

Original Article

Uncontrolled hypertension due to volume overload contributes to higher left ventricular mass index in CAPD patients

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Abstract

Background. Hypertension (HT) is common in patients on continuous ambulatory peritoneal dialysis (CAPD) and is responsible for increased cardiovascular morbidity and mortality. In this study, we aimed to determine the prevalence of ‘uncontrolled HT’ during background therapy in CAPD patients by using office measurements and ambulatory blood pressure monitoring (ABPM). We further determined whether intravascular volume status, assessed by inferior vena cava diameter (IVCD) index, contributes to higher blood pressure (BP) and increased left ventricular mass index (LVMI).

Methods. Seventy-four CAPD patients were included in the final analysis. All patients underwent echocardiographic examination and received ABPM. Patients undergoing CAPD were categorized into two groups: ‘uncontrolled HT’ (Group A) and ‘normotensive and controlled HT’ (Group B). Intravascular volume status was determined using the IVCD index and collapsibility index (CI) on the same day as ABPM.

Results. The prevalence of HT was 84% when using office measurements and 82% when using daytime ABPM. Daytime BP was 147/92 mm Hg by office measurements and 145/91 mm Hg by ABPM ($P > 0.05$). The prevalence of ‘uncontrolled HT’ measured by ABPM was 73% ($n = 54$). Patients with uncontrolled HT (Group A) were taking more anti-hypertensive medications than patients with ‘normotension and controlled HT’ (Group B, $n = 20$; 1.0 ± 0.8 vs 0.5 ± 0.7 , $P = 0.008$). The IVCD index was higher in Group A than in Group B (9.2 ± 2.1 vs 7.7 ± 1.9 mm/m², $P = 0.007$). There was no correlation between IVCD index and office BP, ABPM measurements or LVMI. The LVMI was also higher in Group A than in Group B (145 ± 39 vs 118 ± 34 g/m²,

$P < 0.01$). Stepwise multiple regression analysis revealed that 24 h diastolic BP and haemoglobin were independent determinants of LVMI.

Conclusion. Uncontrolled HT on background therapy is highly prevalent among volume overloaded CAPD patients. Further long-term prospective studies examining effects of salt restriction and ultrafiltration on BP control and left ventricle wall thickness are warranted.

Keywords: CAPD; hypertension; inferior vena cava diameter (index); left ventricular hypertrophy; plasma volume overload

Introduction

Cardiovascular diseases are the most common causes of morbidity and mortality in patients undergoing continuous ambulatory peritoneal dialysis (CAPD) [1]. In these patients, the prevalence of hypertension (HT) is high and ranges from 29% to 88% [2,3]. Volume overload has been associated with increased prevalence of uncontrolled HT in end-stage renal disease (ESRD) patients [4,5]. The measurement of inferior vena cava diameter (IVCD) is now accepted as a reliable non-invasive technique to determine volume status in dialysis patients [6].

Earlier studies suggested that patients on long-term CAPD treatment were more volume overloaded than patients on short-term CAPD [7]. Additionally, patients on long-term CAPD had higher left ventricular mass [8]. A recent study suggested that CAPD patients were more volume overloaded and had higher prevalence of left ventricular hypertrophy (LVH) compared with haemodialysis (HD) patients [9].

Ambulatory blood pressure monitoring (ABPM) has several advantages over office blood pressure (BP) measurements. A continuous 24 h measurement of BP can be performed by ABPM, providing a superior assessment of BP control. In addition, circadian BP variability was better associated with HT-induced end-organ damage in dialysis patients [10,11].

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The present study aimed to determine the prevalence of 'uncontrolled HT' during background therapy in CAPD patients by using office measurements and ABPM. We additionally determined whether intravascular volume status, which was assessed by IVCD index, contributes to higher BP and increased left ventricular mass index (LVMI).

