32) Blood-CSF barrier in stroke-prone SHR. Motoki Tagami*, Akiyoshi Kubota*, Toshiaki Sunaga, Hidenori Maezawa, Masahiro Kihara, Yasuo Nara and Yukio Yamori. * Department of Internal Medicine, Sanraku Hospital, Tokyo 101.

Davis et al have shown that the choroidal epithelium is a morphologically distinct blood-brain barrier that prevents the entry of proteins into the cerebrospinal fluid (CSF). Concerning choroid plexus uptake from the cerebrospinal side information is sparse and conflicting. In the present study we have used SHRSP and have tried to clarify the influence of hypertension toward the barrier function of the choroid plexus epithelium.

Materials and Methods: Twenty two SHRSP from 8 to 24 weeks of age were used. Twenty WKY served as controls. Horseradish peroxidase (Sigma Type II: HRP) was used as tracer molecules. Fifteen mg/100g body weight of HRP were injected into the femoral vein. The choroid plexus were perfused with a fixative through the femoral artery, and were then dissected. An inflow cannula was placed in each lateral ventricle of 3 SHRSP at the age of 8 weeks and 3 SHRSP at the age of 16 weeks. Ten μ ml of 0.5 % HRP were injected into the lateral ventricle through the cannula. The choroid plexus were then perfused with a fixative through the femoral artery.

Results: (1) Intravenous injection study of HRP.

One min after the intravenous injection of HRP, the tracer appeared in the following sites within the choroid plexus: (a) within capillary lumina, (b) in the perivascular area surrounding the capillaries, (c) in the extracellular space between epithelial cells, (d) in the basal foldings of the epithelial cells.

Three min after the injection, the tracer was found in the pinocytotic pits and vesicles of the lateral and basal surfaces. Moreover endocytic vacuoles were observed adjacent to the pinocytotic vesicles. The vesicles sometimes fused with the endocytic vacuoles.

Examination of the choroid plexus between 15 min and 30 min after the injection of HRP demonstrated an apparent increase in the uptake of the tracer by the pinocytotic vesicles. Although the pinocytotic vesicles and the multivesicular bodies were randomly distributed throughout the cytoplasm, they were generally found in greater concentration near the apical surface. HRP-filled vesicles frequently showed a raindrop-like formation and were found in juxtaposition with the apical cytoplasmic membranes of the epithelium. In some instances the HRP-filled vesicles fused with the apical cytoplasmic membranes. Around these sites HRP was found within the dense bodies of the phagocytes which adhered to the apical microvilli of the epithelial cells. These findings were common to WKY and young SHRSP.

In SHRSP over 16 weeks of age HRP-filled vesicles frequently fused with the apical cytoplasmic membrane of the epithelium. Moreover degenerate epithelial cells were observed in some instances. The degenerate cells were filled with enormous amounts of HRP. HRP was also evident around the apical microvilli. Several phagocytes, which lay scattered in the cerebral ventricle, trapped great amounts of HRP around these sites.

(2) Cerebro-ventricular injection study of HRP.

Five min after cerebro-ventricular injection of HRP, the tracer was trapped by the pinocytotic vesicles of the apical epithelial surface. HRP was carried into the epithelial cells by the vesicles. Thereafter the vesicles fused with the endocytic vacuoles. The number of HRP-labeled vesicles in the cytoplasm increased considerably at 15 and 30 min after HRP injection. HRP labeling was distinct in many vacuoles, multivesicular bodies and some dense bodies in the cytoplasm. HRP was transported to the extracellular spaces of the endothelial cells, below the apical tight junctions, by the pinocytotic vesicles.

Conclusion: (1) The blood-CSF barrier is not as total as has been indicated in other studies.

(2) The choroid plexus epithelium seemed to maintain a constant intraventricular environment.