Alteration in flow (shear stress)-induced remodelling in rat resistance arteries with aging: improvement by a treatment with hydralazine

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Résumé en anglais

AIMS: The link between aging and vascular diseases remains unclear, especially in resistance arteries. As a decreased vasodilator capacity of the endothelium is usually described in aging, we hypothesized that arteriolar remodelling in response to a chronic increase in blood flow might be altered. In addition, we tested the capacity of a vasodilator treatment with hydralazine to restore remodelling, as we have previously shown that hydralazine has a potent effect on the process.
METHODS AND RESULTS: Mesenteric resistance arteries (350 microm diameter) from 3- and 24-month-old rats were exposed to high blood flow (HF) and normal blood flow (NF), for 2 weeks by sequential ligating second-order arteries in vivo. In HF arteries, diameter increased by 21% when intraluminal pressure was 100 mmHg, in association with a rise in superoxide production in young rats. On the other hand, both diameter and superoxide levels failed to increase in old rats. Hydralazine restored HF-induced remodelling in old rats in association with an increased superoxide production and a decreased superoxide dismutase (SOD) expression. The SOD-mimetic 4-hydroxy-2,2,6,6-tetramethyl piperidinoxyl (TEMPOL) prevented the effect of hydralazine on the arterial diameter. In old rats, hydralazine increased the arterial diameter in HF arteries without increasing eNOS expression. Furthermore, hydralazine also restored HF remodelling in eNOS knockout mice.
CONCLUSION: Thus, flow remodelling in resistance arteries failed to occur in aging but it could be restored by hydralazine via a reactive oxygen species-dependent mechanism. These findings may have serious pathophysiological consequences in situations requiring flow-dependent remodelling such as ischaemic and metabolic diseases, more frequent in the elderly.

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