of renal function, exercise training may or may not benefit renal function, as indicated by changes in GFR.

Key Words: Exercise, hypertension, renal function

P-158
BLOOD OXYGEN LEVEL-DEPENDANT MAGNETIC RESONANCE IMAGING IN RENAL ARTERY STENOSIS
Laurent Juillard, Lilach O Lerman, David G Krager, John A Haas, Brian C Rucker, Stephen J Riederer, Juan C Romero. Physiology, Mayo Clinic, Rochester, MN; MRI Research Lab, Mayo Clinic, Rochester, MN.

Background: Ischemic nephropathy is a common cause of end-stage renal disease. Exploration of the mechanisms of deterioration of renal function is limited due to lack of non invasive techniques available to study the single kidney. The Blood Oxygen Level-Dependent (BOLD) MRI method can measure desoxyhemoglobin and therefore estimates indirectly renal oxygen content, but has never been evaluated in renal artery stenosis (RAS). This study was therefore designed to test if BOLD can detect renal hypoxia induced by RAS.

Methods: RAS was induced in 8 pigs using an occluder placed around the right renal artery. Renal blood flow (RBF) was measured continuously with an ultrasound probe. BOLD signal was measured bilaterally in the cortex and medulla (as the slope of the logarithm of MR signal) at baseline and at the lower limit of RBF autoregulation. The measurements were then repeated during six sequential graded decreases in RBF (80 to 0%) of baseline and during recovery.

Results: At baseline, BOLD signals were not significantly different between the right and the left kidneys. In the occluded kidney, BOLD signal of the cortex (19.3±1.0%) and the medulla (17.3±2.0%) increased during occlusion gradually and significantly (p<0.0001) to a maximum (at total occlusion) of 33.8±2.0% (+79%) and 29.8±2.3% (+78%) respectively, and returned to baseline values during recovery.

Conclusion: This study shows that the BOLD technique can non invasively detect change in intra-renal oxygenation during an acute reduction of RBF. This study provides a strong rational for developing the BOLD method for the detection and evaluation of renal hypoxia induced by RAS, which may be potentially be applicable in humans.

Key Words: Ischemic nephropathy, experimental renal artery stenosis, MRI BOLD

P-159
NA+/H+ EXCHANGE ACTIVITY AND DIURETIC RESPONSE TO NOVEL NHE3 INHIBITORS IN THE SPONTANEOUSLY HYPERTENSIVE RAT (SHR)
Michael S LaPointe, Daniel Battle. Division of Nephrology, Northwestern University; VA Chicago HCS/Lakeside, Chicago, IL.

We recently reported that NHE3 protein abundance and activity are increased in proximal tubules in both the pre-hypertensive and hypertensive SHR as compared to WKY rats. The present study examined Na+/H+ exchange in brush border membrane vesicles (BBMV) and the effect of in vivo infusion of two novel specific NHE-3 inhibitors, S3226 and S1611 on blood pressure and renal function in SHR and age matched WKY rats 10–17 weeks of age. In the absence of the NHE-1 inhibitor, canicporide, Na+/H+ exchange activity (NHE3) was increased in BBMV from the SHR (1238±89, n=11 vs 935±47, n=10 units/5 sec, p<0.01). In vivo studies revealed that there were no differences in renal function between SHR and WKY rats after correction for body weight (Table). Infusion of S3226 or S1611, resulted in significant increases in urine flow and Na and Li excretions in both rat strains, although neither the absolute excretion rates, nor the percent changes from baseline were different between SHR and WKY rats (Table). Neither S3226 nor S1611 acutely lowered blood pressure in either rat strain.

Key Words: Sodium hydrogen exchange, SHR, NHE3

P-160
CARDIAC TROPONIN T (cTNT) LEVELS IN CHRONIC RENAL FAILURE (CRF) PATIENTS (pts)
Marian Guicochea, Maria Jose Gutierrez, Soledad Garcia De Vinuesa, Francisco Gomez-Campadera, Paula Blanco, Jose Luno. Nephrology, Hospital Gregorio Marañon, Madrid, Spain.

A significant and continuing controversy over cTNT levels is their specificity as serum marker of cardiac injury in patients with CRF. An increase in serum cTNT levels may be associated with subclinical myocardial injury and/or abnormalities of troponins catalysis induced by renal failure and/or hemodialysis itself. The aim of this study was to evaluate the factors which are associated with serum cTNT levels in a large population of patients (pts) with chronic renal failure.

176 consecutive outpatients (109 M, 67 F) were included. The exclusion criteria were any cardiovascular event during the three months preceding the start of the study. Prevalent cardiovascular disease, arterial pressure, antihypertensive drugs and lifestyle habits were recorded. We choose ECG criteria for left ventricular hypertrophy diagnosis. Routine clinical chemical variables and ischemic myocardial markers (mioglobin, CK-MB mass and cTNT) were measured. The pts were divided in two groups: group A (with normal renal function, creatinine levels ≤1.3 mg/dl) (n= 58) and group B (with renal insufficiency, creatinine levels > 1.3 mg/dl) (n=118).

Key Words: Sodium hydrogen exchange, SHR, NHE3