bosis. This paper describes a model in which thrombosis is initiated by an electrical stimulus. The thrombus produced has the histological and biochemical features of human deep vein thrombosis (DVT).

The minimum stimulus necessary to induce thrombosis was first determined by passing a fixed current for timed intervals along the femoral veins of 10 rabbits. Thrombi were seen 24 hours later if the total charge passed exceeded a threshold value of 25 millicoulombes. With this small current, no endothelial changes were visible immediately after the passage of the charge on light or scanning electron microscopy. At 24 hours a mural thrombus formed, which had fully corss-linked fibrin and histological features resembling human DVT.

In the second series of experiments, the sequence of changes occurring in thrombus production was investigated in 3 groups of 18 rabbits each. After passage of the critical charge along the femoral vein in each animal, veins were removed at fixed intervals, the contralateral vein acting as a control. The veins were examined by scanning electron-microscopy (Group I), transmission electron-microscopy (Group II) and light microscopy (Group III). The earliest changes were detectable at 5 minutes and consisted of the laying down of an organised structure of criss-crossing fibrin strands with small platelet clumps at fibrin intersections. Later the fibrin structure spread towards the lumen; platelet clumps fused and a coralline thrombus was formed by 24 hours. The significance of these changes will be discussed.

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The capacity to induce fibrin retraction has been considered a specific property of platelets until recently, when Niewiarowski et al. (Proc. Soc. Exp. Biol. Med. *140*, 1199, 1972) observed fibrin retraction induced by human fibroblasts. As a part of a larger study on the interactions of cultured cells with fibrin, we have investigated the ability of the following cell lines to retract fibrin: KB (human oral epidermoid carcinoma); HeLa (human cervix carcinoma); Chang Liver (human, normal epithelium; Chang Conjunctiva (human normal epithelium); NCTC clone 929 (L) (fibroblasts from C3H/AN mice) and BA 1112 (rhabdomyosarcoma developed on WAG/Rji inbred rats). Cells were cultured in Eagle's MEM, in Hank's blanced salt solution plus 10% calf serum, removed from the flasks by trypsin treatment and resuspended at a concentration of $2 \times 10^6$/ml in Tyrode-albumin solution, containing $Ca^{++}$ and $Mg^{++}$. Human citrated platelet-poor plasma was clotted in a test tube at 37° C by thrombin in the presence of either the cell suspensions or buffer. Only BA 1112 cells were able to retract fibrin; the presence of $Ca^{++}$, cellular integrity and random distribution in the sample were required for this activity. BA 1112 cells were able to modify the structuration of thrombin-induced fibrin as indicated by the marked increase of the maximal amplitude of thrombelastogram. BA 1112-induced fibrin retraction was inhibited by $PGE_1$ and by some pyrimido-pyrimidine derivatives, not by apsirin. No retraction occurred when reptilase instead of thrombin was used as the clotting agent, even if the cells were preincubated with ADP. These results suggest that BA 1112 cells have a susceptibility to thrombin similar to that of platelets; this hypothesis is interesting in view of the muscular origin of these cells.

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*A. Poggi, N. Polentarutti, M. B. Donati, G. de Gaetano and S. Garattini* (Istituto di Ricerche Farmacologiche 'Mario Negri,' Via Eritrea, 62 – 20157 Milano, Italy): **Fibrinogen, Platelet and Red Cell Kinetics in Mice Bearing an Experimental Tumor. (524)**

In view of the possible role of platelets and coagulation mechanisms in the growth and dissemination of solid tumors, a number of haematological parameters have been followed during development of an experimental syngeneic tumor in mice (Lewis Lung Carcinoma, 3LL). This tumor, when transplanted intramuscularly in $C_{57}$Bl/6 mice, grows locally and