completed 4 examinations, were analyzed. Left ventricular mass decreased significantly during the first 6 months (from 302.4 ± 6.6 to 151.8 ± 13.3 g; mean ± SEM) after renal transplantation, but increased during the following 6 months (293 ± 10.5 g; mean ± SEM). Pulse pressure remained unchanged throughout the study period (before renal transplantation: 60.7 ± 2.7 mmHg, 12 months after allografting: 61.8 ± 3.2 mmHg; mean ± SEM). There was no correlation (Pearson) between LVH and pulse pressure. Pulse pressure was strongly correlated with age (P<0.01), whereas LVM correlated positively with BMI (P<0.04). In renal transplant recipients, there is no association between LVH and pulse pressure.

Key Words: left ventricular hypertrophy, kidney transplantation, pulse pressure

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RELATIONSHIP BETWEEN ECHO-DOPPLER VELOCIMETRIC INDICES AND RENAL HEMODYNAMICS IN STENOTIC AND NON STENOTIC KIDNEYS

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Echocardiograms were obtained for controlling blood pressure (BP) and PP in subjects with normal renal status but with a history of hypertension. They were compared to the control data in patients with normal renal status but without a history of hypertension.

Key Words: echo-Doppler velocimetric indices, renal scintigraphy, renal hemodynamics

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REPLENISHMENT OF PTH1 RECEPTOR POOL BY PERIPHERAL RECEPTOR GENE DELIVERY REDUCES RENAL TONUS IN SPONTANEOUSLY HYPERTENSIVE RATS (SHR)

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A number of observations prompted us to explore the PTHrP system in renal vessels of SHR. First, the kidney is acknowledged as a major target of the dilatory effect of PTHrP in human and animal studies. Second, genetically determined renal mechanisms, including high renal vascular resistance (RVR), play a major role in the development of primary hypertension. Third, PTHrP mRNA has been shown to be upregulated in the aorta in genetic, as well as experimental hypertension. Finally, we have reported earlier that the vasodilatory effect of PTHrP was reduced in SHR isolated perfused kidney (IPK), as compared to normotensive animals.

We now report that in small arteries isolated from kidney of 12-week-old SHR, immunoreactive PTHrP expression was increased by ~40%, while both PTH1 receptor (PTH1R) mRNA (RT-PCR) and protein (Western blot) were decreased by ~60-80%, as compared to age-matched WKY rats (p<0.05). These observations suggested potential beneficial effects of somatic human (h)PTH1R gene delivery to decrease RVR in SHR. To test this possibility, the hPTH1R DNA (1.9 kb), under the control of the cytomembranosy promoter in the pCDNA1.1 vector was generated. The naked DNA construct (0.5mg) was delivered into SHR via a single intravenous injection. Control-treated SHR received 0.5mg empty vector. Three weeks after plasmid delivery, the expression of hPTH1R mRNA in IPKs was identified by RT-PCR analysis in all main organs, including heart, brain, aorta, liver and lungs. In kidney, hPTH1R transcript was preferentially expressed in isolated intrarenal arteries as compared to total kidney. In IPKs, a submaximal concentration of PTHrP(1-36) (10 nM) decreased the preconstriction induced by 10 μM phenylephrine, by 19±11% in control-treated SHR (n=5, p>0.05). This vasodilatory effect was increased by 54±8% (n=5, p<0.05) in PTH1R-transfected SHR. PTH1R gene delivery, also decreased basal RVR of the IPK from 7.2±0.2 (n=9) in control-treated to 5.2±0.3 mmHg min ml−1 g −1 (n=13) in hPTH1R-transfected (p<0.05). Finally, infusion of 40nM PTHrP(3-36), an antagonist of PTH1R, had no effect in control-treated SHR, but displayed vasocostrictory effect, increasing basal RVR in IPK by ~13% (n=4, p<0.05) in PTH1R-transfected SHR.

Collectively, these results strongly suggest that downregulation of PTH1R contributes to high RVR in SHR, despite the upregulation of its endogenous ligand. Repplenishment of renal vascular pool of PTHrP by somatic gene delivery decreased RVR, owing to endogenous vasodilatory PTHrP. Thus, in SHR, endogenous PTHrP system modulates renal tonus, at least in vivo.

Key Words: spontaneously hypertensive rats, parathyroid hormone-related protein (PTHrP), renal vessels

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EFFECT OF INTRAVENOUS FOLIC ACID OR FOLIC ACID THERAPY ON MEAN ARTERIAL PRESSURE (MAP) AND PULSE PRESSURE (PP) IN HEMODIALYSIS PATIENTS

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Because therapy with non-reduced or reduced folic acid derivatives and the resulting reduction of total homocysteine (tHcy) plasma levels may improve blood pressure (BP) and PP in subjects with normal renal