uncertain. Furthermore, there is no evidence that triglycerides or free fatty acids directly impair vascular function as opposed to reflex or hormonal changes induced following systemic exposure.

Methods: We tested the hypothesis that local HTG impairs resistance vessel endothelial function, by examining whether forearm blood flow (FBF) responses to intra-arterial ae-
ticholine (Ach), bradykinin (BK), and nitroprusside were altered by coinfusion of intralipid (IL 200 mg/min) for 90 min in 10 healthy adults, age 38±4, 7M.

Results: IL increased plasma triglycerides from 80±20 to 433±62 and free fatty acids from 0.5±0.1 to 1.9±0.2 (p<0.0003 for both). Forearm vasodilatation to Ach (p=0.93), BK (p=0.73), and nitroprusside (p=0.34) were not reduced by HTG (figure).

Conclusion: Local HTG sustained for >90 min does not impair resistance vessel endot-
hal function. These results suggest that resistance vessels are less susceptible to the deleterious effects of triglycerides than conduit vessels. Alternatively, the vascular effects of systemic HTG may be mediated indirectly, perhaps via hormonal or reflex mecha-
nisms.

POSTER SESSION

1160 Hormone Intervention in Cardiovascular Disease

Tuesday, March 09, 2004, 3:00 p.m.-5:00 p.m.
Morial Convention Center, Hall G
Presentation Hour: 3:00 p.m.-4:00 p.m.

1160-165 Angiotensin II Receptor 1 Blocker Improves Not Only Hypertension but Also Vascular Arteriogenicity

Chikashi Suga, Kazuo Matsumoto, Ritsushi Tosaka, Atsuaki Sakamoto, Masako Nozawa, Yukia Hotta, Osamu Komoto, Shigeyuki Nishimura, Hiroshi Suga, Saitama Medical School, Saitama, Japan

Background: Angiotensin II receptor 1 blocker (ARB) has been proven to improve car-
diovascular mortality and morbidity. However, the influence of ARB on arterial arrhythmia remained unknown. The purpose of this study was to evaluate ARB if any has beneficial effect on arteriogenic arteriopathy, and also if the effect is associated with the decrease of blood pressure.

Methods: This study consists of 69 patients (30 males, mean age 63.8±10 years) with hypertension in whom valsartan was administered from February 2001 to May 2002. We assessed mean blood pressure (MBP), RPP, SV, on ECG, QT dispersion (QTD) and QTc dispersion (QTDc) before and 10 months later of introduction of valsartan administration. We also assessed correlation between the difference of QTD or QTDc before and 10 month later of the valsartan administration and the difference of MBP or RPP, SV before and 10 month later of the valsartan administration.

Results: MBP (119.9±12.1mmHg vs. 101.9±10.6mmHg, p<0.0001), QTd (60.3±16.8msec vs. 47±11.3msec, p<0.0001) and QTcD (82±11.3msec vs. 68±8.9msec, p<0.0001) decreased after valsartan administration. The decrease of MBP remained significant even after 12 months of valsartan administration.

Conclusion: QT and QTDc decreased after valsartan administration. The decrease of QTcD and QTDc did not correlate with the change of blood pressure or RPP, SV.

1160-166 C-Type Natriuretic Peptide Improves Left Ventricular Performance at Rest and During Exercise After Heart Failure

Hiroshi Hasegawa, Atsushi Morimoto, Heng-Jie Cheng, William C. Little, Che-Ping Cheng, Wake Forest University School of Medicine, Winston-Salem, NC

Background: The diastolic dysfunction present at rest is exacerbated during exercise (Ex) in heart failure (CHF). C-type natriuretic peptide (CNP), the third member of the natriuretic peptide family with vasodilating, natriuretic, and lusitropic actions, may prevent the deleterious effects of CNP, thus improving LV performance, both at rest and during Ex after CHF.

Methods: We tested the hypothesis that local HTG impairs resistance vessel endothelial function, by examining whether forearm blood flow (FBF) responses to intra-arterial ace-
ticholine (Ach), bradykinin (BK), and nitroprusside were altered by coinfusion of intralipid (IL 200 mg/min) for 90 min in 10 healthy adults, age 38±4, 7M.

Results: IL increased plasma triglycerides from 80±20 to 433±62 and free fatty acids from 0.5±0.1 to 1.9±0.2 (p<0.0003 for both). Forearm vasodilatation to Ach (p=0.93), BK (p=0.73), and nitroprusside (p=0.34) were not reduced by HTG (figure).

Conclusion: Local HTG sustained for >90 min does not impair resistance vessel endot-
hal function. These results suggest that resistance vessels are less susceptible to the deleterious effects of triglycerides than conduit vessels. Alternatively, the vascular effects of systemic HTG may be mediated indirectly, perhaps via hormonal or reflex mecha-
nisms.

