HEPARIN'S ENDOGENOUS FUNCTION, PRIMARILY ANTICELLULAR-DESTRUCTIVE, INCIDENTALLY ANTICOAGULANT, IS DOSE, SOURCE, AND PH RELATED. M.J. Saliba, Jr. University of California, San Diego, School of Medicine, La Jolla, California, U.S.A.

Heparin's obscure endogenous function may be resolved by new experimental findings, reported by Saliba and coworker since 1963, with heparin use in controlled animal studies and patients Published online: 2019-04-16 and myocardial ischemia. Large doses of intestinal source heparin, administered topically and parenterally, significantly reduced burn extension, edema, fasciectomy, parenteral fluids, skin grafts, and contractures. Then, similar large doses of intestinal source heparin significantly reduced the cellular-destructive pathophysiology of myocardial ischemia, improved electrocardiographic S-T segments 84%, reduced myocardial necrosis 32%, and salvaged creatine phosphokinase 15%. Lung source heparin produced significant but lesser effects on S-T segment elevations and adenosine triphosphate levels. The large heparin dose needs, without bleeding problems, suggested more than anticoagulation was involved. All known heparin effects were theoretical mechanisms: anticoagulant, antithrombosis, antilipemia, antihistamine, antiserotonin, antiproteolytic enzyme, anticomplement, antilyosomal enzyme, and antiexcess positive ion. For those effects, individually often and collectively always, large doses of heparin were required. Inactivation-combination reactions of heparin with histamine, serotonin and some tested proteolytic enzymes were pH related, acidic pH dependent, alkaline pH reversible. So, thermal burns and myocardial infarcts were two severe cellular-destructive diseases, with ischemic components, that were significantly improved by appropriately large doses of source specific heparin. It is concluded that at acidic pHs intestinal heparin in larger than anticoagulative dose is mainly an anticellular-destructive agent, incidentally anticoagulant. New uses may result.

CHANGE ON BLOOD COAGULATION, PLATELET FUNCTION AND FIBRINOLYSIS IN DIABETES MELLITUS - PRELIMINARY REPORT. R. Giustolisi, R. Musso, T. Lombardo, M. Russo and E. Cacciola - Department of Hematology University of Catania - Italy -

Some coagulation aspects are studied in diabetes mellitus because this dismetabolic disease represents a "high risk factor" of predisposition leading to classical lesions of the vascular wall and thrombosis. Were studied 24 diabetic patients between 16 and 68 years old and 14 healthy subjects. Tests performed are followed: partial thromboplastin time (PTT), plasma coagulation time RVV (RVV-T), antithrombin III (At-III), α_2 macroglobulin (α_2 M), fibrin/fibrinogen degradation products (FDP), ethanol gelation (EG) and protamine sulphate (PS), euglobulin lysis time (ELT), platelet adhesiveness to glass (PAG), platelet adhesiveness in vivo (PAV), platelet factor-3 availability (PF-3), platelet aggregation by ristocetin 1,2-1,5-1,8 mg/ml (RIPA).

Diabetics showed a fall in At-III, increase α_2 M, a significant decrease ELT and increase FDP with often positivity EG. We also noted a shortening of PTT, PF-3 rate and RVV-T. In vitro platelets adhesiveness rises more than it does in vivo. Besides the PAV from diabetics increased the control subjects PAG. The RIPA is increased. Our findings showed, therefore, in diabetic patients a thrombophilic pattern by blood hypercoagulability and fibrinolytic activity decreased.

EFFECTS OF SULOCTIDIL ON CIRCULATING PLATELET AGGREGATES FORMATION IN RATS. J. Roba, M. Claeys and G. Lambelin. Continental Pharma, Research Laboratories, Brussels, Belgium.

In a search for new models allowing testing of antithrombotic agent in vivo, we have adapted the Wu and Hoak technique for measuring circulating platelet aggregates in rats. The pattern of reversible and irreversible platelet aggregates is studied by sampling blood in vena cava and/or orbital sinus following a) i.v. ADP injection; b) implantation of an aortic catheter; c) adjuvant induced arthritis.

Sulocitidil (p.o.) can prevent formation of circulating aggregates. Its effects are compared with those of other antiplatelet agents.