Subjects and methods

Study population

Ninety-seven CAPD patients that were undergoing CAPD treatment for more than three months at Marmara University Hospital and that did not have peritonitis during the last three months constituted the final study group. Excluded from the study were 13 patients with previous myocardial infarction ($n=4$), coronary artery bypass surgery ($n=1$), history of congestive heart failure within the previous 12 months ($n=5$) and atrial fibrillation or any arrhythmia ($n=3$), because these may interrupt ABPM measurements. Patients included in the study were examined echocardiographically and had ABPM. An additional 10 patients were excluded due to technical difficulties during echocardiography ($n=5$), improper ABPM measurements ($n=3$) and severe valvular heart disease on echocardiography ($n=2$). The causes of chronic renal failure in the final cohort of 74 patients were diabetes mellitus (DM; $n=8$), chronic glomerulonephritis ($n=24$), tubulointerstitial nephritis ($n=7$), hypertensive nephrosclerosis ($n=12$), miscellaneous ($n=4$) and unknown ($n=19$). Clinical characteristics of the excluded patients were similar to the study group with respect to age (46 ± 15 vs 44 ± 15 years), gender (48% vs 51% female), duration of CAPD treatment (26 ± 17 vs 26 ± 16 months), body surface area (1.7 ± 0.2 vs 1.7 ± 0.2 m²) and aetiology of renal disease (9% vs 11% with DM, 34% vs 35% with glomerulonephritis, 22% vs 16% with hypertensive nephropathy, 9% vs 8% with interstitial nephritis, 4% vs 5% with miscellaneous aetiology and 22% vs 26% unknown).

Blood pressure measurements

Patients were allowed to continue their antihypertensive treatment during ABPM and during office BP measurements. After a 5 min rest period, office BP measurements were performed three times and the average of the last two measurements was accepted as the final office BP [12]. During the same day, patients were monitored with Spacelab® 90207 ambulatory monitor (Spacelabs Medical, Redmond, USA) every 20 min from 07.00 to 23.00 and every 30 min from 23.00 to 07.00. Monitors were calibrated against a mercury sphygmomanometer at the beginning of each measurement and monitoring was performed in accordance with recent guidelines [13]. Ambulatory blood pressure monitoring data included 24 h systolic blood pressure (SBP), 24 h diastolic blood pressure (DBP), 24 h mean arterial blood pressure (MAP), daytime SBP, daytime DBP, daytime MAP, night-time SBP, night-time DBP and night-time MAP. Hypertension was defined as daytime SBP ≥ 135 mm Hg and/or daytime DBP ≥ 85 mm Hg using ABPM, SBP ≥ 140 mm Hg and/or DBP ≥ 90 mm Hg using office BP measurements, or current treatment with an antihypertensive

drug [12]. Patients undergoing CAPD were categorized as 'uncontrolled HT' with daytime ABPM $\geq 135/85$ mm Hg and as 'normotensive or controlled HT' with daytime ABPM below 135/85 mm Hg according to current definitions and criteria by Burt and colleagues [12,14]. Uncontrolled HT patients constituted Group A, and normotensive and controlled HT patients constituted Group B.

Echocardiographic assessment

Two-dimensional and M-mode echocardiographic examinations were performed using an Ultramark 9 (Advanced Technology Laboratories, Bothell, WA) with a 2.25 MHz transducer. Echocardiographic examinations were performed at midday after having completely emptied the peritoneal dialysate. All examinations were recorded on videotape and assessed at study completion by two independent physicians (AT, HT) who were blinded with respect to patient group. At least three consecutive cardiac cycles were analysed for each patient. All echocardiographic parameters, including interventricular septal thickness (IVST), left ventricular posterior wall thickness (PWT), and left ventricular internal dimension (LVID), were measured at end-diastole (d). Ventricular dimensions were assessed through 2-D guided M-mode tracings according to American Society of Echocardiography (ASE) recommendations using leading edge to leading edge conventions [15]. Left ventricular (LV) mass was calculated using the formula [16]: ASE-cube LV mass = $1.04 \times ((IVSTd + LVIDd + PWTd)^3 - LVIDd^3)$. Because ASE-cube LV mass calculation results in overestimation of LV mass by about 25%, the regression formula of Devereux *et al.* [17] was used to correct LV mass measurements, which is similar to the Penn convention measurement [18] ($0.80 \times (\text{ASE-cube LV mass}) + 0.6$). The LVMI was calculated by dividing LV mass by body surface area (BSA, m²). Left ventricular hypertrophy was defined as LVMI > 131 g/m² in males and > 100 g/m² in females [19].

