The HOPE trial) and high CRP was defined >0.20 mg/dL. Metabolic syndrome is defined traditionally as the clinical risk negatively influencing endothelial function.

Claudia Borghi, Ada Dormi, Antonio Gaddi, Ettore Ambrosini, St. Orsola-Malpighi Hospital, Bologna, Italy

Background. Hypertension (HTN) and high serum cholesterol (HC) level are often combined in the same subject where they contribute to the overall cardiovascular risk profile. HC is associated with an impaired vascular function and an overexpression of vascular angiotensin II receptors, which can contribute to the development of HTN. Aim of the present study was to investigate the role of HC, if any, in the development of HTN in the Bologna Heart Study.

Methods. 1820 normotensive subjects (50/50% female) enrolled in the Bologna Heart Study in 1972 have been divided according to total serum cholesterol (T-Chol) tertiles and followed for 12 years to evaluate the proportion of patients developing HTN. T-Chol was defined as SBP and/or DBP > 140/90 mmHg and/or therapy for HTN.

Results. After adjustment for the main confounding factors (family history of HTN, age, BMI, sex, diabetes) the rate of HTN was significantly increased in subjects with T-Chol > 239 mg/dl both after 8 (1980) and 12 (1984) years of follow-up (>0.001 vs. other subgroups). The rate of development of HTN was enhanced in the two older subgroups of subjects (50-59 and >59 years).

Conclusion. These data suggest that HC could substantially contribute to the development of HTN and strongly support the role of lipid lowering drugs and particular statins in the primary prevention of cardiovascular disease.

1138-164 Chronic Inflammation Add to Metabolic Syndrome in the Prediction of Microalbuminuria
Adrian W. Messerli, Gregory L. Pearce, Byron Hoogwerf, Dennis L. Sprecher, The Cleveland Clinic Foundation, Cleveland, OH

Background. We have demonstrated that chronic inflammation, represented by c-reactive protein (CRP), predicts the presence of endothelial dysfunction, manifest as urinary albumin excretion. We hypothesize that the association of CRP with urine albumin is predominantly mediated through its correlation with the metabolic syndrome. Methods: Serum CRP and urine albumin/creatinine ratios (ACR) from 764 patients were analyzed to estimate age and gender adjusted relative risk of high CRP and metabolic syndrome for high ACR. High ACR was defined as >14.4 units (approximating the upper quartiles of the HOPE trial) and high CRP was defined as >0.20 mg/dL. Metabolic syndrome is defined according to ATP III guidelines. Results. High ACR rates were 11% (50/757) when neither high CRP nor metabolic syndrome were present, 23% (57/243) when isolated high CRP was present, 23% (50/207) when isolated metabolic syndrome was present and 35% (62/179) when concurrent high CRP and metabolic syndrome were present. The odds ratio of 4.2 when both factors were present was significantly higher than either isolated high CRP (p=0.01) or isolated metabolic syndrome (p=0.02). (See tabe) Conclusion. The combined presence of elevated CRP and metabolic syndrome predicts a 4-fold increase in the occurrence of elevated urinary albumin loss. This is consistent with both inflammation and traditional metabolic clinical risk negatively influencing endothelial function.

ORAL CONTRIBUTIONS
828FO Featured Oral Session...Natriuretic Peptides: What Are They Telling Us?
Monday, March 31, 2003, 4:00 p.m.-5:30 p.m.
McCormick Place, Room S105

4:15 p.m.

828FO-2 Plasma Neurohormones as Markers of Right Ventricular Overload and Predictors of Mortality in Acute Pulmonary Embolism
Igor I. Tulevski, Marye Ian Woelcke, Jasper W. M. Mulder, Dirk J. van Veldhuisen, Ernest E. van der Wall, Harry Butler, Barbara J. M. Mulder, Academic Medical Center, Amsterdam, The Netherlands

Background. Right ventricular (RV) function is of major prognostic significance in patients with acute pulmonary embolism (PE). We studied the role of plasma neurohormones Brain Natriuretic Peptide (BNP) and Atrial Natriuretic Peptide (ANP), and their potential to predict mortality, in patients with acute PE. Methods: Plasma neurohormone levels were measured in 114 patients (age 58±17) with acute PE within an hour of pre-sentation after confirmed diagnosis by lung angiography and/or scintigraphy. Twenty seven healthy age-matched volunteers served as controls (age 42±12). Renal impairment, arrhythmias, pre-existing RV pathologic, and left ventricular dysfunction were exclusion criteria. In the patient group, survival was considered for a period of 30 days. Results: Plasma BNP and ANP levels were significantly higher in patients with acute PE than in controls (BNP 34.4±9.1 pg/ml versus 3.1±3.7 pg/ml p<0.01, and ANP 34.1±9.3 pg/ml versus 6.0±12.3 pg/ml p<0.001). The odds ratio of 4.2 (95% CI 2.5-6.9) for later events including 15 restenoses, 11 myocardial infarctions, 6 strokes and 23 deaths was significantly higher in patients who died within 30 days, compared to 108 patients who survived (BNP 129.5±139.1 pg/ml versus 21.5±51.1 pg/ml p<0.001, and ANP 241±45.9 pg/ml versus 10.6±12.3 pg/ml p<0.001).

