ALTERED COAGULATION IN CEREBRAL ISCHEMIA PATIENTS. H. Fisher and R. France, Departments of Neurology and Medicine, USC School of Medicine, Los Angeles, CA, USA.

We investigated coagulation changes in a group of patients with cerebral ischemia, ranging from transient ischemic attacks to cerebral infarction. Patients were studied acutely (within 72 hours of onset of ischemia) and again approximately 2 months following the initial examination. We evaluated platelet activation, fibrin generation, and fibrinolysis by measuring plasma beta-thromboglobulin (BTG), fibrinopeptide A (FPA), and fibrinopeptide B-beta 1-42 (FPB), respectively. We compared measurements in cerebral ischemia patients with a group of age- and sex-matched neurological inpatients without vascular disease ("patient controls") and a similarly matched group of normal volunteers ("normal controls"). BTG levels for 90 patients studied acutely were not significantly different compared to normals (12.2 ± 6.5 ng/ml; n = 44); patient controls (13.2 ± 7.6 ng/ml; n = 18) were not significantly different from normals. In contrast, FPA measurements were significantly increased in acute patients compared to normals (3.3 ± 5.8 versus 1.0 ± 1.7 ng/ml, p < .05) while FPA levels 2 months post-ischemia (0.6 ± 0.9 ng/ml) were no different than normals. FPB measurements were not significantly different among either acute patients (6.5 ± 2.4 pmol/ml) or patients 2 months post-ischemia (4.8 ± 1.5 pmol/ml) compared to normals (6.9 ± 1.8 pmol/ml).

In summary, we have found, among patients with cerebral ischemia, sustained increases in BTG, acute increases in FPA, and normal FPB. These findings are compatible with a model of cerebral ischemia consisting of acutely increased fibrin generation without concomitant increased fibrinolytic activity, superimposed on a background of increased platelet activation.

ENHANCED GLOMERULAR PROCOAGULANT ACTIVITY AND FIBRIN DEPOSITION IN RATS WITH MERCURIC CHLORIDE-INDUCED AUTOIMMUNE NEPHRITIS.

The mechanism involved in glomerular fibrin deposition was investigated during HgCl₂-induced autoimmune glomerulonephritis in the Brown Norway rat. To ascertain whether the local hemostatic system was activated secondarily to the immunological conflict, the ability of glomerular lysates to induce coagulation in vitro was measured in treated and control rats. In day 12 (latent phase of the disease), 20 rats were divided into two groups: Group A rats were used for in vivo experiments; in the course of in vivo experiments the possible passage of MCH through "cell saver" devices was investigated. In Group B experiments the same was studied through a passage of 40 mm pore-size blood transfusion filters. In the in vivo experiments MCH was injected intravenously into different parts of the circulatory system of rabbits after different degrees of dilution and filtering after which the animals were killed at different time intervals and the radioactivity was determined in respective brain sections by autoradiography. The results of these experiments suggest that the passage of MCH indeed occurs through different blood-collecting circuits and cause organ damage. The authors recommend that the substance should not be used if shed blood is intended to be collected and returned to the patients circulation.