TIME COURSE AND MECHANISMS OF MENTAL STRESS-INDUCED CHANGES AND THEIR RECOVERY: HEMATOCRIT, COLLOID OSMOTIC PRESSURE, WHOLE BLOOD VISCOSITY, COAGULATION TIMES, AND HEMODYNAMIC ACTIVITY.

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The mechanisms for stress-induced changes in hematocrit and blood viscosity are unclear. Twenty-two males completed experimental (30 min baseline, 10 min mental stress, 30 min recovery) and no-stress control conditions (70 min). Hemostatic and hemodynamic activity were measured throughout. Hematocrit, colloid osmotic pressure, and blood viscosity displayed parallel patterns: a progressive increase with stress, followed by a gradual recovery. Correlational and covariance analyses indicated that the increase in hematocrit may be mediated by arterial pressure whereas recovery may be mediated by colloid osmotic pressure. Analyses also indicated that acute changes in blood viscosity may depend on hematocrit. These data suggest that stress disturbs hematocrit, colloid osmotic pressure, and blood viscosity through arterial pressure. Poststress, elevated colloid osmotic pressure may drive its own recovery and that of hematocrit and blood viscosity. (**Psychophysiology. 2007; 44:639-649).**

IS RESUSCITATION FROM HEMORRHAGIC SHOCK LIMITED BY BLOOD OXYGEN-CARRYING CAPACITY OR BLOOD VISCOSITY?

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Systemic and microvascular hemodynamic responses to volume restoration from hemorrhagic shock were studied in the hamster window chamber model to determine the significance of blood rheological and oxygen transport properties. Moderated hemorrhage was induced by means of arterial controlled bleeding of 50% of the blood volume. The hypovolemic shock state was maintained for 1 h before resuscitation. The animals were resuscitated by infusion of 25% of blood volume using either fresh plasma or blood and were studied for 90 min. Transfusion was performed with either oxygen-carrying fresh red blood cells (RBCs) or non-oxygen-carrying RBCs whose hemoglobin was converted to methemoglobin (MetHb). Systemic parameters, including cardiac output, vital organ blood flow distribution, microvascular hemodynamics, and capillary perfusion (functional capillary density [FCD]), were measured during the resuscitation period.

Fluorescent-labeled microspheres were used to measure organ blood flow (brain, heart, kidney, liver, lung, spleen, and window chamber). The blood viscosities at the end of the 90-min period were 2.4 cP after resuscitation with plasma, and 2.9 to 3.0 cP after blood transfusion (baseline, 4.2 cP). Resuscitation with RBCs with or without oxygen-carrying capacity had greater mean arterial pressure than did the plasma resuscitation group. The FCD was substantially higher for RBC transfusions (0.56% ± 7% of baseline) compared with plasma (46% ± 7% of baseline), and the presence of MetHb in the fresh RBC did not change the FCD or the microvascular hemodynamics. Oxygen delivery and extraction levels were significantly lower for resuscitation with plasma and MetHb-loaded RBCs compared with oxygen-carrying RBCs. The curtailed recovery of systemic and microvascular conditions after volume restitution with plasma seems to be due to the decrease in blood viscosity. Conversely, the restoration of blood rheological properties improves resuscitation independently of the restitution of oxygen-carrying capacity. **(Shock 2007; 27:380-389).**

THE RELATIVE EFFECTS OF ARTERIAL CURVATURE AND LUMEN DIAMETER ON WALL SHEAR STRESS DISTRIBUTIONS IN HUMAN RIGHT CORONARY ARTERIES

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This study looks at blood flow in four different human right coronary arteries (RCAs), which have been reconstructed from bi-plane angiograms. A generalized power-law model of blood viscosity is used to study the blood flow at a particular point in the cardiac cycle. Large differences are found in the wall shear stress magnitude (WSS) distributions in the four arteries, leading to the conclusion that it is not possible to make generalizations based on the study of a single artery. The pattern of WSS is found to be related to the geometry of a particular artery, that is, lumen diameter and arterial curvature as well as a combination of these two factors. There is a strong correlation between WSS and reciprocal radius and a weaker correlation between high curvature and extremes of WSS, with high WSS on the 'inside' of a bend and low WSS on the 'outside' of a bend. This is in contrast to the situation for a simple curved tube with constant radius where the inverse is observed. For each artery, a region proximal to the acute margin is identified where low WSS is found and where WSS is lower on the 'inner' wall of the RCA than on the 'outer' wall. This region is one where several studies have found that the human RCA preferentially exhibits atherogenesis. **(Phys Med Biol 2007; 52:2531-2544).**

RELATIVE VALUE OF INFLAMMATORY, HEMOSTATIC, AND RHEOLOGICAL FACTORS FOR INCIDENT MYOCARDIAL INFARCTION AND STROKE: THE EDINBURGH ARTERY STUDY.

