< 0.01) and RWT (0.34 vs 0.33 in men and 0.35 vs 0.34 in women, both p < 0.05). There was no difference in ejection fraction or end systolic wall stress. After adjusting for BP, BMI, age and height (HT), only PWT in women remained different between groups (p < 0.05). Linear regression analysis in all pts identified increased PWT, IVS and RWT as correlates of IGT (all p < 0.05) independent of BP, BMI, age, HT, heart rate and gender.

Conclusion: Differences in LV structure/function in American Indians with IGT are largely due to differences in BMI, age, HT and BP; however, statistically independent associations with LV wall thickness suggests an influence of IGT on LV geometry.

1236-96 The Cardiac and Hemodynamic Effects of Insulin on Non-diabetic American Indians: The Strong Heart Study

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Background: Insulin (I) has been proposed to have adverse cardiovascular effects, but this has not been studied in large populations.

Methods: We evaluated the association of I and echocardiographic left ventricular (LV) measurements in 1394 (46% men) non-diabetic American Indian participants in the Strong Heart Study (SHS).

Results: Significant (p < 0.05) relations were found in men and women between Log_{10}(I) and LV mass (r = 0.24 and 0.26 respectively), left atrial diameter (r = 0.25 and 0.28), posterior wall thickness (r = 0.20 and 0.20) septal thickness (IVS, r = 0.17 and 0.24), LV diastolic diameter (r = 0.17 and 0.16) and cardiac output (CO, r = 0.20 and 0.24); in men, meridional end systolic stress (ESS, r = 0.12) and in women relative wall thickness (RWT, r = 0.11) and peripheral resistance (TPR, r = −0.17). After adjustment for body mass index (BMI), age, height (HT) and systolic BP, the only significant correlates of Log_{10}(I) were higher CO in men, and higher ESS, RWT, IVS in women, all p < 0.05. In a subset of 163 apparently normal SHS participants (BMI <26, BP <140/90 and absence of diabetes, valvular disease, LV wall motion abnormality or use of BP medication), the 97th percentile for I was 25 μU/ml (men) and 23 μU/ml (women), and was used to separate subjects into groups of 93 men with I ≥25 μU/ml, 534 with I <25 μU/ml; and 161 women with I ≥23 μU/ml and 606 with I <23 μU/ml. After adjusting for age, BMI and HT, the only significant differences (p < 0.05) in LV structural/functional parameters were a higher CO (5.6 vs 4.8 l/min) and lower TPR (1499 vs 1675) in men with I ≥25 μU/ml as compared to I <25 μU/ml.

Conclusions: Positive relations between I and heart size in non-diabetic adults are largely due to effects of body size; similar to published experimental results, higher CO and lower TPR were independently related to higher I in men.

1236-97 Differences in Echocardiographic Findings and Systemic Hemodynamics Among Non-diabetic American Indians in Different Regions: The Strong Heart Study

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Background: Cardiovascular risk factors and morbidity rates in American Indians differ by region but it is unknown whether left ventricular (LV) and systemic hemodynamic findings differ in parallel.

Methods: We evaluated 290 non-diabetic Strong Heart Study (SHS) participants in Arizona, 595 in Oklahoma and 572 in North/South Dakota (ND/SD) by echocardiography.

Results: Subjects in the 3 regions were similar in age and gender but those in Arizona had the highest body mass indices and lowest heart rates and those in ND/SD had the lowest diastolic pressures. In multivariate analyses, ND/SD participants had larger aortic (Ao) anular, Ao root and LV chamber sizes and higher cardiac index and lower peripheral resistance; those in Arizona had increased LV relative wall thickness. LV mass/height$^{2.7}$ was highest and stress-corrected LV midwall shortening lowest in Arizona, possibly related to high proportional rates of cardiovascular death in Arizona Indians.

Conclusions: In non-diabetic SHS participants the known lower arterial pressure in Northern Plains Indians reflects lower peripheral resistance; arterial size is increased in ND/SD Indians; and abnormal LV wall thickness and contractility in Arizona Indians may contribute to a different pattern of cardiovascular events.

