J Am Soc Nephrol 13: 1733–1739, 2002

Cardiac and Renal Effects of Standard Versus Rigorous Blood Pressure Control in Autosomal-Dominant Polycystic Kidney Disease: Results of a Seven-Year Prospective Randomized Study

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Abstract. This study sought to investigate the cardiac and renal effects of rigorous *versus* standard BP control on autosomal-dominant polycystic kidney disease (ADPKD). A prospective, randomized, 7-yr study was performed to examine the effect of rigorous (<120/80 mmHg) *versus* standard (135–140/85–90 mmHg) BP control on left ventricular mass index (LVMI) and kidney function in 75 hypertensive ADPKD patients with left ventricular hypertrophy. LVMI was measured by echocardiogram at baseline and at 1 and 7 yr. Renal function was assessed by measuring serum creatinine and 24-h creatinine clearance every 6 mo for 3 yr, then annually for an additional 4 yr. The baseline characteristics were comparable in the two groups. During the study, average mean arterial pressure was 90 ± 5 mmHg for the rigorous group and 101 ± 4 mmHg for the standard group ($P < 0.0001$). The LVMI decreased by 21% in the standard group and by 35% in the rigorous group. A mixed model longitudinal data analysis revealed that rigorous BP control was significantly more effective in decreasing LVMI ($P < 0.01$). There was no statistically significant difference in renal function between the two groups. In conclusion, left ventricular hypertrophy, a major cardiovascular risk factor, was decreased to a significantly greater extent by rigorous than standard BP control. This finding has particular clinical importance because cardiovascular complications are the most common cause of death in ADPKD patients.

Autosomal dominant polycystic kidney disease (ADPKD) is the most common life-threatening hereditary disease in the United States, occurring in 1 of 400 to 1000 individuals (1). In ADPKD patients, hypertension and left ventricular hypertrophy (LVH) have been identified as factors associated with progression to end-stage renal disease (ESRD) (2). These factors are also extremely important cardiovascular risk factors (3). With the availability of ESRD treatment, cardiovascular complications have become the most common cause of death in ADPKD patients. Although reversal of LVH with antihypertensive treatment has been shown to occur in ADPKD patients (4) as well as patients with essential hypertension, there are no prospective randomized studies comparing rigorous (<120/80 mmHg) *versus* standard (135 to 140/85 to 90 mmHg) BP control in ADPKD patients on either LVH or renal function (5). This prospective, randomized, 7-yr study therefore was undertaken in 75 ADPKD patients to examine the cardiac and renal effects of rigorous *versus* standard BP control.

Received February 7, 2002. Accepted March 23, 2002.

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1046-6673/1307-1733
Journal of the American Society of Nephrology
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DOI: 10.1097/01.ASN.0000018407.60002.B9

Materials and Methods

Between 1991 and 1994, 79 ADPKD patients from our ADPKD Center who had established hypertension (BP ≥140/90 mmHg) and LVH were randomized to either rigorous (<120/80 mmHg) or standard (135 to 140/85 to 90 mmHg) BP control. The primary research hypothesis was that there would be a difference between treatment groups in mean rate of decline of GFR and in mean left ventricular mass index (LVMI) from baseline to year 7. Inclusion criteria for the subjects in the study presented here were as follows: subjects had to be between 20 and 60 yr of age, subjects had to have creatinine clearances more than 30 ml/min per 1.73 m^2, and men had to have a LVMI >125 g/m^2 and women had to have a LVMI >110 g/m^2. The following subjects were excluded: subjects who could not tolerate antihypertensive medication withdrawal, subjects who could not tolerate the study medications, subjects with >3 g urinary protein per day or those with a second renal diagnosis, subjects who required antiarrhythmic medications, lactating or pregnant subjects or subjects taking oral contraceptive medications, subjects with underlying psychiatric disorders, and subjects who, by the discretion of the investigator, were thought to be unable to comply with the guidelines of the protocol. Additionally, subjects with LVH due to primary causes other than hypertension were excluded from the trial.

