20) Pathogenesis of Cerebral Cystic Infarction Observed in SHR-SP. Kenro Takiguchi, Junichi Miyazaki, Masao Kishikawa, Issei Nishimori, and *Masayori Ozaki. Department of Pathology, Atomic Disease Institute, and *Second Department of Pharmacology, Nagasaki University School of Medicine, Nagasaki 852.

The cerebrovascular lesions such as the hemorrhage and the infarction of the stroke-prone spontaneously hypertensive rats (SHR-SP) have been reported in various journals. There exist many controversies regarding their resemblance to the cerebrovascular lesions of human essential hypertension. We attempted to investigate the histogenesis of the large cystic infarction of the SHR-SP. Three dimensional analysis of the cerebral infarctions of the SHR-SP and of human essential hypertension was carried out using serial sections of their brains.

Materials and Methods.

A 6-month-old male SHR-SP and an autopsy case of human malignant hypertension (a male of 43-year-old) were used in this investigation. Complete serial sections, having thickness of 6 microns, of their paraffin embedded tissues were prepared for light microscopy. Sections were usually stained with hematoxylin and eosin, and, if necessary, with Azan-Mallory and phosphotungstic acid hematoxylin staining.

Results and Discussion.

A region of a large cystic infarction of the SHR-SP was found to correspond to an area containing an assembly of small infarctions at the level of adjacent sections, indicating that a large cystic infarction was formed from an assembly of small infarctions. The conspicuous fibrinoid necroses, thrombi, and perivascular necroses were detected in the supplying arterioles and capillaries. On the contrary, the major cerebral arteries at the base of the brain were preserved and did not show any athero- or arteriosclerotic change or thrombus formation. It was thought that these small infarctions of the SHR-SP resulted from fibrinoid necroses and thrombi of such small vessels and that the fusion of the small infarction formed one large cystic infarction.

In the case of human malignant hypertension, essentially the same changes as in the SHR-SP were observed. From the observations described above, it was concluded that the cerebrovascular lesions of SHR-SP differed from those of human essential hypertension in which infarction was rarely seen and infarction of SHR-SP, which was based on the fibrinoid necroses and thrombi of arterioles and capillaries (20-30 μ), was considered to be similar to that of human malignant hypertension in the etiology and the morphology.