The microcirculation is fundamental to the exchange of gases and nutrients necessary for tissue function and intuitively one might suppose that the specialization of a particular microcirculation would preclude generalizations from one circulation to another. Nevertheless, a paper in this issue by Antonios and co-workers is interesting for two reasons, firstly they imply that rarefaction may not be the case. This study examined capillary density, an important determinant of microcirculatory exchange, in the skin of 49 patients with a diagnosis of syndrome X. Patients were compared with age- and sex-matched normal subjects and the syndrome X group was also subdivided into normotensive and hypertensive groups. Interestingly, both hypertensive and normotensive patients with syndrome X had significantly reduced capillary densities both at rest and following venous congestion to maximally perfuse the microcirculation. The association of hypertension with reduced capillary density (or rarefaction) is well recognized and it has been reported that several tissues including skeletal muscle, skin, the conjunctiva, and retina show reduced capillary density in hypertension. In addition it has also been observed that patients with impaired insulin sensitivity and subjects with low birth weight show evidence of reduced capillary density. The data of Antonios and co-workers is interesting for two reasons, firstly they imply that rarefaction may not necessarily be associated with elevated brachial blood pressure. Indeed other studies suggest that rarefaction is present in subjects at high risk of developing hypertension and so may precede the development of hypertension. Secondly they suggest that structural abnormalities of the microcirculation in syndrome X may not be confined to the coronary circulation. Such a possibility is consistent with previous observations showing abnormalities of endothelial function and reduced forearm flow following maximal vasodilation in syndrome X.
If syndrome X is a coronary manifestation of a systemic abnormality, then global disturbance of endothelial function seems an attractive candidate for the underlying cause. This possibility could extend the focus of attention onto microvascular endothelial function in other conditions, such as hyperlipidaemia, where abnormalities in endothelial function in large arteries are well recognized. The accessibility of the finger skin and the relative ease and reproducibility of the technique used by Antonios and colleagues offers a possible means to test such speculation.

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References