Determination of intravascular volume status

Intravascular volume status was determined using the IVCD index and collapsibility index (CI) on the same day as ABPM. The IVCD was measured by two-dimensional guided M-mode echocardiography during expiration and maximal inspiration while avoiding Valsalva-like manoeuvres [6]. Index of IVCD was calculated as the ratio of IVCD at expiration to BSA. Collapsibility index was defined as ((maximal diameter on expiration - minimal diameter on deep inspiration)/maximal diameter on expiration) $\times 100$.

Laboratory measurements

Whole blood counts and blood chemistry were analysed by standard laboratory procedures. Intact parathyroid hormone (PTH) levels were determined using radioimmunoassay (Sigma-Aldrich Laboratories, The Woodlands, Texas, USA). Kt/V was calculated from total loss of urea nitrogen in the spent dialysate using the Watson equation [20]. Serum albumin concentration was measured by the bromocresol green method. The peritoneal equilibration test (PET) and Kt/V measurement were done within two weeks of ABPM measurement.

Table 1. Office and ABPM data of 74 CAPD patients

Blood pressure	SBP (mm Hg)	DBP (mm Hg)	MAP (mm Hg)
Office	147 ± 27	92 ± 18	110 ± 22
24 h ABPM	143 ± 26	90 ± 18	109 ± 20
Daytime ABPM	145 ± 26	91 ± 18	111 ± 21
Night-time ABPM	139 ± 28	86 ± 19	105 ± 22
<i>P</i>	0.427	0.275	0.333

Values are expressed as mean ± SD.

Statistical analysis

All calculations were done using SPSS. Data were expressed as mean ± SD. Office BP measurements and ABPM data were compared by using one-way ANOVA, and comparison between Groups A and B were done by Mann-Whitney U tests. Categorical variables were analysed by the Fisher's exact test and McNemar test. Stepwise multiple regression analysis was performed to define the predictors of LVMI. A two-tailed *P* value less than 0.05 was considered significant.

Results

Seventy-four CAPD patients (38 female, 36 male; mean age: 44 ± 15 years; mean duration of CAPD treatment: 26 ± 16 months) were included in the study. Sixty-one patients (82%) were on CAPD treatment for more than 12 months.

The 24 h SBP and DBP with ABPM were 143 ± 26 and 90 ± 18 mm Hg, daytime were 145 ± 26 and 91 ± 18 mm Hg, and night-time were 139 ± 28 and 86 ± 19 mm Hg, respectively (Table 1). Office BP values were similar to daytime ABPM values (147 ± 27 vs 145 ± 26 mm Hg SBP, *P* = 0.991; 92 ± 18 vs 91 ± 18 mm Hg DBP, *P* > 0.05). Sixty-two patients (84%) were hypertensive according to office BP measurements and 61 patients (82%) were hypertensive according to daytime ABPM (*P* > 0.05). The prevalence of 'uncontrolled HT' was 73% (*n* = 54) according to daytime ABPM measurements.

Patients were categorized into Groups A (uncontrolled HT) and B (normotensive or controlled HT) according to daytime ABPM. The clinical characteristics, biochemistry and BP data of the groups are presented in Tables 2 and 3.

