Neural stimulation of cerebral blood flow in health and hypertension

Schematic illustration showing the mechanism of functional hyperemia deficits in hypertensive small vessel diseases. Stroking mouse's vibrissae (whisker stimulation) results in capillary endothelial cell inward-rectifier potassium Kir2.1 channel activation due to astrocytic and neuronal release of K+. By virtue of the activation of Kir2.1 channels and subsequent membrane hyperpolarization, hyperpolarizing signal rapidly propagates upstream, retrograde to flow, dilates the precapillary arteriole, and increases downstream tissue perfusion. This local increase in blood flow in the neuronally active region of the brain is termed functional hyperemia, which is detected using laser Doppler flowmetry in this study. Chronic hypertension disrupts the functional hyperemia in response to whisker stimulation by the mechanism causing the Kir2.1 channel dysfunction.