step-wise Cox multivariate analyses.
Conclusions: Despite a higher prevalence in AA, ECG strain may not provide the same degree of risk stratification in AA as in non-AA patients.

Outcome According to Race and ECG Strain

Outcome Events	African Americans							Caucasians and Others						
	5-Year Event Rates			Multivariate Cox Analyses				5-Year Event Rates			Multivariate Cox Analyses			
	Strain Absent (n=372)	Strain Present (n=143)	p value	Chi-Square	p value	Hazard Ratio	95% CI	Strain Absent (n=7511)	Strain Present (n=828)	p value	Chi-Square	p value	Hazard Ratio	95% CI
Composite Endpoint	14.8	18.4	0.792	0.52	0.471	-----	---	11.0	21.4	<0.0001	11.73	0.001	1.39	1.15-1.68
Cardiovascular Mortality	8.4	7.7	0.998	0.34	0.558	-----	---	4.1	9.4	<0.0001	12.85	<0.001	1.69	1.28-2.23
Myocardial Infarction	2.4	8.9	0.035	4.86	0.027	3.05	1.13-8.22	4.0	8.0	<0.0001	6.07	0.014	1.47	1.08-1.99
Stroke	7.6	7.0	0.622	1.25	0.264	-----	---	5.7	10.7	<0.0001	6.30	0.012	1.41	1.08-1.85
All-Cause Mortality	14.4	18.3	0.530	0.09	0.759	-----	---	8.1	14.0	<0.0001	4.87	0.027	1.29	1.03-1.62

11:15 a.m.

888-4 **Angiotensin-Converting Enzyme Inhibitors Are Comparable to Angiotensin II Receptor Blockers in the Primary Prevention of Myocardial Infarction in Hypertensive Patients**

Jefferson T. Baer, William H. Sauer, Jesse A. Berlin, Stephen E. Kimmel, University of Pennsylvania, Philadelphia, PA

Background: There has recently been a growing interest in the relative merits of Angiotensin Converting Enzyme (ACE) Inhibitors versus Angiotensin II Receptor Blockers (ARBs) in the prevention of cardiovascular events. However, studies comparing the two are lacking, with no studies to our knowledge directly comparing outcomes in hypertensive patients. For this reason we sought to compare ACE Inhibitors with ARBs in the prevention of first nonfatal myocardial infarction (MI) among hypertensive patients.
Methods: The data were obtained from two case-control studies of first MI, ages 30 through 75, coming from 68 hospitals in an eight county region during a 6 year period. Cases were patients hospitalized with a first nonfatal MI, and controls were randomly selected from the same geographic area. Detailed information regarding cardiac risk factors and current medications was collected via telephone interviews. Patients with congestive heart failure were excluded. Multivariable logistic regression was used to adjust for age, sex, race, cigarette smoking, history of coronary disease, family history, diabetes mellitus, hyperlipidemia, body mass index, physical activity and duration of hypertension. Adjustment for socioeconomic and other clinical variables did not alter the findings.
Results: 386 cases (20.7% were ARB users) and 969 controls (28.0% were ARB users) were interviewed. There was an inverse association between ARB use and MI in the unadjusted model with an odds ratio (OR) of 0.67 and a 95% confidence interval (CI) of 0.51-0.89. However, after adjusting for confounding, there was no difference in risk of MI between ACE Inhibitor and ARB users (OR 1.0, 95% CI: 0.69-1.59). There were no subgroups of patients taking ACE Inhibitors that were at increased risk of MI when compared to ARB users.
Conclusions: Our investigation shows that ARBs and ACE Inhibitors appear to have comparable effects on the risk of MI when used to treat hypertensive patients without known heart failure.

11:30 a.m.

888-5 **Effect of Cardiovascular Disease Risk Factors on Ambulatory Blood Pressure Profile in Obesity**