The causes of ESRD were similar in both groups (Table 3). Age, proportion of patients older than 65 years, BSA, proportion of patients with DM, duration of CAPD treatment, Kt/V, normalized creatinine clearance, residual Kt/V, proportion of anuric patients, daily ultrafiltration (UF) rate, daily urine volume, proportion of patients on erythropoietin (Epo) treatment, weekly dose of Epo, serum albumin and PTH were not different between Groups A and B. Although statistically insignificant, the percentage of patients on previous HD therapy for more than 6 months was higher in Group B than in Group A (40% vs 19%,

Table 2. Comparison of clinical and laboratory characteristics between uncontrolled HT (Group A) and normotensive or controlled HT (Group B) patients

	Group A (<i>n</i> = 54)	Group B (<i>n</i> = 20)	<i>P</i>
Age (years)	44 ± 15	45 ± 16	0.84
Age > 65 (%)	11	15	0.69
Male (%)	55	35	0.18
BSA (m ²)	1.71 ± 0.17	1.73 ± 0.23	0.70
Diabetes (%)	15	5	0.42
Duration of CAPD (months) (Median, range)	24 ± 16 (21, 5–68)	29 ± 16 (29, 4–58)	0.26
Previous HD therapy (> 6 months) (%)	19	40	0.084
Kt/V (units)	2.1 ± 0.5	2.3 ± 0.6	0.25
Creatinine clearance (l/week/1.73 m ²)	63.7 ± 21.7	69.5 ± 26.8	0.36
Residual Kt/V (units)	0.4 ± 0.5	0.4 ± 0.5	0.98
Anuric patients (%)	39	40	1.0
Net UF (ml)	1389 ± 511	1412 ± 554	0.87
Daily urine volume (ml)	465 ± 534	455 ± 549	0.94
Haemoglobin (g/dl)	10.2 ± 1.8	11.3 ± 1.6	0.014
Patients on Epo therapy (%)	87	65	0.16
Dose of Epo (U/kg/week)	76.1 ± 58.5	64.7 ± 65.5	0.49
Serum albumin (g/dl)	3.8 ± 0.5	4.0 ± 0.4	0.14
PTH (pg/ml)	300 ± 330	395 ± 430	0.37

Values are expressed as mean ± SD unless indicated differently.

Table 3. Blood pressure measurements and IVCD index of uncontrolled HT (Group A) and normotensive or controlled HT (Group B) patients

	Group A (<i>n</i> = 54)	Group B (<i>n</i> = 20)	<i>P</i>
Office SBP (mm Hg)	154 ± 20	121 ± 27	< 0.0001
Office DBP (mm Hg)	96 ± 15	75 ± 15	< 0.0001
Daytime SBP (mm Hg)	157 ± 20	114 ± 14	< 0.0001
Daytime DBP (mm Hg)	99 ± 13	71 ± 10	< 0.0001
Night-time SBP (mm Hg)	151 ± 21	106 ± 17	< 0.0001
Night-time DBP (mm Hg)	94 ± 14	64 ± 9	< 0.0001
24 h SBP (mm Hg)	155 ± 19	113 ± 15	< 0.0001
24 h DBP (mm Hg)	97 ± 13	69 ± 10	< 0.0001
Antihypertensive drugs (<i>n</i>)	1.0 ± 0.8	0.5 ± 0.7	0.008
IVCD index (mm/m ²)	9.2 ± 2.1	7.7 ± 1.9	0.007
CI (%)	52.5 ± 10.6	55.5 ± 10.8	0.64
Cause of ESRD			
Diabetes mellitus (%)	7 (13)	1 (5)	0.43
Hypertension (%)	10 (18)	2 (10)	0.49
Glomerulonephritis (%)	20 (37)	4 (20)	0.41
Interstitial nephritis (%)	3 (6)	4 (20)	0.33
Miscellaneous (%)	2 (4)	2 (10)	0.29
Unknown (%)	12 (22)	7 (35)	0.36

Values are expressed as mean ± SD.