1237 Clinical Application of Positron Emission Tomography and Magnetic Resonance Imaging

Wednesday, April 1, 1998, 3:00 p.m.–5:00 p.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 3:00 p.m.–4:00 p.m.

1237-14 Sympathetic Denervation in Patients Following Acute Myocardial Infarction: Relationship to Myocardial Perfusion and Metabolism

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Cardiac sympathetic denervation has been described previously in pts with coronary artery disease, but there are only limited data correlating the extent of denervation with myocardial perfusion and glucose metabolism. PET imaging of myocardial perfusion (with nitrogen-13 ammonia, NH3), metabolism (with fluorine-18 fluorodeoxyglucose, FDG) and sympathetic innervation (with carbon-11 hydroxyephedrine, HED) was performed in 9 nondiabetic pts (5 male, 53 ± 12 yrs) 2.7 ± 1.5 weeks following a first myocardial infarction (MI). A semi-automated program was used to determine NH3 and FDG uptake and HED retention in 177 regions of interest encompassing the left ventricular (LV) myocardium. Perfusion, metabolism and innervation defects were defined as the percentage of LV with tracer uptake or retention > 2 SD below values obtained from a normal database. Five pts had an anterior wall MI and 4 an inferior wall MI; 4 pts underwent thrombolysis and 4 pts had PTCA prior to their PET scan. The extent of defects with each tracer was as follows (mean ± SD, range): NH3 11.5 ± 14.2% (0–46.2), FDG 32.3.4 ± 27.2% (3.9–86.9), and HED 20 ± 18.9 (0–55.3), p = 0.05 for HED vs NH3 and p = 0.02 for FDG vs HED. In 7/9 pts the HED (sympathetic) defects exceeded the NH3 (perfusion) defects. The FDG (metabolism) defects equaled or exceeded the HED defects in all pts.

Conclusions: This is the first report to describe the relationship between the overall extent of myocardial denervation in relationship to perfusion and glucose metabolism. Following acute myocardial infarction, the extent of sympathetic denervation and abnormal metabolism exceeds the extent of ischemic injury. Regions with reduced glucose metabolism frequently extend beyond the denervated territory.

1237-15 Contrast Kinetics on Magnetic Resonance Imaging Predict Microvascular Integrity and Myocardial Damage After Infarction

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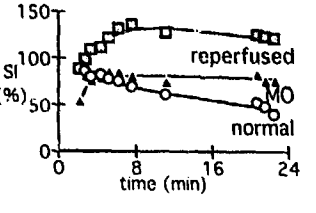
Background: Microvascular obstruction (MO) after acute myocardial infarction (MI) predicts poor clinical outcome. We examined whether a model of contrast kinetics by magnetic resonance imaging (MRI) can predict MO after MI.

Methods: We performed MRI studies with intravenous gadodiamide in 10 normal volunteers and 10 patients with documented acute MI. Normalized myocardial signal intensity (SI) over time (t) was modeled with a bi-exponential: $SI = A1\ e^{-K1t} + A2\ e^{-K2t}$, where K1 and K2 are early and late time constants respectively, and A1 and A2 are the relative magnitudes of the early and late phases of decay.

Results: Normal myocardium followed a biphasic decay pattern with time constants K1 = 0.711 ± 0.170 and K2 = 0.020 ± 0.007, and A1 and A2 both greater than 0. In regions remote to infarction, K1 and K2 had values similar to those f normal volunteers (p = 0.81). Infarcted but reperfused myocardium had severely impaired early contrast washout compared to normal myocardium, with broad delayed peaks (4.02 ± 0.72 vs 1.37 ± 0.22 min., p < 0.005) and negative values of A1 (−61 ± 33 vs 127 ± 24, p < 0.01). Peak SI of reperfused regions was increased 41 ± 13% compared to peak SI in normal myocardium (p < 0.05). Areas of MO present in 5 of 10 infarcts also had impaired early contrast washout with delayed peaks (5.72 ± 1.80



WEDNESDAY POSTER