Endothelial Dysfunction and Hypertension in Aging

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Reduced nitric oxide (NO) bioavailability and reduced number and function of endothelial progenitor cells (EPCs) by reactive oxygen species (ROS) and endothelial cell senescence may contribute to endothelial dysfunction in aging. In hypertension also, similar mechanisms may work in the process of endothelial dysfunction. Aging, aging-associated hypertension, and hypertension per se, either independently or collectively, impair endothelial function, leading to atherosclerosis, resulting in cardiovascular and cerebrovascular outcomes. Improvement or augmentation of endothelial function will prevent the development of atherosclerosis and reduce cardiovascular events. Intervention to reduce oxidative stress should be an effective strategy for treatment of atherosclerosis, including aging and hypertension, through, at least in part, improvement in endothelial function and endothelial cell senescence. In this session, I focus on recent findings and interactions between endothelial function, oxidative stress, and hypertension in aging.