P = 0.084). Group A had lower haemoglobin levels than Group B (*P* = 0.014, Table 2).

Office BP measurements and ABPM levels were significantly higher in Group A compared with Group B (Table 3). Group A used more antihypertensive drugs than Group B (1.0 ± 0.8 vs 0.5 ± 0.7, *P* = 0.008). Index of IVCD at experium was higher in Group A than in Group B (9.2 ± 2.1 vs 7.7 ± 1.9 mm/m², *P* = 0.007). The CI was similar in both groups

Table 4. Echocardiographic parameters of uncontrolled HT (Group A) and normotensive or controlled HT patients (Group B)

	Group A (n = 54)	Group B (n = 20)	P	1-β*
LVID index (mm/m ²)	30.1 ± 3.3	28.6 ± 3.3	0.09	0.40
IVST index (mm/m ²)	7.5 ± 1.3	6.8 ± 1.7	0.07	0.50
PWT index (mm/m ²)	6.7 ± 1.2	6.2 ± 1.2	0.11	0.40
LVMI (g/m ²)	145 ± 39	118 ± 34	<0.01	0.80
LVH (%)	70	60	0.41	0.20

Values are expressed as mean ± SD.

*Power analysis (α: 0.05).

Table 5. Regression coefficients (β) and *t*-test values for predicting LVMI

Independent variables	β (g/m ²)	<i>t</i>	P
Haemoglobin (g/dl)	-6.10	-2.87	0.005
24-h SBP (mm Hg)	0.01	0.07	0.95
24-h DBP (mm Hg)	0.94	4.26	<0.0001
Age (years)	-0.13	-1.33	0.19
Gender	0.15	1.56	0.12
Duration of CAPD	-0.12	-1.20	0.24
Diabetes	-0.07	-0.75	0.46
Serum albumin	0.03	0.25	0.80
IVCD index	-0.01	-0.10	0.92
	<i>r</i> ² = 0.30, <i>P</i> < 0.0001		

(52.5 ± 10.6% vs 55.5 ± 10.8%, *P* = 0.64). The IVCD index did not correlate with any of the variables, including 24 h SBP, 24 h DBP, duration of CAPD treatment, age, serum albumin or haemoglobin. In addition, ABPM did not correlate with age, duration of CAPD treatment, serum PTH or albumin.

The indexes of LVID, IVST and PWT were higher in Group A (uncontrolled HT) compared with Group B (normotensive or controlled HT), but these differences did not reach statistical significance. Our calculations revealed that low statistical power was due to inadequate patient numbers (Table 4). Similarly, the difference in prevalence of LVH was not significant (70% in Group A vs 60% in Group B, *P* = 0.41). The LVMI in Group A (145 ± 39 g/m²) was significantly higher than in Group B (118 ± 34 g/m², *P* < 0.01). The LVMI correlated positively with 24 h SBP (*r* = 0.46, *P* < 0.0001), 24 h DBP (*r* = 0.50, *P* < 0.0001), daytime SBP (*r* = 0.45, *P* < 0.0001), daytime DBP (*r* = 0.49, *P* < 0.0001), night-time SBP (*r* = 0.43, *P* = 0.0001), night-time DBP (*r* = 0.48, *P* < 0.0001), office SBP (*r* = 0.41, *P* < 0.0005) and office DBP (*r* = 0.24, *P* < 0.05), and inversely with haemoglobin (*r* = -0.40, *P* = 0.0005). There was a positive correlation between IVCD index and LVID, but not with other echocardiographic measurements of the left ventricle.

To define the independent determinants of LVMI, we modelled a multivariate analysis. The 24 h DBP and haemoglobin were found to be independent predictors of LVMI (*P* < 0.0001) (Table 5).

