J. W. Ryan and Una S. Ryan (Papanicolaou Institute, P. O. Box 23–6188, Miami, Fla., 33123): **Pulmonary Endothelial Cells/ Metabolism of Vasoactive Substances. (48)**

The lungs metabolize a variety of vasoactive substances, including bradykinin (BK), angiotensin I (AT I), PGE_2 and $F_2\alpha$, norepinephrine, 5-HT, 5'-ATP and 5'-AMP. In contrast, the lungs od not metabolize angiotensin II (AT II), PGA_2, histamine and epinephrine. Of the substances metabolized, all (with the possible exceptions of the prostaglandins) are processed primarily by the pulmonary endothelial cells. Furthermore, the means by which the substances are processed suggest that endothelial cells determine the vasoactive substances allowed to enter the systemic arterial circulation. BK is inactivated while AT I is converted to its potent homolog, AT II. AT II enters the arterial circulation. The metabolism of BK and AT I may be effected by the same enzyme. Pulmonary endothelial cells are a rich source of thromboplastin, an enzyme capable of degrading BK and AT I. However, the relationship of thromboplastin to the fates of these hormones is not clear: The metabolic products produced are not those produced by intact lungs nor by endothelial cells in culture. In addition, thromboplastin degrades substances (e.g. AT II), which are not degraded by intact lungs. Possibly the extrinsic clotting system plays a role when activated but not under physiologic conditions.

S. A. Evensen and T. Henriksen (Institute for Surgical Research and Medical Department A, Rikshospitalet, Oslo, Norway): **Sterol Synthesis in Human Endothelial Cells. (49)**

Endothelial cells were isolated by collagenase digestion of the intima of the vein of umbilical cords and grown in medium 199 or RPMI 1640 supplemented with 20% FCS. After 3–5 days in primary culture fresh medium was added containing either sodium acetate-2-^{14}C or mevalonic acid lactone-2-^{14}C. Following further 24, 48 or 96 h incubation the harvested cells and media were extracted according to Folch and separated on TLC. About 35% of the total cellular radioactivity accumulated in the sterol fraction after 48 h incubation with labelled acetate. Delipidation of the medium resulted in markedly increased incorporation. Incorporation of mevalonic acid into cellular sterols was maximal at 48 h, and larger in RPMI 1640 than in medium 199. In the medium a progressive accumulation of labelled sterols with time was observed. Approximately 20% of the cellular radioactivity was found in the FFA fraction and was saponifiable. Whether this finding represents fatty acid synthesis from mevalonic acid or an intermediary product in sterol synthesis has not yet been clarified.

H. Payling Wright and M. Evans (Charles Salt Research Centre, Orthopaedic Hospital, Oswestry, Shropshire, England): **Reactions of Human Endothelial Cell Cultures Exposed to Adrenalin Concentrations. (50)**

Cultures of vascular endothelium obtained from fresh human umbilical veins and grown *in vitro* in fortified 199 medium for several days have been subjected to differing concentrations of adrenalin for various times. Their reactions to the drug, as seen microscopically, were recorded photographically. The viability of endothelial cells under these cultural conditions gives a measure of the maximal exposure to adrenalin which they are able not only to survive, but also to multiply. Their capacity to mitose was studied autoradiographically.

The significance of the findings will be discussed with reference to atherogenesis and particularly the possible link with infection and in "stress".

G. J. Stewart, W. G. M. Ritchie and P. R. Lynch (Specialized Center for Thrombosis Research, Dept. Med., Temple Health Sciences Center, Philadelphia, Pa. 19140. U.S.A.): **Recovery of the Venous Wall from Leukocyte Induced Injury. (51)**

Surgical trauma to tissue adjacent to canine jugular veins combined with brief local stasis caused massive leukocyte invasion of venous walls. This resulted in the entrapment of pockets of leukocytes between the endothelium and basement membrane and subsequent detachment of patches of endothelium.