Alpha-Adrenergic Mechanisms in the Cardiovascular Hyperreactivity to Norepinephrine Infusion in Essential Hypertension

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Aims: Essential hypertension (EHT) is characterized by cardiovascular hyperreactivity to stress but underlying mechanism are not fully understood. Here, we investigated the role of α-adrenergic receptors (α-AR) in the cardiovascular reactivity to a norepinephrine (NE)-stress reactivity-mimicking NE-infusion in essential hypertensive individuals (HT) as compared to normotensive individuals (NT).

Methods: 24 male HT and 24 male NT participated in three experimental trials on three separate days with a 1-min infusion followed by a 15-min infusion. Trials varied in infusion-substances: placebo saline (Sal)-infusions (trial-1:Sal+Sal), NE-infusion without (trial-2:Sal+NE) or with non-selective α-AR blockade by phentolamine (PHE) (trial-3:PHE+NE). NE-infusion dosage (5μg/ml/min) and duration were chosen to mimic duration and physiological effects of NE-release in reaction to established stress induction protocols. We repeatedly measured systolic (SBP) and diastolic blood pressure (DBP) as well as heart rate before, during, and after infusions.

Results: SBP and DBP reactivity to the three infusion-trials differed between HT and NT (p's$\leq$.014). HT exhibited greater BP reactivity to NE-infusion alone compared to NT (trial-2-vs-trial-1: p's$\leq$.033). Group differences in DBP reactivity to NE disappeared with prior PHE blockade (trial-3: p=.26), while SBP reactivity differences remained (trial-3: p=.016). Heart rate reactivity to infusion-trials did not differ between HT and NT (p=.73).

Conclusion: Our findings suggest a mediating role of α-AR in DBP hyperreactivity to NE-infusion in EHT. However, in SBP hyperreactivity to NE-infusion in EHT, the functioning of α-AR seems impaired suggesting that the SBP hyperreactivity in hypertension is not mediated by α-AR.

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