Dear Sir,

Risk factors for atherosclerosis occur more frequently in patients with chronic renal failure (CRF) than in the general population and hypertension is the main determinant in the aetiology of it [1]. Atherosclerotic cardiovascular disease is a significant cause of morbidity and mortality in patients with end-stage renal failure (ESRF) [2].

A relationship has been shown between the reduction in nocturnal blood pressure (BP) fall and target organ damage. Reduced nocturnal BP fall is frequently found in CRF [3]. The aim of this study was to relate the BP profile of the interdialytic period to atherosclerotic vascular damage.

Eighteen haemodialysis patients (7 M, 11 F; age 61 ± 8.8 years) were studied. Causes of CRF were ischaemic renal disease (n = 5), interstitial nephritis (n = 4), glomerulonephritis (n = 3), polycystic kidney disease (n = 3), eclampsia (n = 1), diabetes (n = 1) and it was unknown in 1 patient. Bicarbonate haemodialysis was performed 3 times/week, 3.5-4 h each session. All the patients had an ambulatory BP monitor (Spacelabs 90207) placed immediately upon completion of the dialysis treatment and had it removed before the initiation of their next haemodialysis session, approximately 45 h later. BP monitoring was recorded every 15 min during the day (07:00-23:00 h) and half hourly during the night (23:00-07:00 h). Mean 48-hour-diurnal and nocturnal BP for both systolic and diastolic BP were calculated. Dipping status was defined as a reduction in nighttime MAP > 10% of the daytime MAP. Vascular damage was evaluated by B-mode ultrasound technique of carotid and leg arteries in order to detect normal vessels (group 1), calcinosis of the arterial wall (group 2) and plaques producing a lumen stenosis ≥ 40% (group 3).

ANOVA for parametric data and Kruskal-Wallis test for nonparametric data were used for statistical analyses.

Seven patients were classified as group 1, 5 as group 2 and 6 as group 3. Age (58 ± 4 vs. 60 ± 1 vs. 65 ± 4 years; NS), history of hypertension (10 ± 2 vs. 11 ± 3 vs. 18 ± 3 years; NS), length of time on dialysis (51 ± 8 vs. 44 ± 17 vs. 49 ± 14 months; NS), choleste-
terol (207 ± 11 vs. 203 ± 30 vs. 208 ± 27 mg/dl; NS), triglycerides (175 ± 44 vs. 186 ± 50 vs. 202 ± 41 mg/dl; NS), haemoglobin (9.1 ± 0.8 vs. 10.7 ± 0.7 vs. 9.2 ± 0.4 g/dl; NS) and weight variation between dialysis (1.8 ± 0.3 vs. 2.2 ± 0.4 vs. 2.0 ± 0.3 kg; NS) were comparable in groups 1, 2 and 3 respectively. Mean 48-hour daytime and nighttime systolic and diastolic BP values of the three groups were not different, however mean day-night difference of systolic and diastolic BP was significantly lower in the group 3 than groups 1 and 2 (table 1). Fourteen out of 18 patients were classified as nondippers and none of the dippers belonged to group 3.

The identification of the true BP in haemodialysis patients is difficult mainly because they have marked variations in extracellular fluid volume [4]. Whether circadian variation exists in dialysis patients is a matter of debate and its role in the pathogenesis of atherosclerosis has not been established yet. It is still unclear whether the changes in 24-hour BP pattern are due to the disturbances in renal function or to classical associated complication of CRF such as autonomic neuropathy, fluid retention and atherosclerosis [5].

Our results indicate that atherosclerotic vascular damage affects the BP profile causing a rise in nocturnal values. The relationship between atherosclerotic vascular damage and rise in nocturnal BP may in part explain the controversial findings of the different authors about circadian BP pattern in ESRF.

We conclude that atherosclerosis should be taken into account in studies dealing with daytime and nighttime BP difference in haemodialysis patients.

References

Nephron 1996;74:491-492
Fabbian/Squerzanti/Malacarne/Cecchetti/Cogliati/Gilli