



4.4 Arterial Stiffness can be Modulated by Pressure-Independent Mechanisms in Hypertension

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ABSTRACT

Background: Effects of short-term interventions on large artery stiffness assessed by pulse wave velocity (PWV) have mainly been explained by concomitant changes in blood pressure (BP). However, lower body negative pressure (LBNP), which increases sympathetic activity (SA) has a specific effect on PWV in healthy volunteers [1].

Methods: We examined effects of lower limb venous occlusion (LVO), a similar intervention to LBNP that reduces BP but increases SA and device guided breathing (DBG), which reduces both BP and SA, on PWV in patients with essential hypertension ($n = 70$ after LVO, $n = 45$ after DGB and LVO in random order). The short acting calcium channel antagonist nifedipine was used as a control for changes in BP.

Results: LVO produced a small but significant reduction in mean arterial pressure of 1.8 (95% confidence-intervals, 0.3–3.4) mmHg. Despite this, aortic and carotid-femoral PWV increased during LVO by 0.8 (0.2–1.4) m/s and 0.7 (0.3–1.05) m/s respectively. When comparing effects of DGB with nifedipine ($n = 19$), nifedipine produced a greater reduction in mean arterial pressure compared to DGB (reduction of 13.4 (10.2–17.7) mmHg and 7.7 (6.4–9.0) mmHg respectively, $p = 0.009$ between the two) but the decrease in PWV was less than that observed with DGB (0.7 (0.1–1.5) m/s and 1.3 (0.8–1.8) m/s respectively, $p = 0.022$ between the two).

Conclusion: Arterial stiffness can be modulated independently of BP in patients with hypertension. The mechanism could involve alterations in SA influencing the tone of smooth muscle in the aortic wall.

REFERENCE

- [1] Nardone M, Incognito AV, Millar PJ. Evidence for pressure-independent sympathetic modulation of central pulse wave velocity. *J Am Heart Assoc* 2018;7:e007971.

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