

THERAPEUTIC ULTRASOUND - AN INDUCER OF PLATELET AGGREGATION IN VITRO.

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Exposure of human platelet rich plasma to therapeutic levels of ultrasound, was found to initiate platelet aggregation and release in vitro. The amount of aggregation, as assessed using a modified platelet aggregometer was found to be related to both the intensity and frequency of the ultrasound. Aggregation occurred at a faster rate and was more extensive at lower frequencies (0.75MHz) and higher intensities. The ability to induce aggregation was found to be directly related to platelet sensitivity to ADP, more sensitive platelets responding to lower intensities of ultrasound. Experiments monitoring the release of β thromboglobulin, a marker of the release reaction, indicated that ultrasound was inducing release in two distinct ways.

Firstly by the physical disruption of a number of platelets, and secondly by ADP released from the disrupted platelets inducing further release. Release and platelet aggregation was found to parallel the disruption of red cells, as measured by plasma haemoglobin levels, and this coupled with the greater effect at lower frequencies is thought to indicate that platelet destruction is occurring by cavitation.

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HAEMOSTATIC FACTORS IN UMBILICAL ARTERY AND VEIN BLOOD. M.E.Foley, J.K.Clayton and G.P.McNicol. University Department of Obstetrics and Gynaecology and Medicine, University of Leeds, Leeds, England.

Increased clotting factor activity has been demonstrated by other workers in uterine vein blood at the time of placental separation. To examine some effects of placental separation on the fetus, haemostatic factors in umbilical artery and vein blood were studied immediately following delivery in 80 normal babies. Clotting activity was increased in umbilical vein blood as compared with umbilical artery blood; the most obvious difference was a significant shortening of the activated partial thromboplastin time. Cord blood showed a high level of fibrinolytic activity; activity measured in plasma by the fibrin plate method and with the euglobulin lysis test was significantly greater in the umbilical vein than in the artery. Normal levels of fibrinogen degradation products indicated no active fibrinogenolysis or fibrinolysis. The mean umbilical vein blood platelet count ($233 \times 10^9/\text{litre}$) was significantly higher than the mean umbilical artery blood platelet count ($205 \times 10^9/\text{litre}$). Platelet factor 3 availability showed no arteriovenous difference. Platelet aggregation to 2 and 4 micromolar adenosinediphosphate was significantly greater in the umbilical vein than in umbilical artery blood. The data indicate that at the time of placental separation, there is increased potential haemostatic activity in the fetus.

MEASURING ANTITHROMBIN III WITH A CHROMOGENIC SUBSTRATE (CHROMOZYM-TH): WHICH BUFFER SYSTEM? N. Troger, P. Spycher, M. Furlan and E.A. Beck. Central Hematology Laboratory, Inselspital, Berne, Switzerland.

Synthetic oligopeptides, coupled with a chromophore, may be used for measurement of thrombin as well as of thrombin inhibitors. This principle was applied to a recently synthesised tripeptide derivative (Tos-Gly-Pro-Arg-PNA, commercial designation: Chromozym-Th). Following partial activation of antithrombin III with heparin we found a marked dependence of the activation kinetics on some common buffer systems. At constant pH (8.2), molarity (0.15) and heparin concentration (0.5 NIH units/ml), thrombin inhibition was as follows: Tris > glycine > imidazole >> triethanolamine. Following full activation of antithrombin III with excess amounts of heparin these differences were less apparent. Since partial activation of antithrombin III might also be expressed as biologic activity of heparin, careful attention should be given to the selection of an appropriate buffer system before such tests are introduced into the clinical laboratory.