p < 0.02], statistically significant stepwise decrease in Ang-(1-7) levels as a function of increases in ACE activity.

This association, independent of renin levels, suggests that Ang-(1-7) plays a role in homeostasis and contributes to imbalances of blood pressure regulation.

Key Words: Angiotensin-(1-7); renin-angiotensin system; blood pressure regulation; hypertension

I021
LACK OF ASSOCIATION BETWEEN ACE GENE POLYMORPHISM AND ATHEROMATOUS RENAL ARTERY STENOSIS

Objective: To test the hypothesis that polymorphism of angiotensin converting enzyme (ACE) gene I/D polymorphism is linked to the atheromatous renal artery stenosis.

Design and methods: ACE gene polymorphism was determined using PCR method in 40 Caucasian patients with angiographically confirmed renal artery stenosis compared to 73 control subjects matched for age and gender from the same ethnic group. Presence of other risk factors (cholesterol, tryglicerides, serum glucose, smoking) was also analyzed as well as the severity of hypertension. History of cardiovascular or cerebrovascular disorders was also analyzed.

Results: The distribution of ACE gene polymorphism was II (32.5%), ID (40%) 16 DD 11 (27.5%); and controls II 18 (24.8%), ID 31 (42.4%), DD 24 (32.8%) (p > 0.05). Therefore, the frequency of D allele was not significantly different from the control group (p > 0.05). We failed to find significant difference in blood pressure values between particular genotypes (p > 0.05). There were also no differences in fasting serum glucose, cholesterol and triglyceride levels between patients with DD genotype and others (p > 0.05). Also no association was found between ACE gene polymorphism and ECG findings and fundus examination (p > 0.05). However, although not statistically significant (p > 0.05), duration of hypertension was the shortest in patients with DD genotype (7.7 ± 3.8 vs. 11.3 ± 2.7 in II and vs. 9.6 ± 1.8 in ID).

Conclusion: According to our results ACE gene polymorphism does not seem to have a predisposing role in the development of atheromatous renal artery stenosis. However, the observed shorter duration of hypertension in patients with DD genotype could implicate that it might have some influence in the progression of this disease.

Key Words: ACE gene polymorphism; renal artery stenosis

I022
INFLUENCE OF ANGIOTENSIN CONVERTING ENZYME (ACE) GENE POLYMORPHISM AND CIRCADIAN BLOOD PRESSURE (BP) CHANGES ON LEFT VENTRICLE (LV) MASS IN COMPETITIVE OARSMEN

Objective: We analyzed correlation between ACE gene polymorphism and adaptive hypertrophic response of LV with regard to BP in competitive oarsmen.

Design and methods: ACE gene polymorphism was determined using PCR method in 52 oarsmen with mean age 26 years (range 18–37) and in age, sex and ethnically matched control group of 34 persons. The LV mass was determined echocardiographically and calculated after Devereux. BP was measured using both mercury sphygmomanometer and Ambulatory BP Monitor SpaceLabs 90207.

Results: The distribution of ACE gene polymorphism was: in competitive oarsmen 16 (47.1%) II, 12 (35.3%) ID, 12 DD (17.6%); in control group 8 (24%) II, 18 (52%) ID and 8 (24%) DD. The frequency of I allele was significantly greater in oarsmen (0.65 vs. 0.50; p < 0.05). We failed to find differences in BP values obtained in daytime (p > 0.05), while in oarsmen with DD genotype systolic BP in nighttime was significantly greater compared to persons with II genotype (121.8 ± 7.4 vs. 114.0 ± 7.8; p < 0.05). LV mass was found to be the largest in oarsmen with DD genotype (p > 0.05). No changes in geometrical structure of LV regarding ACE genotype were established. However, remodeling of LV was associated with increased variability of systolic and diastolic BP.

Conclusion: Observed greater frequency of I allele in competitive rowers could suggest that ACE gene polymorphism might contribute to the athletic performance. According to our results DD genotype and BP variability influences LV hypertrophic response in competitive athletes.

Key Words: ACE gene polymorphism; circadian blood pressure; left ventricle mass

I023
ASSESSMENT OF INTERSTITIAL FLUID ANGIOTENSIN II CONCENTRATION IN THE RAT HEART BY MICRODIALYSIS
F.M. Siri, S.M. Dolgilevich, and S.A. Atlas*. Hypertension Research Laboratory, Bronx Veterans Affairs Medical Center and Mount Sinai School of Medicine, New York, NY

As a model to study the relative contributions of local tissue synthesis vs. systemic delivery of angiotensin II (Ang II), the microdialysis technique was used to estimate cardiac interstitial fluid (ISF) levels of Ang II in the intact beating rat heart in situ. A dialysis probe (10 mm length, 0.5 mm OD) was inserted into the anterior myocardial wall (apex to base) of normal open-chest, pentobarbital-anesthetized rats. Krebs solution with 0.5% BSA was perfused at 2 μL/min for 30–60 min periods and samples were collected on ice in tubes containing EDTA. Blood samples were collected in EDTA prior to and following each period. Ang II was measured by radioimmunoassay following extraction of dialysates or plasma on C18 SepPak cartridges. Further analysis of immunoreactive (ir) Ang II was performed by reversed-phase high performance liquid chromatography (rpHPLC) using a C18 column eluted isocratically with 22% acetonitrile in water. In initial studies (n = 5) irAng II levels averaged 72 ± 53 (mean ± SD) pg/ml in cardiac ISF and 51 ± 4 pg/ml in plasma. By rpHPLC analysis the irAng II in plasma and ISF had retention times (9.5 min) corresponding to that of au-