Discussion

Hypertension is a common cardiovascular disease and leads to severe morbidity in CAPD patients. In this study, prevalence of HT was 84% using office measurements and 82% with ABPM. Seventy-three per cent of the CAPD patients had uncontrolled HT according to daytime ABPM measurements. Patients with uncontrolled HT had significantly higher LVMI compared with patients having normotension or controlled HT. The IVCD index was also significantly increased in patients with uncontrolled HT.

Rocco *et al.* [2] reported that the prevalence of HT was 35% in a cohort of 926 CAPD patients, whereas Gunal *et al.* [5] found 60% using office measurements. Currently, ABPM is used frequently for diagnosing HT, and ABPM data correlated better with end-organ damage compared with office measurements in ESRD patients [21–23]. In our study, ABPM was as effective as office measurements in diagnosing HT and determining adequacy of BP control.

In the present study, LVMI was higher in patients with uncontrolled HT compared with patients having controlled HT and normotension. This finding is in agreement with previous studies. Takeda *et al.* [8] reported higher LVMI in CAPD patients having higher BP levels. In another group of CAPD patients that were followed for 18 months, the initial 52% prevalence of LVH increased to 76% [24]. On multiple regression analysis in the current study, 24 h DBP and haemoglobin were independent determinants of LVMI. However, we failed to show any correlation between IVCD index and LVMI. When analysing chest X-ray in ESRD patients, previous studies demonstrated a correlation between volume control and cardiac structure [5,25]. Ozkahya *et al.* [26] demonstrated regression of cardiothoracic index and LVH in HD patients by ultrafiltration. In this study, LVID index, IVST index and PWT index were increased in uncontrolled HT compared with normotension and controlled HT, but the difference did not reach statistical significance due to low patient numbers. The higher LVMI and regression of LVMI by ultrafiltration may be also explained by higher LVID in the former studies. In contrast to our study, Enia *et al.* [9] reported a higher degree of LVMI in their CAPD population and compared these results with HD patients. To our knowledge, there have been no reports examining the effect of fluid removal on LV wall thickness in CAPD patients.

A positive correlation between IVCD index and intravascular volume status has been described previously in HD patients [27]. Similarly, serum levels of atrial natriuretic peptide, a biochemical marker of intravascular volume status, correlated positively with IVCD index in CAPD patients [28]. In our study, there was no difference in urine output or ultrafiltration between Groups A and B. A volume overload in Group A may have been due to higher salt and water intakes. In a crossover, placebo-controlled study, Fine *et al.* [29] showed that the addition of 60 mmol of

sodium to the daily diet significantly increased blood pressure from 135/77 to 144/82 mm Hg in normotensive or mildly hypertensive CAPD patients. Recent studies have also suggested that peritoneal dialysis solutions with low sodium concentrations improve control of blood pressure by removal of excess sodium without a change in body weight or ultrafiltration volume [30]. Furthermore, Gunal *et al.* [5] reported normalization of BP in 78% of 47 hypertensive CAPD patients by salt restriction and ultrafiltration with hypertonic solutions.

The independent determinants of LVMI in our study were haemoglobin and 24 h DBP. Studies aiming to define the determinants of increased LVMI in CAPD patients are rare in the literature. In reports examining both HD and CAPD patients, haemoglobin and BP values were the primary determinants of LVMI [31–33]. Our study agrees with these previous reports by showing that uncontrolled HT had higher BP values and lower haemoglobin levels compared with controlled HT and normotensive patients.

The present study has several limitations. Firstly, this study was cross-sectional and the effect of volume status on the progression of LVM should be prospectively investigated in a larger group. The effect of volume removal by ultrafiltration on LVMI and on left ventricular wall thickness should also be investigated prospectively. Secondly, the reproducibility of ABPM may be questionable in ESRD patients. However, a previous report indicated that ABPM reproducibility was better than pre- or post-HD office BP measurements [34].