Conclusion: Neurohormone secretion is increased during acute PE in response to right ventricular overload. There was a significant relationship between presenting BNP and ANP levels, and 30-day mortality in patients with acute PE. Plasma neurohormone levels may complement the diagnosis, treatment and in-hospital prognosis of patients with acute RV pressure overload such as seen in acute PE.

4:30 p.m.

828FO-3 Pro-Brain Natriuretic Peptide Predicts Severe Cardiovascular Events in Patients With Heart Disease
Thomas Weber, Johann Auer, Elisabeth Lasang, Martin Strobl, Robert Bleifeld, Bemd Eder. General Hospital of the Kreuzschwestern, Wels, Austria

Background: The value of brain natriuretic peptide (BNP) in the diagnosis of heart failure is well known. Moreover, BNP levels have prognostic significance after myocardial infarction and in heart failure. We tested the prognostic impact of BNP in a variety of different heart diseases.

Methods: We prospectively measured blood levels of n-terminal pro-BNP, using an immunassays (Elicys pro BNP test, Roche diagnostic), in patients undergoing cardic catheterization in our laboratory for routine evaluation of heart failure. Follow-up data were collected at hospital discharge and after 3 months. 68 out of a total of 1710 patients suffered from cardiac events (event group). As a control group served 1050 patients with an uneventful course. Both groups were balanced with respect of age, sex, lipids, hypertension, diabetes, creatinine, smoking status, diagnosis, extent of corony artery disease, left ventricular function, percutaneous interventions ( percentage balloon and stent diameters, multivessel interventions ) and medication use. For statistics we used Mann-Whitney U test and t-test, as appropriate. BNP values are given as mean ± standard deviation.

Results: The main diagnosis in both groups was coronary heart disease (79 ± 80%), fewer patients had valvular disease (15 vs 14 % ) or non-cardiac chest pain ( 6 % both groups ). Percutaneous interventions were performed in 45 ( control group ) vs 50 ( event group ) of the patients. 18 patients had events during the first hospitalization ( includig pericardial infarction and 5 deaths ). After the event, 15 restenoses, 11 myocardial infarctions, 6 strokes and 23 deaths ) BNP levels were higher in the event group than in the control group ( 298 ± 730 vs 1021 ± 1391 pg/ml, p<0.01 ). BNP tended to be higher in patients with early events than with later events ( 5072 ± 11117 vs 1154 ± 1447 pg/ml, p<0.006).

Conclusion: Pro-BNP is an independent prognostic marker in a broad spectrum of patients with ischemic and valvular heart disease.

4:45 p.m.

828FO-4 What Causes Elevated B-Type Natriuretic Peptide in Patients Without Heart Failure?
Peter A. McCullough, Philippe Gabriel Steg, Marie-Claude Aumont, Philippe Duc, Torgny Oenblad, Charlotte, K. Weinrodt, Richard M. Nisenwalt, James McCool, Judd F. Hollandar, Arne Westheim, Alan S. Storrow, William T. Abraham, Sumant Lamba, Alan H. Wu, Alan S. Maisel. BNP Multinational Study Investigators, William Beaumont Hospital, Royal Oak, MI, University of California, San Diego School of Medicine, Veterans Affairs Medical Center, San Diego, CA

Introduction: BNP is a peptide that is released from ventricular myocardium in response to stretch. In the setting of acute myocardial ischemia or injury, BNP levels can rise in the absence of structural heart disease. This phenomenon is known to be due to vascular remodeling and diastolic dysfunction. In patients presenting to the emergency department (ED) with acute dyspnea, left ventricular (LV) systolic and diastolic function are associated with increased LV end systolic volume. Widespread use of BNP after its approval as a diagnostic aid in CHF, and as a prognostic indicator in acute coronary syndromes, has raised the issue of what other disease processes can cause a markedly elevated BNP?

Methods: The BNP Multinational Study was a seven-center prospective study of 1856 patients presenting to the ED with acute dyspnea and who had blinded BNP levels measured with a rapid, point-of-care device on arrival. The reference standard for CHF was adjudicated by two independent cardiologists, also blinded to BNP results, who reviewed all clinical data and standard CHF scores.

Results: A total of 717 subjects (mean age 57.6 years. 54.3% male) had no history of CHF or LV dysfunction and were confirmed two cardiology to have HV. Of these, 87717 (5.6%) and 15717 (2.1%) were found to have BNP values >500 and >1000 pg/ml, respectively. Of these, 38717 (9.2%) and 5817 (5.8%) had a history of ischemic heart disease, 12.5% with ischemic changes by electrocardiogram, 42.4% had a history of asthma or chronic obstructive pulmonary disease (COPD), and 1.0% with cor pulmonale. Physical exam findings included: elevated jugular venous pressure in 16%, hepatic congestion in 17.5%, and peripheral edema in 50.0%. Chest x-rays were abnormal in 52.2% and oxygen saturations were 90% in 14.8% of subjects. The most common final diagnoses were: asthma or COPD with right ventricular strain, pneumonia, pulmonary embolism, anemia, cardiac ischemia, and sepsis. Chronic kidney disease