INTRODUCTION: In some M-SHRSP and/or SHRSP, we observed an enlargement of the head, a thinning of the skull and a marked increase in brain volume. Intracranial pressure appeared to be higher than normal and to be a cause for death in such rats. This study was conducted in order to obtain basic research data on the relationship between hypertension and intracranial hypertension.

MATERIALS AND METHODS: Blood pressures in M-SHRSP (Okamoto, K. et al. Hypertensive Mechanisms, Proc. Fourth Int. Symp. on Spontaneously Hypertensive Rats and Related Studies, pp. 143-146, F.K. Schattauer Verlag, Stuttgart-New York, 1982), SHRSP and WKY at the age of 6 to 25 weeks were measured using the tail-pulse pickup method without anesthesia. Following measurement, the rats were locally anesthetized with a subcutaneous injection of 1% lidocaine, and a 25-gauge needle joined to a polyethylene catheter was inserted into the cisterna magna. The polyethylene catheters were attached to identically calibrated pressure transducers (Toyo Baldwin), and cerebrospinal fluid pressures were recorded on polygraph (Saneisokki). Cerebral vascular permeability was demonstrated using Evans blue method (Masaki, S. Experimental and Molecular Pathology, 2, 330-337, 1978) and Graham and Karnovsky’s peroxidase methods (Graham, R.C. et al. J. Histochem. Cytochem. 14, 291-302, 1966). The rats were intravenously injected first with 1% Evans blue (1ml/kg body weight), then with horseradish peroxidase type II (100mg/kg body weight) fifteen minutes later. They were sacrificed an additional fifteen minutes later. The existence of cerebellar herniation was observed. The lengths between the under edge of the occipital bone and the cerebellar end were measured using slide calipers. Cerebellar herniation was considered to exist if this length was 1.5 mm or more. The brains were carefully removed for macroscopical and/or microscopical observation of dye leakage.

RESULTS AND DISCUSSION: 1) The cerebrospinal fluid pressures in M-SHRSP and SHRSP with blood pressures under 210mmHg was the same as in WKY (average 6.5 cmH2O), but were proportionally elevated to between 11.6 and 25.3 cmH2O for blood pressures between 210mmHg and 270mmHg (figure). 2) Hyperpermeability of the brain vessels and increases in cerebrospinal fluid pressures were found in some M-SHRSP and SHRSP with blood pressures were over 210 mmHg. The incidence of brain vessel hyperpermeability also accelerated in accord with increases in blood pressures. 3) Cerebellar herniations resulting from increased brain volume (perhaps due to brain edema) and brain vessel hyperpermeability occurred in rats with high blood pressures. The incidence of cerebellar herniation increased conjunction with elevations in blood pressures, and was higher than the incidence of stroke lesions. It is assumed that severe brain edema, cerebellar herniation and increased cerebrospinal fluid pressures were the cause of death in these M-SHRSP and/or SHRSP. 4) Cerebrospinal fluid pressures of SHRSP were not greatly affected, at least within the first thirty minutes, by artificial acute elevation and/or reduction in blood pressures induced by injection of noradrenaline or apresoline.