All 79 subjects were sequentially randomized with stratification by renal function to rigorous or standard BP control via computer-generated randomization codes. After the medication washout period, 72 patients were randomized to either enalapril or amlodipine: 36 were randomized to enalapril (escalating dose 5, 10, 20, 40 mg) and 36 to amlodipine (escalating dose 5, 10 mg). However, the randomization to the 2 antihypertensive medications was terminated prema-

turely after a mean of 2.1 yr of follow-up because funding was lost. More patients thereafter received enalapril, rather than amlodipine, a decision based on physician and patient choice. The comparative effect of the two drugs was still considered by including in a subanalysis 69 of the original patients who continued to receive enalapril or amlodipine for at least 80% of their study time.

After a 2- to 4-wk medication washout period baseline, sitting BP was measured in the General Clinical Research Center (GCRC) at the University of Colorado Health Sciences Center by trained nurses and physicians with a Dinamap apparatus (Critikon Inc., Tampa, FL). All subjects underwent a baseline two-dimensional echocardiogram. Baseline echocardiography was performed on all subjects with a HP (Hanover, MA), Sonos, Model 500, 1989 cardiac ultrasound machine. At year 1, echocardiography was performed on all subjects with a HP, Sonos, Model 1000, 1991 cardiac ultrasound machine. At year 7, echocardiography was performed on all subjects with an Agilant, Sonos, Model 5500, 1999 cardiac ultrasound machine. The standardized method of the American Society of Echocardiography was used for all echocardiograms.

Over the 7 yr of the study, there were multiple readers similarly trained and blinded to the study protocol. LVMI was calculated by the Penn equation and corrected for body surface area (6). LVH was defined as a LVMI >125 g/m^2 for men or >110 g/m^2 for women; these definitions were chosen on the basis of the mean plus 2 SD in a healthy control population examined at our center (3). Renal function was assessed by 24-h creatinine clearance obtained on the GCRC. Laboratory technicians calculating clearance studies were blinded to the study protocol. All subjects were maintained on their standard diets with recommended moderate restriction of sodium intake during the 7-yr study.

Eleven subjects from the Denver metropolitan area were initially followed with weekly visits to the GCRC, where dose adjustments were made until the desired BP goal was reached. The remaining 68 subjects were given BP cuffs for home monitoring. The BP cuffs were calibrated against the GCRC Dinamap by the nurse coordinator. Subjects were contacted weekly by the nurse coordinator to record home BP measurements and make dose adjustments. In all subjects, the mean of three sitting BP was used to determine BP level. If more medications were needed to achieve the BP goal, hydrochlorothiazide, clonidine, spironolactone, or some combination of these were added as necessary. Rarely, other antihypertensive medications were added at the discretion of the study physician.

Once the BP goal was reached, patients were contacted monthly during the first year and every 2 mo thereafter either by phone or by a GCRC clinic visit to check their BP and review, and if necessary adjust, the medications. In addition, BP was rechecked 1 wk after any medication alteration. Nurse and physician clinicians could not be blinded to the study because of the need to monitor the subjects' BP to the standard or rigorous BP goal. Subjects returned to the GCRC every 6 mo during the first 3 yr of the study and then annually for 4 yr more. Each GCRC visit included a history, physical examination, and renal function assessment. Echocardiograms were obtained at baseline and at the 1- and 7-yr visits.

A Data Safety Monitoring Committee composed of independent physicians reviewed the data annually and was instructed to recommend stopping the study if there were any safety issues.

Wilcoxon rank sum tests were performed to examine whether the two BP control groups and two medication groups were equivalent at baseline. Longitudinal data were analyzed by PROC MIXED (SAS Inc., Cary, NC). PROC MIXED accounts for the fact that multiple observations on a subject are correlated and is able to use all data collected on each subject. For all analyses, time was measured in years from the first visit. Results were considered to be significant at the $\alpha = 0.05$ level. A mixed-model longitudinal data analysis with a random intercept was used to test for an effect of BP control group on LVMI in patients with ADPKD with gender as a covariate. A mixed model longitudinal data analysis was also used to test for an effect of BP control group on kidney function, as measured by the log of the mean 24-h creatinine clearance in patients with ADPKD. The log of the 24-h creatinine clearance was used because of the heavy-tailed distribution of this variable. PROC MIXED was also used to test for an effect of drug used (amlodipine *versus* enalapril) on LVMI and renal function. Only subjects who remained on the same drug (either amlodipine or enalapril) for 80% or more of their study time were included in these analyses.