Poster Presentation

1160-167 Defining the Acute Cardiorenal Response to High Dose Nesiritide in Severe Experimental Congestive Heart Failure

John A. Schinger, Alessandro Catalaottii, Hory Chen, Guido Boerrigter, Lisa C. Costello-Boerrigter, Fernando L. Martin, Gail J. Harty, John C. Burnett, Jr., Mayo Clinic, Rochester, MN

Background: Previous human studies report improved cardiac filling pressures and glo-
bal clinical status in acute decompensated congestive heart failure (CHF) using human brain natriuretic peptide (BNP)/Nesiritide. Severe CHF however may be associated with emergence of reduced sensitivity to BNP due to receptor down-regulation, enhanced degradation or reduced renal perfusion pressure. We investigated the effects of clinical and high dose IV Nesiritide on systemic hemodynamics, renal function, and sodium excretion in severe canine CHF, hypothesizing that supraclinical doses further reduce cardiac filling pressures and enhance sodium excretion without marked hypotension.

Methods: We used an established model of pacing induced (240 bpm, 10 days) severe canine (n=7) CHF characterized by decreased cardiac output, increased cardiac filling pressures, sodium retention and neurohumoral activation. After a baseline clearance IV Nesiritide was infused with a lead in period followed by 30 minute clearances at doses of 100 (approximating doses in clinical trials) and 1000 ng/kg/min (10 fold greater than clini-
tical trials).

Results: Clinical dosing decreased pulmonary capillary wedge pressure (PCWP) (23±1 vs 18±1 mmHg, p<0.05), pulmonary artery pressure (PAP) (31±3 vs 27±2 mmHg, p<0.05), and mean arterial pressure (MAP) (105±5 vs 87±4 mmHg, p<0.05), and tended to increase cardiac output (CO) (1.43±0.21 vs 1.91±0.39 L/min) vs baseline. Clinical dose IV Nesiritide further decreased PCWP (17±2 mmHg, p<0.05) and PAP (23±1 mmHg, p<0.05), without further decreasing MAP, and tended to further increase CO. Importantly, glomerular filtration rate tended to increase compared to baseline (28±5 L/min) with both clinical (41±15 L/min) and high (48±15 L/min) doses of IV Nesiritide. Urinary sodium excretion tended to increase at clinical dosing (7±4 µEq/min)and significantly increased (12±6 µEq/min, p<0.05) with high dose IV Nesiritide vs baseline (2± 0.4 µEq/min) in the absence of further decreases in MAP.

Conclusion: These studies in experimental CHF suggest it may be possible to increase the dosing of IV Nesiritide, thus warranting carefully designed future human investiga-
tions in severe CHF.

1160-168 The Estrogen-Induced Alterations in Arterial Stiffness are Independent of the Dipping Status but Attenuated by Progestrone in Hypertensive Postmenopausal Women

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Background: Large arterial stiffness and attenuated nocturnal blood pressure (BP) fall are associated with unfavorable cardiovascular outcome, in essential hypertension. We assessed the hypothesis that the addition of progestrone and the absence of normal cir-
cadian BP variation may modify the beneficial effects of hormonal replacement therapy (HRT) on large artery distensibility in hypertensive postmenopausal women.

Methods: For this purpose, we studied aortic stiffness in 56 postmenopausal women (aged 52 years, 3.4 years after menopause) with untreated, mild essential hypertension randomized to conjugated estrogen alone (n=20), estrogen plus medroxyprogesterone (n=20) or placebo (n=16). Aortic elasticity was evaluated, non-invasively, on the basis of pulse wave velocity (PWV) measurements at baseline and at 12 weeks after treatment. At baseline, women receiving conjugated estrogen alone, underwent 24h ambulatory BP monitoring and were classified to non-dippers (defined by a reduction in the night-time systolic and diastolic BP <10% from day values) (n=7) and dippers (the remaining sub-
jects) (n=49).

Results: In the entire study population office BP was 146±9 mmHg and left ventricular mass index was 104±26 gm2/m2. The patients’ groups were matched for age, time since menopause, smoking status, office blood pressure and PWV values at baseline. At 12 weeks of treatment, in women receiving estrogen alone, arterial PWV was reduced (6.51±6.09 m/sec, p<0.005), while in those receiving combined HRT or pla-
cbo, PWV did not change (6.32 vs 6.28 mmHg, p<0.005), while in those receiving combined HRT or placebo, PWV did not change (6.28 vs 6.30 m/sec, respectively, p=NS for both cases). Treatment with conjugated estrogen induced a significant reduction in aortic PWV in both groups of dippers and non-dippers, after 12 weeks. Furthermore, the degree of reduction in PWV did not differ in dippers (by 0.73 m/sec) and non-dippers (by 0.72 m/sec).