Involvement of α-1A Adrenoreceptors in Mediating the Neurogenic Vasoconstriction of the Renal Cortical Microcirculatory Vascular Beds in Normotensive WKY and SHR Rats with High Salt Load

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The contribution of alpha1A adrenoreceptors to neurogenic constriction of the renal cortical resistance vessels of the normotensive WKY and SHR rats were assessed in animals subjected to high salt load (0.9% saline) for six weeks. The metabolic data collected that include body weight, water intake, urine output, collection of blood for urinary and plasma sodium analysis was obtained every week. At the end of the six weeks the rats were anesthetized (60 mg/kg i.p. sodium pentobarbitone) and subjected to hemodynamic study. The reductions in the renal blood flow caused by the adrenergic agonist noradrenaline, phenylephrine and methoxamine were assessed before and after 5-Methylurapidil administration an alpha1A adrenergic antagonist. Data were recorded in a computerized data acquisition system, expressed as mean ± s.e.m, and compared by 2-way ANOVA followed by Bonferroni post-hoc test with a significance level at 5%. Result obtained showed significant increase in the water intake, diuresis and natriuresis. The renal hemodynamic responses to adrenergic stimuli showed a dose dependent acute (within 3 min) transient increase in renal resistance to noradrenalin, phenylephrine and methoxamine in renal cortical blood flow in high salt load and control (0.9% + normal diet) WKY and SHR rats. The increase in renal vascular resistance was more enhanced for phenylephrine and methoxamine in the WKY rats and only for methoxamine in SHR with high salt load compared to the control group. The constriction of the renal vasculature in vivo are attenuated by 5-Methylurapidil which preferentially block the alpha1A adrenoreceptors. These observations implicate mediation and up regulation of the alpha1A adrenoreceptors in the renal cortical microcirculatory resistance vessels in the normotensive and hypertensive rats with high salt load compared to the control counter part.