Time
16.00
cont.

0833 THE RELATIONSHIP OF BLOOD VISCOSITY TO CORONARY ARTERY DISEASE.

G.D.O. Lowe*, M.M. Drummond, A.R. Lorimer, I. Hutton, C.D. Forbes, C.R.M. Prentice, and J.C. Barbenel, University Departments of Medicine and Cardiology, Glasgow Royal Infirmary, and Bioengineering Unit, University of Strathclyde, Glasgow.

Increases in blood viscosity and its determinants (haematocrit and plasma fibrinogen) have been described in groups of subjects with clinical manifestations of arterial disease (myocardial infarction, angina, claudication). Viscosity factors may also be risk associations in prospective studies. We have studied the relationship of blood viscosity (shear rate $100s^{-1}$), haematocrit and fibrinogen to the extent of coronary artery occlusion in 50 males, aged 30-55, prior to coronary arteriography for chest pain. 26 subjects had significant occlusion (>50 per cent stenosis) of 2 or 3 major coronary arteries: 24 had single vessel disease or normal coronary arteries. 25 healthy controls were also studied. There were no significant differences in age or smoking habits between the three groups: use of beta-adrenergic blockers and plasma lipid levels were comparable in the two arteriography groups. Patients with extensive coronary artery disease had increased levels of viscosity and haematocrit ($p<0.005$), fibrinogen ($p<0.02$) and viscosity corrected for haematocrit ($p<0.05$), compared to angiographic or asymptomatic controls.

0834 MICRORHEOLOGY OF NORMAL AND PLATELET AGGREGATE-CONTAINING BLOOD

K.U.Benner, Anatom. Institute, Technical University, D-8000 Munich 40, FRG.

Preformed arterio-venous (a-v)-shunts are assumed to be the normal pathway for the non-nutritive portion of total blood flow passing different organs. Partial or total blockage of minute vessels can result in a redistribution of flow towards these a-v-channels. There is, however, a substantial area of circulation, where the existence of a-v-shunts sensu strictiori has been denied (e.g. in the skeletal muscle). Therefore, experiments were designed to study microrheological effects of ADP-induced platelet aggregates (or latex globules, Ø 25 µm) on isolated and autoperfused canine gastrocnemius muscles. During volume-constant perfusion of the maximally vasodilated, resting muscle the capillary transport coefficients (PxS) of 4-amino-antipyrine (indicator diffusion method; CRONE, 1963) decreased from 138.2 ± 25.9 to 21.3 $ml \times min^{-1} \times 100g^{-1}$ upon microembolization. During pressure-constant perfusion the aggregates produced likewise a decrease of PxS in the working muscle associated with an increase of flow resistance and a decrease of O_2-uptake and muscle performance. It is concluded, that the capillary exchange surface is reduced by aggregate-emboli decreasing the worcapacity of the muscles, and due to the microembolization some fraction of muscle blood flow may be non-nutritional.

0835 RELEASE OF ADP FROM ERYTHROCYTES UNDER HIGH SHEAR STRESSES IN TUBE FLOW

H. Schmid-Schönbein, I. Rohling-Winkel, P. Blasberg, E. Jüngling, A. Wehmeyer, G.V.R. Born *, and P. Richardson. Dept. Physiology, Med. Faculty, RWTH Aachen, Dept. Pharmacology, King's College, London, Division of Engineering, Brown University, Providence, R.I.

ADP stemming from red blood has been shown to "activate platelets" producing shape change, as well as aggregation and release. However, the mode of release of ADP from intact RBC has never been established. Contrary to popular misconceptions, high shear stresses (τ) prevail during natural hemostatic plug formation in arteries and arterioles. Therefore, we tested ADP-release from RBC subjected for 5-100 msec in tube flow ($\tau = 0\text{-}200 N/m^2$) during passage through a hollow fiber (Ø 400 µm, L = 20 cm) with semipermeable walls (AMICON R). Samples from the fluid layer near the wall were ultrafiltered through it and became accessible for chemical analysis. Concentrations of K^+, adenosine nucleotides (HPLC), and Hb (in the supernatant) before and after shear exposure were measured. At $\tau > 50 N/m^2$, K^+, adenosine nucleotides, and hemoglobin concentrations rose in the supernatant. Only K^+ was higher in the ultrafiltrate than in the latter, whereas total concentration of adenosine nucleotides were not different and hemoglobin did not permeate. There was no difference between the relative molar concentration of total adenosine nucleotides of hemoglobin, i.e. the nucleotides and hemoglobin content of 10^{-4} and 10^{-3} of all RBC were liberated. In the ultrafiltrate (ADP) > 2×10^{-7} M/L, sufficient to activate platelets in the presence of Ca^{++}.