N = 15)
ΔAlb	9.7 ± 2.3
ΔHb	7.8 ± 1.9
ΔAlb − ΔHb	1.9 ± 0.8 (95% CI: 0.2–3.6)[a]
(ΔAlb − ΔHb)/ΔAlb	34.7 ± 9.3 (95% CI: −9.4–+112.1)

Data are mean ± SEM. [a] $P < 0.05$ (Wilcoxon signed rank test)

There was a significant mean difference of estimated blood volume decrease during HD using Alb versus Hb concentration measurements (ΔAlb − ΔHb). This difference was on average 35% of the Alb-estimated blood volume decrease with a wide 95% CI of −9% to +112% (ΔAlb − ΔHb)/ΔAlb). Various possible explanations for this difference include capillary leakage of Alb, intravascular pooling of Hb, and measurement inaccuracy. However, procentual changes in Alb and Hb concentrations correlated well ($r = 0.8857$, $P < 0.001$). The reliability of both methods remains questionable without comparison to an accurate standard measurement.

Fractures of long bones in dialysis and renal transplant patients: Incidence and complication rate. *M.J. Wiezer, M.H.J.L. Pannekoek, I.A.J.M. Broeders, R.J. Hené, and Chr. van der Werken, Departments of Surgery and Nefrology, University Hospital Utrecht and Stichting Thuisdialyse, Midden Nederland (STD), the Netherlands.* Renal osteodystrophy and corticosteroid induced osteoporosis may affect the quality of bone in patients on renal replacement treatment. This might cause an increase in the incidence of long bone fractures as well as delayed healing. Therefore, we investigated the cause, localization and the complication rate of fractures in all patients who were treated with hemodialysis or had a functional renal transplant between January 1986 and December 1995 in our hospital. All patient records were investigated, and the living patients were interviewed by telephone or by mail. In 19 of the 629 hemodialysis patients we found 21 fractures. The total incidence of fractures was 951/100,000 fractures/patient year. For the fractures of the collum femoris this incidence was 498/100,000, being higher than the incidence of the Dutch population (148/100,000). The mean age of patients with this type of fracture was 62.7 year, compared to 77.6 in the control population. Seven were treated with immobilization only, and in one of them a complication (infection) occurred. Fourteen fractures were operated, 2 patients died in the postoperative period (myocardial infarction, sepsis after infection of the prosthesis), in 4 cases healing was complicated (pseudoarthrosis, refracturated, infection, thrombosis). In 367 renal transplant patients we found 24 fractures. The total incidence was 978/100,000 fracture/patient year. Fourteen were treated by immobilization only, all healed without delay or complications. Ten fractures were operated, in one the consolidation was delayed. From our data we conclude that the incidence of fractures of the hip is increased in dialysis and renal transplant patients. The incidence of complications appears to be normal.

Effects of low dose nifedipine on urinary protein excretion (UPE) rate in patients with renal disease. *H.J. Kloke, J.F.M. Wetzels, R.A.P. Koene, and F.T.M. Huysmans, Department of Nephrology, University Hospital, Nijmegen, The Netherlands.* The reported effects of calcium channel blockers on UPE are quite equivocal. It has been suggested that the short-acting dihydropyridine calcium channel blocker, nifedipine, increases UPE by interference with tubular protein reabsorption. In a randomized controlled trial, ten patients with renal disease (UPE 5.0 ± 1.1 g/day, mean ± SEM) were treated with a dose of 10 mg nifedipine o.d. during one week. The acute effects on renal and systemic hemodynamics and on urinary albumin, IgG and β_2-microglobulin (β_2m) excretion were investigated during a clearance study in supine position after the first dose. After one week of treatment UPE rates were measured in 24-hour urine samples collected ambulatory in consecutive fractions of four to eight hours during normal daily activities. After the first dose nifedipine lowered mean arterial blood pressure in supine position by 7 ± 1 mm Hg ($P < 0.01$), attenuated proximal tubular sodium reabsorption [fractional excretion (FE) of sodium 3.48 ± 0.49 vs. 2.62 ± 0.35% during control, $P < 0.02$], but did not affect proximal tubular protein reabsorption (FE of β_2m 0.97 ± 0.30 vs. 0.98 ± 0.32% during control, NS). The decrease in blood pressure was not accompanied by decreases in urinary albumin or IgG excretion rates. The selectivity index as well as GFR, RPF and filtration fraction did not change. Continued treatment for one week with nifedipine did not influence 24 hour UPE. However, we observed an effect when comparing the urine collected during daily activities in the first four hours after drug intake with the urine collected at the start of the study when supine. During control measurements there was a slight increase in UPE. During nifedipine the increase in albuminuria was more marked and correlated with the selectivity index ($r = -0.82$, $P < 0.01$). In conclusion: (1.) nifedipine 10 mg orally did not impair tubular protein reabsorption; (2.) nifedipine had no immediate antiproteinuric effect despite the observed blood pressure reduction; and (3.) nifedipine increased UPE in ambulatory urine collections. This latter observation might explain the seemingly different effects of dihydropyridine calcium channel blockers as reported in previous studies.