(SI) and CSR(%), that is, cases with low SI have high CSR. This fact shows that there is a compensating mechanism for ceerbral circulation. This fact is clear in cases with cardiac diseases, especially with valvular diseases. On the other hand, in cases with cerebral thrombosis or cerebral vascular insufficiency, distribution of SI and CSR is very wide, and low CSR is not detectable. We would like to think that cerebral thrombosis is partial change in brain, and compensating mechanism is active in cases with cerebral vascular insufficiency. In cases with hypertension and/or arteriosclerosis, wide distribution of SI and CSR is observed. We would like to say this group contains the patients in various stage of diseases.

(Conclusion) We find necessity to think cerebral circulation in connection with cardiac function, and our new method is useful in this point. Furthermore, this test gives few trouble to the subjects.

The Elasticity of the Intracranial CSF Volume

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Generally speaking, the intracranial cavity is inelastic except in infancy, because it is surrounded by inelastic hard skull. We devided the intracranial volume (V) into three compartments as follows: brain volume (VBr), brain blood volume (VBl) and intracranial CSF volume (VCSF).

We got a fomula, $V = VBr + VBl + VCSF$.

The total CSF volume is composed of intracranial CSF volume and the extracranial CSF volume. Volume changes in the brain tissue are slow but that in the brain blood pool are quite rapid. The spinal canal has more elasticity than the intracranial cavity.

It is conceivable that the intracranial CSF volume changes rapidly according to the changes in the brain blood pool volume.

We studied the rapid volume changes in the cranial blood pool and intracranial CSF volume with nondiffusible radio isotope.

A dose of ^{131}I-RIHSA (500 microcuries) was injected intravenously after brocking of thyroid glands. The total cranial counts were recorded by renogram for thirty minutes. Ten minutes after injection, cervical veins were compressed manually for one minute. After the total cranial counts regained the counts before cervical compression and became stable, we observed the effects of three minutes hyperventilation on the total cranial counts.

Other patients who had indication of gamma cysternography were administered a dose of ^{131}I-RIHSA (100 microcuries) intrathecally. Three hours after intrathecal injection of ^{131}I-RIHSA, the effects of cervical compression and three minutes hyperventilation on the total cranial counts were observed.

We found that the cranial blood volume increased by 10% and the intracranial CSF volume decreased by the same with the compression of cervical veins. The cranial blood volume decreased by 7% and the intracranial CSF volume increased by the same with the hyperventilation.