Results

Seventy-nine hypertensive ADPKD patients with LVH were randomized to either rigorous or standard BP control. Four subjects were excluded from analysis for the following reasons: 2 subjects had cardiac valvular disease (1 with mitral insufficiency, 1 with aortic insufficiency), which had caused their LVH; 1 subject had protein excretion >3 g/d; and 1 subject was determined not to have ADPKD. Of the four excluded from analyses, three were randomized to standard BP control and one was randomized to rigorous BP control. The remaining study population consisted of 41 men and 34 women; the mean age at study entry was 41 yr (range, 27 to 59 yr). Table 1 lists the baseline characteristics of the two study groups. Wilcoxon rank sum tests confirmed that the standard and rigorous BP control groups were equivalent at baseline with respect to LVMI, systolic and diastolic BP, and renal function as measured by serum creatinine and 24-h creatinine clearance. The two groups were also statistically equivalent on gender distribution.

During follow-up, a significant separation between the two groups was achieved for both systolic and diastolic BP, as shown in Figures 1 and 2. Average mean arterial pressure during the study was 90 ± 5 mmHg for the rigorous group and 101 ± 4 mmHg for the standard group ($P < 0.0001$). The number of antihypertensive medications needed to control BP was significantly different between the two groups. On average, 1.4 ± 0.6 drugs were needed in the standard control group and 2.7 ± 0.8 in the rigorous control group ($P < 0.0001$).

Of the 75 patients that began the study, 53 patients completed the study through year 7, returning for the final echocardiogram and renal function assessment. Of the 22 subjects who left the study early, 2 women died as a result of breast cancer and stomach cancer (1 in each group), 8 entered ESRD (3 in the standard and 5 in the rigorous control group), and 12 dropped out by choice (5 in the standard and 7 in the rigorous group). A χ^2 test of independence showed that there was no relationship between designated BP control group (*i.e.* standard *versus* rigorous) and those patients who dropped out of the study (P = NS). Patients who entered ESRD had worse renal function at baseline than those who remained in the study (24-h creatinine clearance 45 ± 12 *versus* 88 ± 24 ml/min per 1.73 m^2, $P < 0.05$). Those patients who progressed to ESRD experienced a faster rate of decline in GFR than those patients who remained in the study (-7.5 ± 22 *versus* -3.9 ± 3.7 ml/min per 1.73 m^2 per year, $P < 0.05$). Of the

Table 1. Baseline characteristics of standard and rigorous BP control groups

Parameter	Standard Group			Rigorous Group			*P* Values
	Mean	SD	*n*	Mean	SD	*n*	
Age (yr)	40	8	34	42	8	41	NS
Left ventricular mass index (g/m^2)	156	27	34	161	24	41	NS
Systolic BP (mmHg)[a]	142	17	34	143	15	41	NS
Diastolic BP (mmHg)[a]	96	11	34	95	11	41	NS
Mean arterial pressure (mmHg)[a]	111	12	34	111	12	41	NS
Hematocrit (%)	42.5	4.69	34	42.3	4.30	41	NS
Serum creatinine (mg/dl)[b]	1.4	0.51	34	1.3	0.47	41	NS
Creatinine clearance (ml/min per 1.73 m^2)	82	28	34	84	29	41	NS
Male/Female	19/15		34	22/19		41	NS
Amlodipine/enalapril	15/19			15/26			NS

[a] Blood pressures were measured after a 2- to 4-week washout period without hypertensive medications.
[b] To convert serum creatinine values to micromoles per liter, multiply by 88.4.

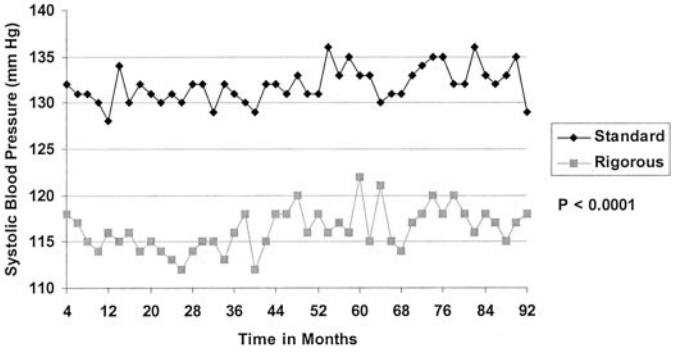

Figure 1. Mean sitting systolic BP from the fourth month through year 7 of patients with ADPKD randomized to rigorous (<120/80 mmHg) or standard (135 to 140/85 to 90 mmHg) BP control. The systolic BP were significantly different between groups ($P < 0.0001$).

