Time

14.30 **0809** FIBRINOLYSIS IN SOLID TUMORS

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The fibrinolytic activity of 120 malignant and 25 benign solid tumors from autopsy and biopsy specimens was studied by the fibrin slide technique as described by Kwaan and Astrup. The inhibitory activity against fibrinolysis was graded according to the lysis time of vascular tissues within the tumor. The results show that all malignant solid tumors, with the exception of carcinoma of prostate demonstrate varying degrees of inhibition of fibrinolysis. Persistently high inhibitory activity was found in squamous cell carcinoma of esophagus, the respiratory tract (including squamous cell carcinoma of sinuses, larynx and lung), cervix uteri and skin; carcinoma of uterus, colo-rectal carcinoma; small cell anaplastic carcinoma of lung; neuroblastoma; carcinoma of bile duct; while malignant tumors of the kidney show a lesser degree of inhibition. In contrast, with the exception of hydatidiform mole, benign solid tumors show little or no inhibition. There is a difference in fibrinolytic activity between the peripheral and the central portions of a tumor with distinctly greater degree of activity in the former. The pathophysiologic implications of the presence of such a strong inhibition of fibrinolysis is apparent. Biochemical characterization of this inhibition is currently in progress.

14.45 **0810** PLATELET AGGREGABILITY AND THROMBOGENESIS DURING CHEMICAL CARCINOGENESIS

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There are suggestions that the metastatic spread of malignant tumours depends *inter alia* on platelets. To find out whether platelets are affected by a common technique for experimentally inducing malignancies, rats and mice were injected with 3-methylcholanthrene to induce non-metastasising fibrosarcomata. After 14-21 days, platelet aggregation measured *in vitro* (Born, 1962, Nature, 194, 927) and arteriolar thrombogenesis by laser irradiation *in vivo* (Kovacs *et al*., 1973, Microvascular Research, 6, 194) were significantly inhibited. Therafter these inhibitions disappeared but reappeared when tumours became palpable, before any change in blood platelet concentration.

15.00 **0811** HYPERCOAGULABILITY ASSOCIATED WITH BREAST CANCER. J.A. Caprini,* L.Zuckerman, J.P. Vagher and E. Cohen. Evanston Hospital, Northwestern University, Evanston IL, U.S.A.

Previous thrombelastographic (TEG) studies in 262 individuals (not on medications) has resulted in the derivation and verification of a discriminant function equation which can classify individuals into a normal or accelerated coagulability range based on the TEG results. The significance of the separation between the groups was related to the combination of the individual's TEG parameters for whole blood (WB) and their celite activated WB. The celite activated rate of clot formation appeared to be the most discriminating of the TEG variables. The equation separated the individuals with known malignancies (121) from the benign or healthy volunteers (141) with an overall accuracy of 99%. This breaks downinto a 97-98% sensitivity and a 100% specificity (false positives). These findings of hypercoagulability are not specific for cancer and can be seen in other disease states, e.g. sepsis or even the result of some medications. Therefore the present study was done to apply this analysis to a consecutive series of consenting patients (119) just prior to open breast biopsy, without selecting out patients on medications. 50/68 patients with benign lesions had normal TEG results (false positive rate 26%), while 48/51 patients with pathologic evidence of malignancy had positive results. In two of the three tumor patients with a negative analysis, no invasion of the tumor was seen. The reduced specificity found with this group may be attributable to the presence of patients on medications or a higher than normal incidence of other disease states in this population. The results demonstrate one clinical application of TEG analysis to the identification of breast malignancies.