In conclusion, hypervolaemia as indicated by a higher IVCD index is a risk factor for uncontrolled HT and increased LVMI. Dietary instructions to limit salt intake may prevent volume overload in CAPD patients. We believe that a new prospective study with increased patient numbers would probably overcome the limitations of the present study.

Acknowledgements. The authors express their appreciation to Nural Bekiroglu for her assistance in the statistical analysis of the manuscript. Mehmet Koc is an ISN research fellow at the University of Florida (Gainesville), Division of Nephrology. Azra Bihorac, currently at the University of Florida (Gainesville), Division of Nephrology, was a fellow at the Division of Nephrology, Marmara University Medical Faculty at the time that this study was started.

References

- Excerpts from the USRDS 1996 Annual Date Report. *Am J Kidney Dis* 1996; 28 [Suppl 2]: S93–S102
- Rocco MV, Flanigan MJ, Beaver S *et al.* Report from the 1995 Core Indicators for Peritoneal Dialysis Study Group. *Am J Kidney Dis* 1997; 30: 165–173
- Cocchi R, Esposti ED, Fabbri A *et al.* Prevalence of hypertension in patients on peritoneal dialysis: results of an Italian multicentre study. *Nephrol Dial Transplant* 1999; 14: 1536–1540
- Rahman M, Dixit A, Donley V *et al.* Factors associated with inadequate blood pressure control in hypertensive hemodialysis patients. *Am J Kidney Dis* 1999; 33: 498–506
- Gunal AI, Duman S, Ozkahya M *et al.* Strict volume control normalise hypertension in peritoneal dialysis patients. *Am J Kidney Dis* 2001; 37: 588–593
- Cherix EC, Leunissen KML, Janssen JHA, Mooy JMV, van Hooff JP. Echography of the inferior vena cava is a simple and reliable tool for estimation of dry weight in hemodialysis patients. *Nephrol Dial Transplant* 1989; 4: 563–568
- Faller B, Lameire N. Evolution of clinical parameters and peritoneal function in a cohort of CAPD patients followed over 7 years. *Nephrol Dial Transplant* 1994; 9: 280–286
- Takeda K, Nakamoto M, Hirakata H, Baba M, Kubo M, Fujishima M. Disadvantage of long-term CAPD for preserving cardiac performance: an echocardiographic study. *Am J Kidney Dis* 1998; 32: 482–487
- Enia G, Mallamaci F, Benedetto FA *et al.* Long-term CAPD patients are volume expanded and display more severe left ventricular hypertrophy than haemodialysis patients. *Nephrol Dial Transplant* 2001; 7: 1459–1464
- Verdecchia P, Porcellati C, Schillaci G *et al.* Ambulatory blood pressure: an independent predictor of prognosis in essential hypertension. *Hypertension* 1994; 24: 793–801
- Luik AJ, Struijk DG, Gladziwa U *et al.* Diurnal blood-pressure variations in hemodialysis and CAPD patients. *Nephrol Dial Transplant* 1994; 9: 1616–1621
- The Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure. *Arch Int Med* 1997; 157: 2413–2448
- White WB, Berson AS, Robbins C *et al.* National standard for measurement of resting and ambulatory blood pressures with automated sphygmomanometers. *Hypertension* 1993; 21: 504–509
- Burt VL, Cutler JA, Higgins M *et al.* Trends in the prevalence, awareness, treatment, and control of hypertension in the adult US population: data from the health examination surveys, 1960 to 1991. *Hypertension* 1995; 26: 60–69
- Sahn DJ, DeMaria A, Kisslo J, Weyman A. Recommendations regarding quantitation in M-mode echocardiography: results of a survey of echocardiographic measurements. *Circulation* 1978; 58: 1072–1083
- Troy BL, Pombo J, Rackley CE. Measurements of left ventricular wall thickness and mass by echocardiography. *Circulation* 1972; 45: 602–611
- Devereux RB, Alonso DR, Lutas EM *et al.* Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. *Am J Cardiol* 1986; 57: 450–458
- Devereux RB, Reichek N. Echocardiographic determination of left ventricular mass in man. Anatomic validation of the method. *Circulation* 1977; 55: 613–618
- Levy D, Savage DD, Garrison RJ, Anderson KM, Kannel WB, Castelli WP. Echocardiographic criteria for left ventricular hypertrophy: The Framingham heart study. *Am J Cardiol* 1987; 59: 956–960
- Watson PE, Watson ID, Batt RD. Total body water volumes for adult males and females estimated from simple anthropometric measurements. *Am J Clin Nutr* 1980; 33: 27–39
- Erturk S, Ertug AE, Ates K *et al.* Relationship of ambulatory blood pressure monitoring data to echocardiographic findings in haemodialysis patients. *Nephrol Dial Transplant* 1996; 11: 2050–2054
- Tucker B, Fabbian F, Giles M, Thuraishingham RC, Raine AEG, Baker LRI. Left ventricular hypertrophy and ambulatory blood pressure monitoring in chronic renal failure. *Nephrol Dial Transplant* 1997; 12: 724–728
- Mansoor GA, White WB. Ambulatory blood pressure is a useful clinical tool in nephrology. *Am J Kidney Dis* 1997; 30: 591–605
- Eisenberg M, Prichard S, Barre P, Patton R, Hutchinson T, Sniderman A. Left ventricular hypertrophy in end-stage renal disease on peritoneal dialysis. *Am J Cardiol* 1987; 60: 418–419
- Amann K, Mandelbaum A, Schwarz U, Ritz E. Hypertension and left ventricular hypertrophy in the CAPD patient. *Kidney Int* 1996; 56: S37–S40