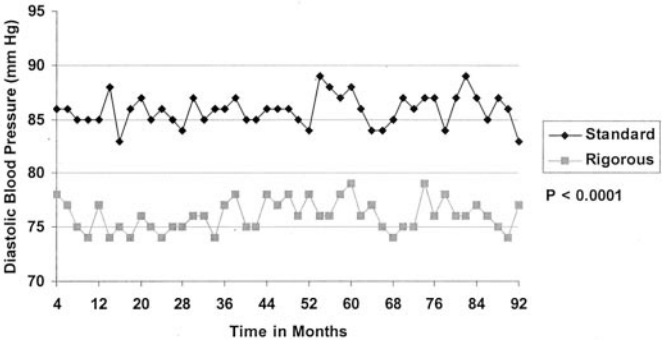

Figure 2. Mean sitting diastolic BP from the fourth month through year 7 of patients with ADPKD randomized to rigorous (<120/80 mmHg) or standard (135 to 140/85 to 90 mmHg) BP control. The diastolic BP were significantly different between groups ($P < 0.0001$).

subjects who entered ESRD, three were women and five were men (P = NS).

Time to ESRD was similar for standard (3.2 ± 1.8 yr) and rigorous (4.0 ± 1.4 yr) BP control groups (P = NS). Patients who dropped out of the study by choice were similar in LVMI, renal function, BP, and number of medications taken to those who remained in the study. Moreover, for those patients who dropped out by choice, baseline renal function and time to dropout were similar in patients randomized to standard *versus* rigorous BP control. Six were women and six were men (P = NS). Because of the characteristics of PROC MIXED, all data points contributed by an eligible patient until the time of dropout were used in the analyses.

Both rigorous and standard BP control resulted in a significant reduction of LVMI at year 7 compared with baseline (Figure 3). However, rigorous BP control resulted in a significantly lower LVMI than standard BP control over time ($P < 0.006$). After 7 yr of follow-up, 71% of patients in the rigorous BP control group achieved a LVMI in the normal range for women (<110 g/m^2) and men (<125 g/m^2) as compared with 44% in the standard BP control group ($P < 0.05$). The proportion of patients who received enalapril *versus* amlodipine was similar in the rigorous (20/7) and standard control (18/7) groups (P = NS). One patient in the standard group received propanolol for the last year of the study. Men had greater LVMI than women. There was a significant interaction between BP control group and gender on LVMI over time ($P < 0.05$); rigorous BP control was demonstrated to be particularly important for male ADPKD patients with LVH. Men in the standard group experienced a decrease in LVMI from 163 ± 28 to 134 ± 27 compared with a decline from 174 ± 21 to 108 ± 23 for men in the rigorous group ($P < 0.005$). Women in the standard group experienced a decrease in LVMI from 147 ± 23 to 112 ± 31 compared with a decline from 144 ± 16 to 99 ± 29 in the rigorous group, a difference that did not reach statistical difference (Tables 2 and 3).

Renal function as assessed by 24-h creatinine clearance decreased in both BP control groups over the 7-yr study (Figure 4). To compare the renal function changes over time between the two BP control groups, a mixed-model longitudinal data analysis was used. For the log of the mean 24-h creatinine clearance, the model with the best fit was one that estimated the random intercept and

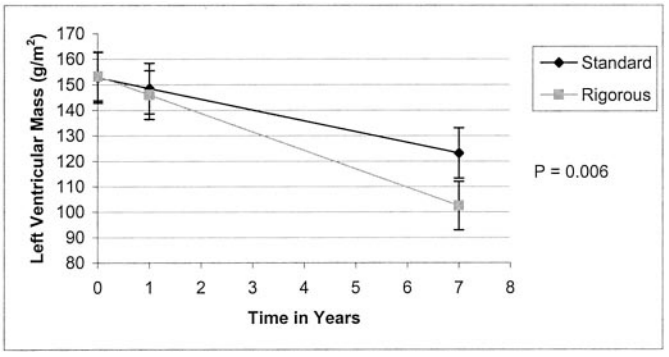

Figure 3. Effect of rigorous *versus* standard BP control on left ventricular mass index (LVMI) over 7 yr. Measurements on LVMI were taken at baseline, 1, and 7 yr and are expressed as the maximum likelihood estimates for each group with 95% confidence intervals. The number of patients at baseline, 1 yr, and 7 yr in the standard group were 34, 34, and 25, respectively, and in the rigorous group were 41, 39, and 28, respectively. Reversal of left ventricular hypertrophy was significantly greater with rigorous than with standard BP control.

