to 112+/4 mmHg (p<0.0001) while MSNA decreased to 28+/-5% of baseline values (p<0.0001) and HR decreased from 71+/-3 to 61+/-3 bpm (p<0.0001). The largest increases in BP were accompanied by the most marked decreases in MSNA (r=-0.79, p=0.003) and HR (r=-0.49; p=0.01) during the first 5 seconds of the AVF occlusion. During AVF occlusion baseline CO of 6.9+/-0.3 decreased to 5.6+/-0.3 l/min (p<0.0001) while baroreflex sensitivity increased from 10+/-1 to 17+/-2 ms/mmHg (p<0.001).
Conclusions: Arterial baroreceptor activation and increased arterial baroreflex sensitivity decrease heart rate during AVF occlusion. In addition our study is the first to demonstrate that arterial baroreflex activation decreases sympathetic nerve traffic during the Nicoladoni-Branham sign.

1009-197 Preeclampsia: An Under-Recognized Risk Factor for Hypertension Later in Life

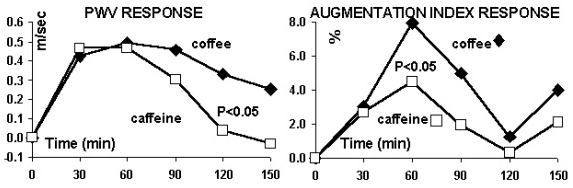
Ahmad A. Elesber, Stephen T. Turner, Kenneth P. Offord, Vesna D. Garovic, Mayo Clinic, Rochester, MN

Background: Preeclampsia is a pregnancy-specific disorder that occurs in 3-5% of pregnancies. It is a multisystem disease characterized by hypertension (blood pressure ≥140/90 mmHg) and proteinuria (protein level ≥ 300mg/24-hour urine). It remains unclear if preeclampsia is associated with an increased risk for cardiovascular disease later in life. The aim of this study was to assess the frequency of subsequent hypertension in a group of women who were diagnosed with preeclampsia, eclampsia, or toxemia more than 20 years ago. Methods: We reviewed Rochester Mayo Clinic medical records between 1960-1979 and mailed hypertension questionnaires to 144 women who were diagnosed with any of these 3 conditions. Our control group consisted of 154 women who did not have a history of preeclampsia. We received 103 completed questionnaires for the cases group (response rate 71.5%) and 96 for the control group (response rate 62%). Results: There were no significant differences in age, race, education, parity, and time interval from index pregnancy to survey completion date between cases and controls. Women with histories of preeclampsia reported a higher frequency of hypertension compared to women with normal pregnancies: 52/103 (50%) vs. 23/96 (24%), respectively (p<0.0001). Conclusion: History of preeclampsia, eclampsia, or toxemia is associated with an increased risk for hypertension later in life. The list of risk factors common for both atherosclerosis and preeclampsia is extensive, including obesity, endothelial dysfunction and oxidative stress. The association between preeclampsia and atherosclerotic disease later in life remains to be determined.

1009-198 Coffee Has a More Potent Effect on Arterial Stiffness Than Caffeine

Charalambos Vlachopoulos, Nikolaos Ioakeimidis, Nikolaos Alexopoulos, Konstantinos Aznaouridis, Foteini Kosmopoulou, Dimitris Tousoulis, Christos Pitsavos, Christodoulos Stefanadis, Athens Medical School, Athens, Greece

Background: Large arteries stiffness and arterial wave reflection are prognosticators of cardiovascular risk and are involved in the pathogenesis of hypertension. Whether there is a differential effect of coffee and caffeine on arterial function has not been defined.
Methods: We studied 10 healthy volunteers (33±11 yrs) in a randomized, sham-procedure controlled, crossover fashion on 3 separate occasions receiving: a) a triple espresso b) 240mg of caffeine alone (= amount contained in a triple espresso) and c) placebo. Carotid-femoral pulse wave velocity (PWV) was measured as an index of aortic stiffness using an automated, non-invasive device (Complior®) and augmentation index (AIx) as a measure of wave reflection using a validated system (Sphygmocor®). **Results:** Both coffee and caffeine increased PWV and AIx indicating increased aortic stiffness and wave reflection, however, the effect of coffee was more pronounced (figure). Pressures also increased, however, the increases were less discernible between coffee and caffeine (e.g. aortic systolic increased by 10.8 and 8.8 mmHg respectively).
Conclusions: Both coffee and caffeine increase aortic stiffness and wave reflection, however, the effect of coffee is more potent. These findings are particularly important for the precise evaluation of their consequences on the cardiovascular system. Furthermore, they indicate that the terms "coffee" and "caffeine" should not be used indistinguishably in the design and interpretation of studies.



