leukocytes that produce reactive oxygen species. With an injection of antineutrophil serum, we reduced the number of circulating neutrophils (and also the total number of leukocytes) to one-third the value normally found, then measured and correlated capillary $V_{\text{RBC}}$ as a function of arteriovenular pairing (11). This treatment was highly effective in preventing arteriolar constriction, increasing capillary perfusion, and restoring the correlation between perfusion and venular pairing to nearly the same as that observed in normocholesterolemic rats. We are optimistic that similar protocols may be found to be effective with other cardiovascular risk factors.

Summary

In conclusion, capillary filtration and perfusion appear to be dependent on communication between venules and closely paired arterioles. In a rat mesenteric model, this communication is altered with inflammation: venular accumulation of leukocytes increases capillary filtration through an arteriolar signaling pathway while at the same time constricting these arterioles to limit capillary perfusion. Restricted capillary flow in the mesentery also accompanies the inflammation present with cardiovascular risk factors such as hypercholesterolemia; however, treatments aimed at restoring arteriovenular communication in this model are successful in improving capillary perfusion.

I thank Robert Hester for helpful comments and proofreading.

Funding for my continued research on this topic is currently supplied by The Whitaker Foundation.

References


In Forthcoming Issue

Mitochondrial Regulation of Apoptosis
Bernd Mayer and Rainer Oberbauer

Nonredundant Gap Junction Functions
Thomas W. White

New Roles for Connexons
Lisa Ebihara

Immune Adherence Revisited: Novel Players in an Old Game
Christoph Hess and Jürg A. Schifferli