This simple and atraumatic testing of the cerebral circulation will become more useful and accurate, if these analytical methods are adopted.

g-7. Experimental Study of Regional Blood Flow in Increased and Non-increased Intracranial Pressure

—Regional Blood Flow in Hemorrhagic Shock under Hypercapnia and Hypocapnia—

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The experiment was performed about two groups, (A) the non-increased intracranial pressure and (B) the increased intracranial pressure, using 17 dogs under the intravenous anesthesia with Nembutal. The effects of the acute blood loss under the hypercapnia and the hypocapnia on the regional blood flows were investigated. The determination of the blood flows at the internal carotid artery, the superior mesenteric artery, the renal artery and the peripheral artery (mainly femoral artery) was used of the electromagnetic flowmeter.

(A) Non-increased intracranial pressure group

On the condition of the 15% CO₂, 40% O₂, 45% N₂ gas inhalation (PaCO₂ 130–200 mm Hg), the blood pressure rose and each regional blood flows increased. The internal carotid arterial blood flow (ICBF) increased more than twice, but the renal arterial blood flow (RBF) scarcely changed, while the superior mesenteric arterial blood flow (MBF) and the femoral arterial blood flow (FBF) were kept between ICBF to RBF. On such condition, the oligemic shock was made by the femoral bleeding. In that cases, ICBF decreased in proportion to the mean blood pressure, but the decreasing of RBF was a little. When the mean blood pressure fell less than about 70 mmHg, RBF decreased remarkably but ICBF did not so changed, while the reduction of MBF and FBF was throughout more proportional to the mean blood pressure.

On the condition of the forcefull hyperventilation with 50% O₂, 50% N₂ gas (PaCO₂ 16–25 mmHg), the blood pressure fell and each regional blood flows decreased; ICBF decreased to 50–60% but RBF a little. MBF and FBF were kept between ICBF to RBF. On that condition, the oligemic shock was made. In that cases, MBF and FBF decreased in proportion to the blood pressure but ICBF did not decreased or scarcely in its early stage. When the mean blood pressure fell less than 50–55 mmHg, ICBF began to decrease and RBF decreased remarkably. In severe hypotension, ICBF was kept in comparison with the decrease of the blood flows of the other organs.

In the stage of the critical shock, ICBF was not so changed by the hyper-

capnia but was reduced remarkably by the hypocapnia.

(B) Increased intracranial pressure group

The increasing of the intracranial pressure was made by the epidural ballooning. Each blood flows were reduced in the increased intracranial pressure; RBF and ICBF decreased more than MBF and FBF.

At 1000 mmH₂O balloon pressure, the effects of the acute hemorrhage under the hypercapnia and the hypocapnia on the blood flows showed the same tendency to the results of the non-increased intracranial pressure group. But ICBF under the hypercapnia was not increased so remarkably as that of the non-increased intracranial pressure group. On the other hand, the reduction of ICBF under the hypocapnia was as almost same as that of the non-increased intracranial pressure group.

g-8. Cerebral Circulation and Intracranial Hypertension

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Our previous investigations of the pathophysiology of intracranial hypertension have indicated that various secondary pathophysiological changes do not always parallel to the degree of the pressure elevation but are influenced both by degree and duration of the increased intracranial pressure. Also, we have pointed out that the secondary circulatory change due to increased intracranial pressure plays an important role in further elevation of intracranial pressure. The present experimental study was made in an attempt to clarify the circulatory changes under increased intracranial pressure with special regard to time consequence. Also, it was our intension to find out which part of the cerebral circulatory system is most influenced by the intracranial hypertension.

The experiment was carried out on 22 adult mongrel cats. While measuring intracranial pressure continuously, an extradurally placed rubber balloon was inflated to elevate intracranial pressure. The pressure was elevated to various levels between 300–2,000 mmH₂O and the same pressure was maintained for various length of time between 30 minutes to 12 hours. Rapid serial photographs of the cortical vessels were taken through a glass window tightly placed over the parietal area following to injection of sodium fluorescein 5 mg/kg.B.W. into inferior vena cava. The series of photographs were taken right before and right after the elevation of the intracranial pressure, and were repeated every 3 hours thereafter. Before the pressure was elevated the dye appears in cortical arteries of approximately 100–300 μ in diameter within 8 seconds after injection of the dye. Within 4 seconds, the dye flows into arterioles and it advances to venule before it disappears from the arterioles. The dye disappears from veins of