POSTER SESSION

1027

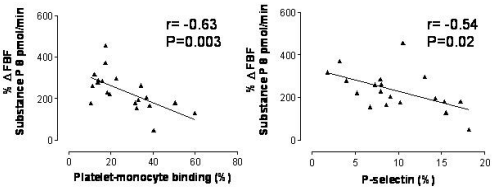
Mechanisms and Implication of Vascular Dysfunction

Sunday, March 07, 2004, Noon-2:00 p.m.
Morial Convention Center, Hall G
Presentation Hour: 1:00 p.m.-2:00 p.m.

1027-183 Functional Interplay Between Endothelial Dysfunction and Platelet Activation in Patients With Stable Coronary Heart Disease

Simon D. Robinson, Scott A. Harding, Paula Cummins, Jaydeep Sarma, I. Davidson, Keith A. Fox, Nicholas A. Boon, David E. Newby, University of Edinburgh, Edinburgh, United Kingdom, Lothian University Hospital NHS Trust, Edinburgh, United Kingdom

Background: Impaired endothelium dependent vasodilatation is an independent predictor of acute atherothrombotic events although the biological basis for this is uncertain. Endothelium-derived nitric oxide and prostacyclin strongly inhibit platelet activation. Platelet-monocyte binding (PMB) and platelet surface expression of P-selectin are sensitive markers of platelet activation. We determined the relationship between endothelium dependent vaso-relaxation and platelet activation in patients with stable coronary heart disease (CHD).
Methods: Twenty male subjects with angiographically confirmed CHD were recruited. All subjects were receiving aspirin and had stable symptoms. PMB and platelet surface expression of P-selectin were assessed using 2-colour flow cytometry on whole blood. Forearm blood flow (FBF) was assessed using venous occlusion plethysmography during intra-arterial infusions of the endothelium dependent vasodilator substance P (2-8 pmol/min) and the endothelium independent vasodilator sodium nitroprusside (2-8 mcg/min).
Results: PMB and P-selectin expression were strongly correlated with maximal endothelium dependent, but not endothelium independent, vasodilatation (figure 1).
Conclusions: In patients with CHD there is a strong inverse relationship between endothelium dependent vasomotor function and platelet activation. This suggests a direct mechanism whereby endothelial dysfunction increases the risk of acute atherothrombotic events.



1027-186 High Angiotensin II Responsiveness Relates to Decreased Endothelium Dependent Relaxation in Human Arteries

Peter Paul van Geel, Yigal M. Pinto, Hendrik Buikema, Adriaan A. Voors, Margreeth Oosterga, Dirk Jan van Veldhuisen, Wiek H. van Gilst, University of Groningen, Groningen, The Netherlands, University Hospital Groningen, Groningen, The Netherlands

Background: We investigated the relation between Angiotensin II (Ang II) responsiveness and endothelium dependent relaxation (EDR) in isolated human arteries. Angiotensin converting enzyme (ACE) inhibition, which is known to modulate Ang II responsiveness, was studied in this relation.
Methods: Hundred eighty-seven patients, undergoing coronary bypass surgery, were randomized to receive an ACE-inhibitor or placebo, one week prior to surgery. Segments of the internal mammary artery (IMA) were exposed in organ bath experiments to metacholine (ME; 10 nmol/l -0.1 mmol/l) after precontraction with phenylephrine (PE; 10 µmol/l). After washing and renewed stabilization, the rings were preincubated with NG – monomethyl-L-arginine and exposed to increasing concentrations of Ang II (0.1 nmol/l – 1µmol/l), followed by a control response to PE (10 µmol/l).
Results: There was a significant inverse relation between maximal Ang II (%PE) contraction and maximal ME (%PE) relaxation. Patients with the highest contraction to Ang II showed the lowest ME relaxation (r=0.312; p=0.003). ACE-inhibition significantly increased Ang II sensitivity (p=0.03). This increase was accompanied by a tendency toward decreased EDR (p=0.07).
Conclusion: High Ang II responsiveness inversely correlates to EDR in IMA's of patients with established coronary artery disease. ACE-inhibition, which in itself increases the response to Ang II, had an adverse effect on EDR. These findings suggest that any type of increased Ang II responsiveness may adversely affect endothelial function.