Renovascular disease due to progressive atherosclerotic renal artery stenosis is being diagnosed with increasing frequency in the elderly. At what degree of renal artery stenosis should intervention be recommended is not clear. To answer this question, unilateral and bilateral activation of the renin-angiotensin system (RAS) or its absence were detected by captopril-stimulated renal vein renin measurements in 49 hypertensive patients, aged 63, and the information was matched against radiographic measurements of the extent of renal artery stenosis. All patients had normal or near-normal renal function (serum creatinine <2.0 mg/ml), single artery per kidney, and no previous renal artery intervention. There were 9 patients with normal secretion, 27 with unilateral hypersecretion and contralateral suppression, and 13 with bilateral hypersecretion of renin. Of the 53 hypersecreting kidneys all but 4 had renal artery (luminal area) stenosis of 80% or greater. All 45 kidneys with normal secretion or complete suppression of renin release contralateral to a hypersecreting kidney had either normal caliber renal artery or renal artery stenosis less than 80%, with two exceptions of 80% stenosis each. These findings suggest that renal artery stenosis of at least 80% is required for activation of the RAS.

Consequently, patients with renal artery stenosis less than 80% should be monitored rather than treated because improvement of renal function and amelioration of hypertension are not expected unless the RAS has been activated in the affected kidneys.

Key Words: Hypertension; renovascular; renal vein renins; angioplasty; stenting

**M002**

**LEFT VENTRICULAR HYPERTROPHY (LVH) IN HYPERTENSIVE PATIENTS WITH AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DISEASE (ADPKD): INFLUENCE OF BLOOD PRESSURE (BP) AND HUMORAL AND NEUROHORMONAL FACTORS**

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**Background.** LVH is a common finding in hypertensive ADPKD patients. There are few studies on the influence of ambulatory BP profile and non-hemodynamic factors on LVH in these patients. The aim of this study was to evaluate the relationship between BP, humoral and neurohormonal factors and left ventricular mass (LVM) in hypertensive ADPKD patients.

**Methods.** In 20 hypertensive ADPKD patients with normal or mildly impaired renal function (serum creatinine <177 umol/l), ambulatory BP was monitored for 24 hours, left ventricular dimensions were estimated by echocardiography, and plasma renin activity (PRA), plasma noradrenaline (NA), angiotensin II (Ang II), aldosterone, atrial natriuretic peptide (ANP) and insulin-like growth factor I (IGF-I) were also determined. Twenty age- and sex-matched essential hypertensive subjects served as controls.

**Results.** Ambulatory BP profile and LVM index were similar in the two groups, although male ADPKD patients had higher LVM indices than their matched controls (130 ± 18 vs 109 ± 19.6 g/m², p = 0.02). The prevalence of LVH (40% vs 30%), left ventricular geometry, Doppler parameters and the percentage of patients with diastolic dysfunction was similar in the two groups. PRA, Ang II, aldosterone, ANP and IGF-I levels did not differ between groups, but plasma NA levels were higher in ADPKD patients than in controls (281 ± 158 vs 160 ± 62 pg/ml, p = 0.004). ADPKD patients with LVH did not differ from those without LVH with regard to humoral and neurohormonal parameters, but had higher ambulatory BP levels. In ADPKD patients, simple regression analysis revealed a significant association between LVM index and 24-hour, daytime and nighttime systolic and diastolic BP, but not with any of the hormonal factors evaluated. On multiple regression analysis, nighttime diastolic BP was the only independent variable linked to LVM index.

**Conclusions.** Our data emphasize a different interaction between LVM, BP and humoral and neuro-hormonal factors in hypertensive ADPKD patients. LVM is closely related to ambulatory BP but not to the level of neurohormonal activity. Further studies are warranted to elucidate the role of non-hemodynamic factors in the pathogenesis of LVH in the ADPKD population.

Key Words: Blood pressure; left ventricular hypertrophy; neurohormonal factors; autosomal dominant polycystic kidney disease

**M003**

**CARDIAC INVOLVEMENT IN AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DISEASE (ADPKD): AN HYPERTENSIVE HEART DISEASE**

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**Background.** ADPKD is a multisystemic disease in which the cardiovascular system is involved. Valvular abnormalities (specifically mitral valve prolapse) are the main cardiac manifestation of the disease. Given the variable prevalence of these abnormalities and the fact that cardiac structure and function may be affected by the high prevalence of hypertension in these patients, the extent of cardiac involvement in ADPKD has yet to be well defined.

**Methods.** In order to determine the profile and prevalence of cardiovascular disorders during the course of ADPKD, Doppler Color Echocardiography was performed in 130 ADPKD patients. Patients were divided into normotensive