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BACKGROUND: Ambulatory blood pressure (ABP) monitoring has been linked to organ damage and cardiovascular events. We examined the effect of different cardiovascular disease (CVD) risk factors on ABP in obesity.
METHODS: We studied 215 obese African Americans (88% female, with average BMI of 43 kg/m^2) who enrolled in a diet-exercise program of weight reduction at Howard University General Clinical Research Center. Dyslipidemia was present in 36% of the patients, hypertension in 66% and diabetes mellitus in 24%. We classified the patients as: normotensives with no other risk factor, (Group I), normotensives with other risk factor (Group II), hypertensives with no other risk factor (Group III) and hypertensives with other risk factor (Group IV). Blood pressure and heart rate were obtained using an oscillometric automatic recorder set to take readings every 30 min for 24 hrs. Pulse pressure (PP) is the difference of systolic BP (SBP) and diastolic BP (DBP). Daytime BP was defined as the average readings from 6AM -10PM and nighttime, from 10PM - 6AM. Nondippers exhibited a nocturnal fall in mean blood pressure (MBP) of less than 10%. The average BP and heart rate as well as the rate of nondipping were compared.
RESULTS: Results are tabulated. While there was a progressive increase in ABP and PP from Group I to Group IV, HR and nondipping rate did not vary significantly.
CONCLUSION: In this obese population, the other CVD risk factors did not have additional effect on nondipping, a marker of target organ damage and CV events.

Comparison of Ambulatory Blood Pressure and Heart Rate

Characteristic	Group I (n=36)	Group II (n=41)	Group III (n=71)	Group IV (n=67)
SBP/DBP/MBP (mmHg)				
24- hour	122/74/90	125/76/92	132/81/98	133/80/98
Daytime	123/76/92	126/79/95	134/84/101	135/84/101
Nighttime	119/69/86	123/71/89	127/76/93	130/76/94
Nondipping rate	0.64	0.61	0.57	0.61
PP(mmHg)/HR(bpm)				
24-hours	48/84	49/83	50/78	53/81
Daytime	47/86	47/86	50/80	52/83
Nighttime	50/79	52/79	51/77	54/76

11:45 a.m.

888-6 **Differential Effects of Selective COX-2 Inhibitors on Endothelial Function in Salt-induced Hypertension**

Matthias Hermann, Giovanni Camici, Jens P. Hellermann, Joachim Thiery, Steffen Gay, Thomas F. Luscher, Frank Ruschitzka, University Hospital Zurich, Zurich, Switzerland

Background: In view of the ongoing controversy of potential differences in cardiovascular safety of selective COX-2 inhibitors (coxibs), we compared the effects of two different coxibs and a traditional NSAID on endothelial dysfunction, a well established surrogate of cardiovascular disease, in salt-induced hypertension.
Methods and Results: Salt-sensitive (DS) and salt-resistant (DR) Dahl rats were treated with a high-sodium diet (4% NaCl) for 56 days. From days 35 to 56, diclofenac (6 mg/kg/d; DS-NaCl-dic), rofecoxib (2 mg/kg/d; DS-NaCl-rof), celecoxib (25 mg/kg/d; DS-NaCl-cel) or placebo (DS-NaCl-pla) were added to the chow. Systolic blood pressure, eNOS and protein expression, plasma levels of prostaglandins, isoprostanes and inflammatory cytokines were studied and vascular reactivity of aortic rings was assessed. Blood pressure increased with sodium diet in the DS-groups which was more pronounced after diclofenac and rofecoxib treatment ($p<0.005$ vs DS-NaCl-pla), but slightly blunted by celecoxib ($p<0.001$ vs DS-NaCl-pla). Sodium diet reduced NO-mediated endothelium-dependent relaxations to acetylcholine (ACh, 10^{-10}-10^{-5} mol/L) in untreated hypertensive rats ($p<0.0001$ vs DR-NaCl-pla). Relaxation to ACh improved after celecoxib ($p<0.005$ vs DS-NaCl-pla and DS-NaCl-rof), but remained unchanged after rofecoxib and diclofenac treatment. Vasoconstriction after NOS inhibition with L-NAME (10^{-4} mol/L) was blunted in DS rats ($p<0.05$ vs DR-NaCl-pla), normalized by celecoxib, but not affected by rofecoxib or diclofenac. Protein expression of eNOS was decreased in DS rats with a trend for increased eNOS levels in the DS-NaCl-cel group (97.8±25.6 vs 54.8±2.8 %. Indicators of oxidative stress, 8-isoprostane levels, were elevated in untreated DS rats on 4% NaCl (6.55±0.58 vs 3.65±1.05 ng/ml, $p<0.05$) and normalized by celecoxib only (4.29±0.58 ng/ml). Plasma levels of prostaglandins did not change during sodium diet or any treatment.
Conclusion: These data show that celecoxib, but not rofecoxib or diclofenac, improves endothelial dysfunction and reduces oxidative stress, thus pointing to differential effects of coxibs in salt-sensitive hypertension.