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Influence of Brain Sodium Concentration for the Washout of Waste Products from Brain and Onset of Frail.
Subtitle: Stroke Onset and Sarcopenia by Brain Sodium

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Summary

Background and purpose: High sodium intake induces hypertension, resulting increase of stroke onset. However, it is undetermined whether high concentration of brain sodium is cytotoxic and provides enhancement of frail including stroke onset and sarcopenia. We herein examined the role of brain sodium on the stroke onset and sarcopenia using hypertensive rats.

Methods: Eleven-week-old male spontaneously hypertensive stroke-prone rats (SHRSP) were divided into 1) artificial cerebrospinal fluid (artcereb) injection group (art, n=10) and saline injection group (n=30). The SHRSP in the latter group were further assigned to the following 3 groups; 1) 0.9% saline injection group (S, n=10), 2) 2.7% saline injection group (low-S, n=10), 3) 9% saline injection group (high-S, n=10). Continuous intracerebroventriculr administration with those 4 solutions was performed using osmotic minipump (Alzet model 2004) and the brain infusion kit. The animals were monitored stroke onset and mortality every day for 28 days. In addition, they were evaluated body weight, blood pressure, coordination and motor activity by rotarod and beam walking test, stroke related symptom by symptom score in appropriate time points. Then, we evaluated their cortical vascular morphology, vasoreactivity by administration of acetazolaminde, weight of brain and lower limb muscle, and brain water content at 28 days after the administration.

Results: Although body weight in the art group was increased through 28 days, that in the saline injection groups was not changed. The value of blood pressure in saline injection groups was similar, but % increase from the preinjection in high-S group was significantly elevated in comparison with that in S-group (S group, 97 $\pm$ 3%; low-S group, 100 $\pm$ 3%; high-S group, 108 $\pm$ 3%). Rotarod and beam walking test at 21 days and symptom score at 28 days were not changed among the groups. Although there were no significant changes of stroke onset and mortality among the groups, the former was frequently seen in saline injection groups compared with art group and the latter in S group was highest than that in other 3 groups. There were no significant changes in brain phenotypes such as cortical vascular morphology, vasoreactivity, brain weight, and brain water content, and also in the weight of lower limb muscle among the groups. The values of pH in saline injection groups were 5.83 in S group, 5.66 in low-S group, and 5.65 in high-S group (the value of pH in artcereb were 7.3 according to the attached document).

Conclusions: In hypertensive rats, continuous intracerebroventricular administration of saline in various concentration induced little phenotypes on stroke and sarcopenia, although the slight elevation of blood pressure in higher brain sodium concentration. Based on those result, we concluded that brain sodium provided relatively weak effect on hypertensive organ dysfunction in hypertensive rats which already show significant upregulation of sympathetic nerve activity.