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The Interaction between the Gain of Water Drink-Induced Pressor Response and the Incidence of Aarrhythmia in Dahl Salt-Sensitive Rats

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Summary

Water drinking is known to induce the pressor response in animals and human subjects. The pressor response was observed at the onset of drinking, and then smoothly returned to the baseline level after the cessation of drinking in rats. The pressor response was enhanced by the decrease in arterial barorefelx gain. Although sympathoexcitation is known to be participated in the efferent, the afferent mechanism has not been clarified. Accordingly, the first purpose of the present study was to examine the afferent mechanism in water drinking-induced pressor response using sinoaortic denervated (SAD) rats. Furthermore, if mean arterial pressure (AP) was over 200 mmHg during water drinking, the bradyarrhythmia was observed. In case the gain of water drinking-induced pressor response was not altered, the baseline AP might influence to the occurrence of bradyarrhythmia. Thus, the second purpose was to examine the mechanism of bradyarrhythmia during water drinking in Dahl Salt-Sensitive hypertensive rats (Dahl-S) with SAD. The pressor response was not suppressed by following method; 1) transient oral surface anesthesia using lidocaine, 2) denervation of both glossopharyngeal nerves and afferent superior laryngeal nerves, and 3) denervation of tunica adventitia of the esophagus. However, the pressor response was significantly suppressed (-52%) by gadolinium chloride administration. Furthermore, electrical stimulation of the hypoglossal nerve induced the pressor response, and it was significantly suppressed (-57%) by gadolinium chloride administration and completely abolished by severance of the distal end of this nerve. Thus, the afferent signals from the mechanoreceptor in the drinking-related muscles were involved in the water drinking-induced pressor response. The bradyarrhythmia was observed in Dahl-S but not in Dahl Salt-Resistance rats (Dahl-R). Since this bradyarrhythmia was completely abolished by atropine administration, vagotonic response participated in bradyarrhythmia during water drinking. Although the mean water drinking-induced pressor response was significantly suppressed in Dahl-S compared with Dahl-R (44 ± 3 mmHg vs 71 ± 3 mmHg), the baseline AP was significantly high (158 ± 6 mmHg vs 105 ± 4 mmHg), suggesting that the high baseline AP in Dahl-S rats might be important rather than the gain of water drinking-induced pressor response to occure the bradyarrhythmia during water drinking.