

Role of the renal sympathetic nerve activity in determining salt sensitivity.

Hiroshi Kannan, Kazuo Kato*, Takato Kunitake and Takamitsu Hanamori
Department of Physiology, Department of Medicine*
Miyazaki Medical College

Summary

Dahl salt-sensitive (DS) rats develop hypertension when fed a high-salt diet, but remain normotensive on a low-salt diet. The mechanisms underlying salt-dependent hypertension are not entirely known, although several abnormalities in DS have been proposed to be of pathogenic importance; involvement of the kidneys, including abnormal renal excretory and humoral mechanisms, is one such suggested contributory factor. Renal sympathetic nerve activity (RSNA) has a direct effect on the control of renal blood flow, renin release and urinary sodium excretion. We have recorded RSNA in free-moving Dahl-rats on a low NaCl diet to characterize the RSNA discharge pattern and evaluate the RSNA response to intracerebroventricular (i.c.v.) administration of hypertonic sodium chloride. Under resting conditions, two types of discharge patterns were seen, a grouped cardiac-related discharge (GD) and a non-grouped irregular discharge (NGD). The GD was inhibited by i.v. administration of phenylephrine chloride, while the NGD was not. Both GD and NGD were seen in DS and Dahl-salt-resistant (DR) rats and both were completely abolished by i.v. administration of the ganglionic blocker, hexamethonium chloride, indicating postganglionic efferent nerve involvement. I.c.v. infusion of 0.3 M NaCl at 1 μ l/min for 20 min significantly increased arterial blood pressure and decreased RSNA compared with the resting levels in both Dahl-R and Dahl-S rats. Heart rate did not significantly change. However, the RSNA response to 0.3 M NaCl injection was not significantly different from the response to 0.15 M NaCl injection in Dahl-S rats, while the response in Dahl-R rats remained significant. During 0.3 M NaCl infusion, water intake in Dahl-S rats was diminished compared with that in Dahl-R-rats. In addition, baroreflex control of heart rate did not differ in either group. These findings support the hypothesis that salt-sensitive hypertension is due to the failure to suppress RSNA in response to salt loading, and suggest the importance of recording with a high signal-to-noise ratio to evaluate autonomic nervous system activity.