Nervous System

10.4 Acute Administration of Vitamin C Decreases Sympathetic Nervous Activity and Blood Pressure in Hypertensive Patients

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Introduction. Essential hypertension is characterized by both increased oxidative stress and sympathetic nervous system (SNS) hyperactivity. Experimental studies suggest that increased oxidative stress contributes to the development of hypertension through activation of the SNS.

Aim. To determine whether an acute administration of the antioxidant vitamin C can influence SNS activity in hypertensive patients.

Methods. Fifteen never treated, essential hypertensive patients were included. Heart rate (HR), non-invasive beat-to-beat blood pressure (BP, Portapres) and muscle sympathetic nervous activity (mSNA, microneurography) were monitored in basal conditions and until 20 minutes after infusion of vitamin C (3 g iv in 5 minutes). Venous samples for assessment of norepinephrine (NE), oxidative stress (malonyldialdehyde, MDA) and antioxidant capacity (ferric reducing ability of plasma, FRAP) were obtained before and at the end of recording. HR, BP and mSNA values were analyzed as means of 5 minutes at baseline and between 16'-20' after infusion and expressed as mean $\pm$ SEM.

Results. Vitamin C infusion significantly reduced oxidative stress (MDA from 5.4 ± 0.8 to 4.6 ± 0.6 micromol/L, $p < 0.05$) increased plasma antioxidant capacity (FRAP from 838 ± 88 to 2531 ± 230 micromol/L, $p < 0.05$). Moreover, vitamin C infusion was associated with a significant reduction in BP (systolic BP from 142.1 ± 5.2 to 138.0 ± 5.7 mmHg, $p < 0.05$, -3.0%; diastolic BP from 92.4 ± 3.3 to 85.6 ± 3.4 mmHg, $p < 0.01$, -7.0%; mean BP from 110.5 ± 3.8 to 104.6 ± 4.1 mmHg, $p = 0.01$, -5.3%). mSNA was significantly reduced (from 32.9 ± 2.9 to 29.4 ± 2.8 bursts/min, $p = 0.01$, -10.7%; from 52.1 ± 3.6 to 46.3 ± 3.7 bursts/100 heart beats, $p < 0.01$, -11.3%). NE and HR were unchanged.

Conclusions. Acute administration of vitamin C is associated with reduced BP and mSNA in hypertensive patients. These preliminary results suggest that oxidative stress may influence the sympathetic hyperactivity characterizing essential hypertension.