slope for time with age at first visit as a covariate. There was no significant difference between rigorous *versus* standard BP control on the mean creatinine clearance over time ($F_{1, 484} = 0.44$, $P = 0.5064$) (Figure 4). There was a significant effect of age at first visit ($F_{1, 484} = 16.93$, $P < 0.0001$); specifically, the older the patient at entry into the study, the lower the 24-h creatinine clearance. Creatinine clearance decreased with time ($F_{1, 71} = 46.24$, $P < 0.0001$). For the entire group of patients, the creatinine clearance declined at a rate of 4.2 ± 4.2 ml/min per 1.73 m^2 per year. Additional analyses demonstrated no significant difference on 24-h creatinine clearance between subjects receiving diuretics ($n = 46$) to meet their BP goal and subjects who were not receiving diuretics ($n = 29$).

The effects of enalapril *versus* amlodipine on LVMI were also studied in a nonrandomized manner over the 7 yr of the study. Data from 69 subjects who were on one of the two drugs for at least 80% of their study time were used in this analysis. There were 20 subjects in the amlodipine group and 49 in the enalapril group. The amlodipine and enalapril groups, respectively, were not significantly different at baseline with respect to LVMI (159 ± 25 *versus* 159 ± 25 g/m^2), mean arterial pressure (109 ± 14 *versus* 113 ± 11 mmHg), age (43 ± 9 *versus* 41 ± 7 yr), gender distribution, serum creatinine (1.54 ± 0.65 *versus* 1.31 ± 0.34 ml/min per 1.73 m^2), or 24-h creatinine clearance (79 ± 31 *versus* 84 ± 24 ml/min per 1.73 m^2).

Mean BP levels during the 7 yr of the study were not significantly different between the enalapril and amlodipine groups. Longitudinal data analyses revealed that enalapril was more effective than amlodipine for reversing LVH over time ($F_{1,116} = 7.83$, $P < 0.01$). LVMI decreased from 159 ± 25 to 106 ± 25 g/m^2 ($P < 0.001$) in the enalapril group and from 159 ± 25 to 133 ± 33 g/m^2 ($P < 0.05$) in the amlodipine group. After 7 yr of follow-up, 67% of subjects receiving enalapril achieved a mean LVMI in the normal range for women and men, as compared with 36% of patients receiving amlodipine ($P < 0.05$). Additional analyses revealed that there was a significant interaction between BP control group and drug over time ($P < 0.005$). Specifically, rigorous BP control with enalapril lead to the greatest reduction in LVMI over time. There was no significant difference between enalapril and amlodipine on the 24-h creatinine clearance over time.

Discussion

Hypertension occurs early in patients with ADPKD before any substantial decrease in kidney function. At our ADPKD center, the average age at onset of hypertension (BP >140/90 mmHg) was 29 yr (7). It is not known, however, whether early treatment of the hypertension associated with ADPKD will alter the course of the associated renal and cardiac abnormalities. With the availability of ESRD treatment, including transplantation and dialysis, the main cause of death in ADPKD patients is due to cardiovascular complications (8–11). Our studies have shown that 48% of hypertensive ADPKD patients have LVH at a mean age of only 44 yr (3).

Of all known cardiovascular risk factors for morbidity and mortality, there is none more ominous than LVH. In the Framingham study, one-third of men and one-fifth of women with LVH by electrocardiogram were dead at 5 yr of follow-up (12). The associations of LVH with systolic and diastolic dysfunction, congestive heart failure, ischemic cardiac disease, arrhythmias, and sudden death account for this ominous prognosis. Although meta-analysis showed regression of LVH with reductions in BP (5), there have been no prospective studies comparing standard *versus* rigorous BP control in either patients with kidney disease or essential hypertension.

