In vitro protection of vascular function from oxidative stress and inflammation by pulsatility in resistance arteries

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Résumé en anglais	ObjectiveResistance arteries remain subject to pulsatility, a potent regulator of large elastic artery tone and structure, but the effect is incompletely understood. Extracorporeal circulation during cardiac surgery is often associated with absence of pulsatility, which may affect vascular tone. To define the role of the vascular wall in the inflammatory process that may occur with or without pulsatility, we studied resistance arteries functions ex vivo. We measured vascular reactivity, oxidative stress, and inflammation in the arterial wall. Methods Isolated rat mesenteric resistance arteries were mounted in an arteriograph and subjected to pulsatility or not in vitro. Arteries were perfused with a physiologic salt solution without circulating cells. Results After 180 minutes, flow-mediated dilation was higher and pressure-induced myogenic tone lower in arteries subjected to pulsatility. Without pulsatility, reactive oxygen species and markers of inflammation (monocyte chemotactic protein 1 and tumor necrosis factor α) were higher than baseline. In perfused mesenteric beds under similar conditions, tumor necrosis factor α was higher in perfusate after 180 minutes of nonpulsatility (5.7 ± 1.6 pg/mL vs 1.1 ± 0.4 pg/mL; $P < .01$). In arteries treated with the antioxidant 4-hydroxy-2,2,6,6-tetramethylpiperidin-1-oxyl (tempol), flow-mediated dilation and myogenic tone were similar in nonpulsatile and pulsatile arteries; monocyte chemotactic protein 1 and nuclear factor κB expression levels were not increased in tempol-treated nonpulsatile arteries. Conclusions Absence of pulsatility in resistance arteries increased oxidative stress, which in turn induced inflammation and preferentially altered pressure and flow-dependent tone, which play a key role in control of local blood flow.
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