26. Ozkahya M, Ok E, Cirit M *et al.* Regression of left ventricular hypertrophy in hemodialysis patients by ultrafiltration and reduced salt intake without antihypertensive drugs. *Nephrol Dial Transplant* 1998; 13: 1489–1493
27. Katzarski KS, Nisell J, Randmaa I, Danielsson A, Freyschuss U, Bergström J. A critical evaluation of ultrasound measurement of inferior vena cava diameter in assessing dry weight in normotensive and hypertensive hemodialysis patients. *Am J Kidney Dis* 1997; 30: 459–465
28. Sakurai T, Ando Y, Masunaga Y, Kusano E, Asano Y. Diameter of the inferior vena cava as an index of dry weight in patients undergoing CAPD. *Perit Dial Int* 1996; 16: 183–185
29. Fine A, Fontaine B, Ma M. Commonly prescribed salt intake in continuous ambulatory peritoneal dialysis patients is too restrictive: results of a double-blind crossover study. *J Am Soc Nephrol* 1997; 8: 1311–1314
30. Nakayama M, Yokoyama K, Kubo H *et al.* The effect of ultra-low sodium dialysate in CAPD. A kinetic and clinical analysis. *Clin Nephrol* 1996; 45: 188–193
31. Foley RN, Parfrey PS, Harnett JD *et al.* Clinical and echocardiographic disease in patients starting end-stage renal disease therapy. *Kidney Int* 1995; 47: 186–192
32. Foley RN, Parfrey PS, Harnett JD, Kent GM, Murray DC, Barre PE. Impact of hypertension on cardiomyopathy, morbidity and mortality in end-stage renal disease. *Kidney Int* 1996; 49: 1379–1385
33. Foley RN, Parfrey PS, Harnett JD, Kent GM, Murray DC, Barre PE. The impact of anemia on cardiomyopathy, morbidity, and mortality in end-stage renal disease. *Am J Kidney Dis* 1996; 28: 53–61
34. Peixoto AJ, Santos SF, Mendes RB *et al.* Reproducibility of ambulatory blood pressure monitoring in hemodialysis patients. *Am J Kidney Dis* 2000; 36: 983–990

Received for publication: 5.12.01

Accepted in revised form: 24.4.02