Against this background was performed the 7-yr prospective randomized study presented here in 75 hypertensive ADPKD patients with LVH to examine the effect of rigorous (<120/80 mmHg) *versus* standard (goal 135 to 140/85 to 90 mmHg) BP control on LVH and renal function. The baseline characteristics, including age, gender, systolic and diastolic BP, and renal function (creatinine clearance), were comparable in the standard and rigorous BP control groups. Over the 7 yr of follow-up, the mean BP in the standard group was 101 ± 4 mmHg compared with 90 ± 5 mmHg in the rigorous group.

The results of the study presented here demonstrate that both standard and rigorous BP control can decrease LVH significantly over a 7-yr period. The LVMI decreased from 156 to 123 g/m^2 in the standard group and from 161 to 104 g/m^2 in the rigorous group. By use of mixed-model longitudinal data analysis, the difference between the standard and rigorous groups was found to be significant. Significantly more subjects in the rigorous BP control group (71%) than in the standard BP control group (44%) achieved normal LVMI. The percentage of patients receiving an angiotensin-converting enzyme (ACE) inhibitor *versus* a calcium channel blocker was not significantly different between the rigorous and standard treatment groups. A subanalysis of patients on either enalapril or amlodipine and assigned to rigorous or standard BP control, however, revealed greater benefit from rigorous BP control with enalapril.

These results therefore support a BP goal of less than 120/80

Table 2. Mean and SD for men in standard and rigorous BP control groups on all measurements at baseline and 7 years

Parameter[a]	Standard			Rigorous		
	Mean	SD	*n*	Mean	SD	*n*
Baseline						
LVMI (g/m^2)	163	28	19	174	21	22
systolic BP (mmHg)[b]	145	16	19	145	15	22
diastolic BP (mmHg)[b]	99	10	19	98	11	22
MAP (mmHg)[b]	115	11	19	114	12	22
hematocrit	45.0	4.04	19	44.6	3.80	22
serum creatinine (mg/dl)[c]	1.45	0.45	19	1.48	0.57	22
creatinine clearance (ml/min per 1.73 m^2)	89	26	19	86	26	22
Year 7						
LVMI (g/m^2)	134	27	13	108	23	17
systolic BP (mmHg)	131	10	13	117	14	17
diastolic BP (mmHg)	84	6	13	77	7	17
MAP (mmHg)	99	6	13	90	8	17
hematocrit	41.3	4.21	13	42.1	5.34	17
serum creatinine (mg/dl)[c]	2.75	2.21	13	2.04	0.93	17
creatinine clearance (ml/min per 1.73 m^2)	65	40	13	64	29	17

[a] LVMI, left ventricular mass index; MAP, mean arterial pressures.
[b] Blood pressures were measured a after 2- to 4-week washout period without hypertensive medications.
[c] To convert serum creatinine values to micromoles per liter, multiply by 88.4.

Table 3. Mean and SD for women in standard and rigorous BP control groups on all measurements at baseline and 7 years

Parameter[a]	Standard			Rigorous		
	Mean	SD	*n*	Mean	SD	*n*
Baseline						
LVMI (g/m^2)	147	23	15	144	16	19
systolic BP (mmHg)[b]	138	17	15	140	15	19
diastolic BP (mmHg)[b]	91	11	15	93	11	19
MAP (mmHg)[b]	107	12	15	109	11	19
hematocrit	39.3	3.38	15	39.6	3.12	19
serum creatinine (mg/dl)[c]	1.43	0.59	15	1.15	0.22	19
creatinine clearance (ml/min per 1.73 m^2)	74	30	15	80	21	19
Year 7						
LVMI (g/m^2)	112	31	12	99	29	11
systolic BP (mmHg)	130	24	12	123	12	11
diastolic BP (mmHg)	80	9	12	77	7	11
MAP (mmHg)	97	14	12	93	7	11
hematocrit	37.0	5.45	12	38.9	3.45	11
serum creatinine (mg/dl)[c]	2.12	1.24	12	1.53	0.69	11
creatinine clearance (ml/min per 1.73 m^2)	50	25	12	64	26	11

[a] LVMI, left ventricular mass index; MAP, mean arterial pressures.
[b] Blood pressures were measured after a 2- to 4-week washout period without hypertensive medications.
[c] To convert serum creatinine values to micromoles per liter, multiply by 88.4.

mmHg and the use of an ACE inhibitor for hypertensive ADPKD patients with LVH. This observation has important clinical implications because several studies have shown that regression of LVH is associated with fewer cardiovascular events (13–15). Whether the BP goal of less than 120/80 mmHg should be recommended for hypertensive ADPKD patients without LVH cannot be determined from the study presented here. However, because of the frequency of LVH in hypertensive ADPKD patients, such a recommendation would seem reasonable.

