A proposed mechanism for metabolic flow regulation involves the oxygen-dependent release of ATP by red blood cells, which triggers an upstream conducted response signal and arteriolar vasodilation. To analyze this mechanism, a theoretical model is used to simulate the variation of oxygen and ATP levels along a pathway of seven representative segments, including two vasoactive arteriolar segments. An expression for the conducted response signal is defined by integrating the ATP concentration along the vascular pathway, taking into account exponential decay of the signal in the upstream direction. Arteriolar tone depends on the conducted metabolic signal and on local wall shear stress and wall tension. Arteriolar diameters are calculated based on vascular smooth muscle mechanics. The model predicts that combining the conducted, myogenic, and shear-dependent responses can account for a nearly 10-fold increase in perfusion in response to a 20-fold increase in oxygen demand. Excluding myogenic and shear-dependent responses from the model does not greatly alter the perfusion attained at high oxygen demand. Supported by NIH grant HL070657.
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- vasomotion
- small arteriole dominance
- autoregulation
- perfusion increase

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