

By an hour smooth muscle showed a decrease in myofibrils and an increase in rough endoplasmic reticulum and collagen lost its ability to stain with uranyl acetate-lead citrate.

By 24 hours smooth muscle cells had undergone various changes and formed a partial covering layer over the surface of denuded areas, often by extending long "arms" from a bulky, fibroblast-like "body". Various stages of de-differentiation and re-differentiation were abundant. By 72 hours these cells had become thinner and resembled the "immature" endothelium of Florey and others. However, the sheet was still discontinuous. By 7 days and thereafter to 28 days the endothelium was continuous and typical of "immature" endothelium morphologically but the surface membrane stained intensely in some instances.

At 24 hours there was a perivascular zone of edema with cell ghosts and amorphous debris which decreased thereafter. Collagen staining was again typical by 7 days.

These observations indicate that the luminal surface of blood veins can be initially repaired by the rapid de-differentiation and re-differentiation of smooth muscle rather than waiting for the ingrowth of endothelial cells from the margins of the denuded areas.

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J. Lough and S. Moore (Montreal General Hospital, 1650 Cedar Ave. Montreal, Quebec):
Early Endothelial Injury. (52)

A variety of blood constituents were injected into an isolated segment of rabbit aorta to determine which elements might be involved in early endothelial injury. Test materials consisted of platelet rich plasma (P. R. P.) alone; P. R. P. plus adenosine diphosphate (A. D. P.); P. R. P. plus tendon extract; P. R. P. plus thrombin; ultrasonicated P. R. P. alone; platepoor plasma (P. P. P.) alone and thrombin in saline. Each experimental mixture was left in the aorta for 15 minutes, followed by reflow for 20 min. The vessel was then fixed by glutaraldehyde perfusion. Thick sections of the entire circumference of the aorta were taken for phase contrast microscopy and representative areas were selected for electron microscopy. In control experiments and with sonicated P. R. P. and P. R. P. plus tendon extract, the endothelium was normal. With P. R. P. alone, P. P. P. alone and with P. R. P. plus A. D. P. there were occasional subendothelial vesicles. When P. R. P. plus thrombin and P. P. P. plus thrombin were injected separately to form a thrombus or when thrombin in saline was used, there was extensive subendothelial vesiculation with focal ulceration and adherence of thrombus to endothelium. Severe injury was associated with the presence of thrombin initiating the polymerization of fibrinogen to fibrin. Electron micrographs demonstrate the earliest lesion as a disruption of the superficial fibrillary elastica with separation of overlying endothelium.

Additional Key Words: Plasminogen activation, turbulent flow, atherosclerosis, encrustation.

I. B. Kovács, A. Sebes, K. Trombitás, L. Csalay and P. Görög (Korvin Otto Hospital, H-1071 Budapest, University School of Medicine, Pécs and EGYT Pharmacochemical Works, Budapest, Hungary): **Improved Technique to Produce Endothelial Injury by Laser Beam without direct Damage of Blood Cells.** (53)

Ultrastructural study of microcirculatory thrombus induced by He-Ne laser and application of energy absorbing dye Evans blue demonstrated severe endothelial damage covered by extremely disintegrated platelets. To exclude the direct effect of laser beam to platelets and red cells, we used the technique of Baez for the perfusion of intact mesentery microvasculature of the living rat in situ. During laser irradiation arteries deriving from the ileocolic artery were filled with albumin solution containing Evans blue, thereafter the blood circulation was restored and thrombus formation was investigated at the site of irradiation. According to the ultrastructural findings, thrombus formation is based on the interaction between the exposed subendothelium and intact platelets. Additional advantage of this technique that lower dose of Evans blue is sufficient.