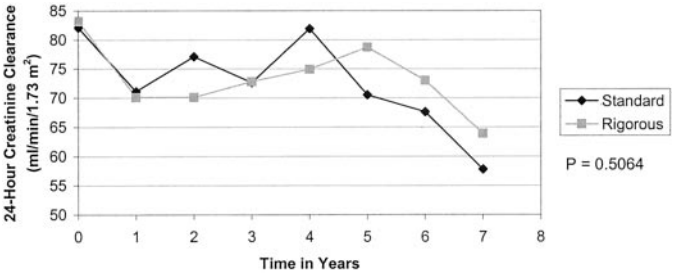

Figure 4. Effect of rigorous *versus* standard BP control on 24-h creatinine clearance over 7 yr. Measurements were obtained annually for 7 yr. There is no difference between groups on renal function.

In the study presented here, a subgroup analysis of patients receiving enalapril *versus* amlodipine for at least 80% of their study time demonstrated a significantly greater decrease in LVH in the enalapril group than the amlodipine group in spite of similar BP control. This finding is consonant with meta-analysis results in patients with essential hypertension that have indicated a greater reversal of LVH with ACE inhibitors than other antihypertensive agents (16). Other investigators have, however, found a comparable reversal of LVH with ACE inhibitors *versus* calcium channel blocker (5). It should be emphasized, however, that a greater activation of the renin-angiotensin-aldosterone system has been shown in hypertensive ADPKD patients as compared with matched patients with essential hypertension (17–21). This finding in ADPKD patients therefore could explain the greater benefit of ACE inhibitors with enalapril *versus* amlodipine in the study presented here.

No difference in renal function was detected in the study presented here between the standard and rigorous BP control groups. The failure to detect a difference in renal function between the rigorous and standard groups, however, may be the result of inadequate statistical power. It nevertheless should be emphasized that ADPKD patients with hypertension (BP >140/90 mmHg) progress much faster to ESRD than those without hypertension (22,23). The results presented here, however, do not indicate that a BP goal of less than 120/80 mmHg over 7 yr can protect renal function better than a goal of 135 to 140/85 to 90 mmHg in middle-aged ADPKD patients with slightly decreased renal function at baseline. Because hypertension in ADPKD patients may develop in the second or third decade of life, it is possible that earlier intervention than that undertaken in the study presented here to maintain BP at less than 120/80 mmHg could postpone ESRD. In an earlier longitudinal study, it was shown that ADPKD patients only treated with ACE inhibitor exhibited a slower renal progression than ADPKD patients treated only with diuretics (24). A comparison with the results presented here is, however, not possible because ADPKD patients treated with either enalapril or amlodipine received diuretics.

In summary, the results presented here of a 7-yr prospective randomized study in hypertensive ADPKD patients with LVH demonstrate a dramatic decrease in LVMI with rigorous BP control. Specifically, the reversal of LVH was significantly greater in subjects with a mean BP of 90 ± 5 mmHg as compared with 101 ± 4 mmHg. A subgroup analysis supports ACE inhibition to be the preferred initial antihypertensive agent as compared with a calcium channel blocker. Reversal of LVH in hypertensive ADPKD patients with a BP goal of less than 120/80 mmHg should have a major effect on cardiovascular morbidity and mortality in ADPKD.

Acknowledgments

This research was supported by grant 5 P01 DK34039, Human Polycystic Kidney Disease, awarded by the Department of Health and Human Services, Public Health Service, National Institute of Diabetes, Digestive, and Kidney Diseases, and the Clinical Research Center, and grant MORR-00051 from the GCRC Research Program of the Division of Research Resources, National Institutes of Health. Funds were also provided by the Zell Family Foundation. Pfizer Inc. provided funding for part of the study, for up to 3 yr per patient. We thank the study coordinator, Nora Glass, M.S.N.; and we thank Richard H. Jones, Ph.D., for his help with the longitudinal data analysis.

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