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Background: The aim of our present study was to compare the association of a wide range of 17 biomarkers of inflammation, hemostasis, and blood rheology with incident heart disease and stroke after accounting for an indicator of subclinical atherosclerotic disease and traditional risk factors and also to determine their incremental predictive ability. **Methods and results:** We used data from the Edinburgh Artery Study, a population cohort study started in 1987 that comprised 1592 men and women aged 55 to 74 years. Subjects were followed for a mean of 17 years, and 416 of them suffered at least 1 cardiovascular event. In analyses adjusted for cardiovascular risk factors and history of cardiovascular disease (CVD): C-reactive protein, interleukin-6, fibrinogen, fibrin D--dimer, tissue plasminogen activator (t--PA), leukocyte elastase, and lipoprotein(a) (all P<0.01), as well as von Willebrand factor and plasma viscosity (both P<0.05), had significant hazard ratios for incident CVD. Further adjustment for a measure of subclinical atherosclerosis (ankle brachial index) had little impact on these associations. The hazard ratios (95% CI) for incident CVD between top and bottom tertiles in the latter analysis were 1.78 (1.30 to 2.45) for C-reactive protein, 1.85 (1.33 to 2.58) for interleukin-6, and 1.76 (1.35 to 2.31) for fibrinogen. Single biomarkers provided little additional discrimination of incident CVD to that obtained from cardiovascular risk factors and the ankle brachial index. An incremental score of multiple markers [interleukin-6, t-PA, intercellular adhesion molecule 1, and lipoprotein(a)] provided some added discrimination. **Conclusions:** Several "novel" risk factors predicted CVD after adjustments for conventional risk factors and also for a measure of asymptomatic disease. However, their incremental predictive ability was modest and their clinical utility remains uncertain. **(Circulation 2007; 115:2119-2127).**

WALL SHEAR STRESS: THEORETICAL CONSIDERATIONS AND METHODS OF MEASUREMENT.

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In arterial blood flow, the wall shear stress expresses the force per unit area exerted by the wall on the fluid in a direction on the local tangent plane. There is substantial evidence that the wall shear stress in-

duced by the pulsatile blood flow in the arterial system affects the atherogenic process. It is now widely accepted that the vessel segments that appear to be at the highest risk for development of atherosclerosis are those with low wall shear stress or oscillating wall shear stress. The purpose of this article is to define wall shear stress, to introduce relevant concepts of fluid mechanics to non-experts, and to critically review the various methods that have been used for the assessment of wall shear stress in animal and human blood circulation, paying special attention to the case of coronary arteries. **(Prog Cardiovasc Dis 2007; 49:307-329).**

MILK ENRICHED WITH PHYTOSTEROLS REDUCES PLASMA CHOLESTEROL LEVELS IN HEALTHY AND HYPERCHOLESTEROLEMIC SUBJECTS

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The consumption of plant sterols has been shown to decrease plasma concentrations of cholesterol without adverse effects in human subjects. To evaluate if milk would be a good vehicle for phytosterols to lower plasma levels of cholesterol, we performed a randomized blind study with healthy subjects (n = 22) and hypercholesterolemic patients (n = 19), both groups treated with phytosterol-enriched milk (2 g/d). Another hypercholesterolemic group (n = 15) was used as a control group. Lipid profile and biochemical, hematologic, and hemorheological parameters were determined at the beginning and after 15 and 30 days of milk beverage intake. After 15 days of treatment, healthy individuals showed lowered total cholesterol and LDL-C levels, by 8.31% ($P = .05$) and 11% ($P < .05$), respectively. After 30 days of the trial, these values did not change significantly. Hypercholesterolemic patients treated with phytosterol-enriched milk demonstrated significantly diminished levels of total cholesterol and LDL-C concentrations, by 9.62% ($P < .05$) and 12.20 % ($P < .05$), respectively. After 30 days, an increase in the total cholesterol and LDL-C levels was observed for hypercholesterolemic subjects, 6.69% ($P < .05$) and 8.68% ($P < .05$), respectively. In the hypercholesterolemic control subjects, no difference was found between plasma levels of total cholesterol, high-density lipoprotein cholesterol, and LDL-C. Only healthy subjects showed significant changes during the intake of phytosterol-enriched milk. The results obtained indicate that phytosterol-enriched milk is a good vehicle for reducing plasma cholesterol in hypercholesterolemic subjects. **(Nutrition Research 2007; 27:200-205).**

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