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**The effect of arginine on postocclusive reactive hyperaemia**

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**Purpose:** Postocclusive reactive hyperaemia (PORH) is one of the provocation tests for registering differences in vasodilation capabilities in healthy and diseased subjects. One of the hypotheses of essential hypertension is that endothelial dysfunction due to reduced availability of nitric oxide plays a key role in initiation, development and progression of the disease. The aim of our study was to determine whether the ingestion of L-arginine improves PORH in subjects with a family history of hypertension.

**Methods:** 53 healthy male volunteers, from 20 to 56 years old, were divided into four groups according to the family history of hypertension and age (YN - young without a family history of hypertension, YH - young with a family history of hypertension, ON - older without and OH - older with a family history of hypertension). We measured ECG, heart rate, systolic and diastolic blood pressure, cardiac output, stroke volume, total peripheral resistance (Task Force Monitor) and laser Doppler (LD) flux in the cutaneous microvessels in the fingertip at rest and after eight minutes lasting occlusion of digital arteries. Measurements were repeated after administration of 0.9 g L-arginine. Values were expressed as mean ± SEM. The study was approved by the National Medical Ethics Committee; written informed consent was obtained from each subject.

**Results:** After the ingestion of L-arginine the heart rate (YN 67.7 ± 4.3 vs. 62.7 ± 3.3; YH 63.2 ± 2.2 vs. 60.3 ± 2.0; ON 64.7 ± 3.3 vs. 57.7 ± 2.6; OH 62.5 ± 2.9 vs. 58.1 ± 2.2 beats/min) and the cardiac output (YN 7.07 ± 0.5 vs. 6.53 ± 0.4; YH 7.19 ± 0.3 vs. 6.38 ± 0.2; ON 7.7 ± 0.5 vs. 6.7 ± 0.4; OH 6.4 ± 0.5 vs. 5.9 ± 0.4 L/min) statistically significantly decreased in all groups (paired t-test, p < 0.05), while SBP and DBP and LD flux did not change. In contrast, stroke volume decreased (116.0 ± 6.5 vs. 108.2 ± 5.9 mL) and total peripheral resistance increased (868.9 ± 96 vs. 1114.4 ± 48 MPa.s/m²) only in YH group (paired t-test, p < 0.05). PORH remained the same as before ingestion of L-arginine except in the ON group, where it statistically significantly decreased (area and maximal LD flux; paired t-test, p < 0.05).

**Conclusions:** After ingestion of L-arginine we observed decreased heart rate and cardiac output in all tested groups. LD flux during PORH decreased only in the group of older subjects without a family history of hypertension. These results support the view that the PORH is largely independent of NO production.