PRE- AND POSTCAPILLARY INTERACTION IN THE MICROCIRCULATION

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Rhythmic changes in the diameter of small pre- and postcapillary microvessels are prominent characteristics of unanesthetized and carefully maintained anesthetised animal preparations[1]. In view of the functional and spatial relationship of these vessels to the capillaries and based on previous analysis[2], there is reason to suspect that capillary blood pressure, flow, and ultimately cellular homeostasis are in part dependent and possibly controlled by these microvessel dynamics. A microvascular model[3] has been developed and utilized to help interpret and quantify the separate and interactive effects of in vivo pre- and postcapillary microvessel dynamics on microcirculatory function.

Methods

The bat wing was used as an experimental preparation and as a self-contained vascular bed. The number, dimensions, and distribution of the vessels of the real vascular bed were included into an analyzable, representative geometric configuration. Based on theoretical analysis and experimental data, equations were developed and utilized to characterize the pressure-flow relationships for each branching order of the vascular field. The geometric configuration and associated describing equations (topological model) formed the framework on which an extensive computer based microvascular model was built. The microvascular model included the macroscopic properties of the vascular bed as determined and verified using the topological model. In addition it contained a detailed representation of precapillary vessel dynamics (vasomotion), capillary hemodynamics and filtration, and postcapillary dynamics (venomotion). In vivo comparisons including pressure distribution and propagation characteristics served to test the accuracy of the microvascular model prior to its use to interpret the functional significance of vasomotion and venomotion at the capillary level.

Results

venomotion on capillary

1. The contraction phase of venomotion is associated with a decrease in postcapillary segmental compliance (C), and an increase in segmental resistance (R). For example, a 15% diameter reduction (typical of values observed in vivo) produces a -37% change in C and a +60% change in R.

2. These vascular changes are accompanied by an increase in the postcapillary vessel pressure by an amount which depends on the precontraction pressure level, with a range of 4 to 8 mmHg.

3. These combined effects in the postcapillary vasculature produce an increase in capillary pressure (typically 3 to 7 mmHg) and a decrease in capillary flow (typically 25% reduction).

vasomotion on capillary

4. Contraction of terminal arterioles and/or precapillary sphincters reduces the capillary pressure and flow by an amount which naturally depends on the degree of diameter reduction. For typical in vivo diameter changes the capillary pressure falls to a value below the commonly accepted value of plasma osmotic pressure during the entire interval of contraction.

5. Elevation of arterial pressure results in an increase in the average amount of time that the precapillary vessels remain contracted. This property has the tendency to autoregulate the average capillary flow (e.g., 5% change in flow for a 2:1 pressure variation).

combined venomotion and vasomotion

6. The capillary pressure and flow modulation produced by venomotion depends on the contractile state of the precapillary sphincters (PCS). If the PCS are fully relaxed and dilated, venomotion produces a 20% modulation in the flow. However, if the PCS are in their contracted state, the capillary flow change produced by venomotion increases to 50%.

7. The capillary flow modulation in a given capillary produced by vasomotion depends on the contractile state of the postcapillary vessels into which the capillaries feed. If the postcapillary vessels in the vicinity of the capillary are contracting, then the pressure elevation due to this venomotion (4 to 8 mmHg) is sufficient to close previously open venule and small venous valves. Hence the amount of capillary flow change due to vasomotion does not just depend on the amount of the PCS diameter changes and arteriolar pressures.

Conclusions

1. The finding that the contraction phase of vasomotion is associated with a reduction in capillary pressure to a value below osmotic pressure levels implies that PCS contraction is associated with capillary re-absorption for normally occurring arterial pressures. Thus the classical picture of a rather steady filtration on the arterial end of the capillary and re-absorption on the venous end needs to be modified to include this dynamic component.

2. The manner in which pre- and postcapillary dynamics interact implies that the relative importance of the postcapillary vasculature in the control of capillary flow distribution increases in physiologic/pathologic states in which PCS contraction time increases. Further, since this time was shown to increase during arterial pressure elevation, we would predict that the postcapillary vasculature and associated dynamics assume a role of increased functional significance in hypertension.

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This work was supported in part by USPHS HL 103 30 and USPHS HL 07762-12.

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