Seminar/Talk

Neurovascular coupling in health and disease.

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Host: Simon Hippenmeyer

Neuronal activity results in a spatially and temporally localized increase in blood flow to meet the increased demand for oxygen and glucose. This response, termed neurovascular coupling, was classically thought to occur entirely at the arteriole level and mediated by astrocytes. Challenging this view, our work demonstrates that cerebral blood flow can also be controlled at the capillary level. I will present data showing that neuronal activity evokes capillary dilation both ex vivo and in vivo. Capillary dilation depends on calcium entry into astrocytes through the purinergic P2X1 receptors and the subsequent synthesis and release of vasoactive prostaglandin E2. Arteriole dilation, in contrast, does not depend on astrocytic signaling but rather is mediated by nitric oxide, most likely from interneurons. Using an experimental model of stroke, we also show that activity-evoked capillary dilation is reduced by ~75% in the clinically asymptomatic, non-ischemic regions of the stroke hemisphere, replicating clinical observations in stroke patients. Such a reduction in capillary neurovascular coupling would spell a severe energy deprivation state and may contribute to long-term neurological dysfunction. These findings demonstrate a dichotomy in the signaling cascades that regulate cerebral blood flow in different vascular compartments, establish astrocytes as an important mediator of neurovascular signaling to capillaries, and suggest restoration of astrocyte-dependent capillary regulation as a novel putative therapeutic approach in stroke. Our findings also suggest that blood oxygenation level dependent neuroimaging may not be a reliable measure of neuronal function in stroke patients.

Friday, May 17, 2019 11:00am - 12:00pm
Seminar Room, Lab Building East

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