# BENCH-TO-BEDSIDE REVIEW: MICROVASCULAR DYSFUNCTION IN SEPSIS-HEMODYNAMICS, OXYGEN TRANSPORT, AND NITRIC OXIDE

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## ABSTRACT

The microcirculation is a complex and integrated system that supplies and distributes oxygen throughout the tissues. The red blood cell (RBC) facilitates convective oxygen transport via co-operative binding with hemoglobin. In the microcirculation oxygen diffuses from the RBC into neighboring tissues, where it is consumed by mitochondria. Evidence suggests that the RBC acts as deliverer of oxygen and 'sensor' of local oxygen gradients. Within vascular beds RBCs are distributed actively by arteriolar tone and passively by rheologic factors, including vessel geometry and RBC deformability. Microvascular oxygen transport is determined by microvascular geometry, hemodynamics, and RBC hemoglobin oxygen saturation. Sepsis causes abnormal microvascular oxygen transport as significant numbers of capillaries stop flowing and the microcirculation fails to compensate for decreased functional capillary density. The resulting maldistribution of RBC flow results in a mismatch of oxygen delivery with oxygen demand that affects both critical oxygen delivery and oxygen extraction ratio. Nitric oxide (NO) maintains microvascular homeostasis by regulating arteriolar tone, RBC deformability, leukocyte and platelet adhesion to endothelial cells, and blood volume. NO also regulates mitochondrial respiration. During sepsis, NO overproduction mediates systemic hypotension and microvascular reactivity, and is seemingly protective of microvascular blood flow. [**Crit Care. 2003 Oct;7(5):359-73. Epub 2003 Jul 28].**PMID:12974969

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