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PLENARY LECTURES

L-1 Microcirculation in patients with arterial hypertension

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Regardless of the mechanisms that initiate the increase in blood pressure, the development of functional and structural changes in the systemic vasculature is the final result of long-standing hypertension. These changes can occur in the macro- but also in the micro-vasculature. The supply of the tissues with gases, nutrients, and metabolites occurs almost exclusively in the microcirculation (which comprises resistance arterioles, capillaries and venules), and an adequate perfusion via the microcirculatory network is essential for the integrity of tissue and organ function.

This key lecture focuses on results from clinical studies in hypertensive patients, which have been performed in close cooperation with different clinical groups over the last three decades. Intravital microscopy was used to study skin microcirculation, microcatheters for the analysis of skeletal muscle microcirculation, the slit lamp for conjunctival microcirculation and the laser scanning ophthalmoscope for the measurement of the retinal capillary network.

In about 93% of patients with hypertension first changes of the normal microcirculation can be found, long before organ dysfunctions become clinically manifest. The earliest disorders were found in skin capillaries and thereafter in the retina and the skeletal muscle. In general the disorders in the different areas correlated clearly. While capillary rarefaction occurred mainly in the retina and the conjunctiva bulbi, in skin capillaries morphological changes were rare. A significant decrease of capillary erythrocyte velocities under resting conditions together with a marked damping of the postischemic hyperemia was found, both correlating with the duration of hypertension or WHO stage or the fundus hypertonicus stage. Also the mean oxygen tension in the skeletal muscle correlated with the state of the disease.

These data show that the microcirculatory disorders in hypertension are systemically and are hallmarks of the long-term complications of hypertension. There is now a big body of evidence that microvascular changes occur very early and may be important in their pathogenesis and progression.

L-2 Erythrocyte and polymorphonuclear leukocyte rheology in diabetes mellitus

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Using several techniques, we examined erythrocyte rheology in diabetes mellitus. Whole-blood filtration was decreased in type 2 diabetics, while filtration of erythrocyte suspensions did not show differences. The erythrocyte filtration using the Filtrometer MF4, a flow pressure and high haematocrit system, showed

significant differences. Diffractometry showed an increase in elongation index in type 1 diabetics. The microrheological methods were based on fluorescence spectroscopy. We examined membrane microviscosity in ghosts labelled with DPH, while in intact cells we explored the membrane polarity gradient (using the probes 2-AP, 6-AS, 9-AS, 12-AS), the membrane fluidity and protein lateral mobility using respectively pyrene and pyrene-3-maleimide. No difference was noted regarding membrane microviscosity and polarity gradient; differences were observed regarding membrane fluidity and protein lateral mobility. We also investigated the filterability of polymorphonuclear (PMN) - St. George Filtrometer - the PMN membrane fluidity and cytosolic $Ca2^{+}$concentration, at baseline and after activation (PMA and fMLP). At baseline no difference was evident in PMN filtration parameters (IRFR, CP) or in cytosolic calcium content between normals and diabetics; a difference was noted in membrane fluidity. In normals, after PMN activation we observed a decrease in IRFR and an increase in CP without variation in the membrane fluidity and cytosolic $Ca2^{+}$content. In type 1 diabetics after PMN activation we observed a decrease in the PMN filtration parameters and also a decrease in the membrane fluidity and an increase in cytosolic $Ca2^{+}$content. In type 2 diabetics after PMN activation we noted a decrease in the filtration parameters and an increase in cytosolic $Ca2^{+}$content. We also examined the PMN beta2-integrin pattern (CD11a, CD11b, CD11c, CD18) in diabetics of type 1 and 2. At baseline, CD11 was reduced in type 1 and 2 diabetics, CD11c was increased in type 1 and 2 diabetics and CD18, decreased in type 1 diabetics, was increased in type 2 diabetics. After activation in the normal subjects we observed an increase of all beta2-integrins but not in type 1 and 2 diabetics.

L-3 Contribution of erythrocytes to the regulation of tissue perfusion

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Red blood cells (RBC) have long been considered as passive carriers of respiratory gases. More active roles in the maintenance of sufficient tissue perfusion have been attributed to RBC in the last several decades, mainly based on the observations related to vasoactive substances originating from RBC. Experimental evidences include the ATP release from RBC under hypoxia and mechanical stress, which in turn induces nitric oxide (NO) generation by endothelial cells via purinergic receptors. RBC may also directly influence NO bio-availability at microcirculatory level either by acting as a scavenger or a source for NO. Enzymatic and non-enzymatic mechanisms contribute to NO generation in RBC. Furthermore, it has been demonstrated that NO can be exported from RBC under certain circumstances (e.g., hypoxia), with the potential of influencing the local vasomotor balance. Although the details of the mechanisms has not been well understood yet, NO generation and export by RBC has been demonstrated to be enhanced by the effective shear forces. Recent experimental data indicated that NO export by RBC exposed to physiological levels of shear stress may influence the tonus of small arteries.

In addition to their interference with vasomotor tonus, RBC may have more direct contribution to the regulation of hemodynamic resistance, as suggested by observations on the modulation of RBC mechanical properties by shear forces. It has been recently demonstrated that RBC deformability increases in response to shear forces affecting on them, within the physiological ranges. The time course of this response is in the order of seconds, therefore fast enough to contribute to the microvascular resistance ahead, if RBC is exposed to increasing shear forces as it approaches to arteriolar level. The magnitude