approximately 100–300 μ in diameter within 5 seconds following its appearance in the veins. Unless the pressure was elevated to extremely high level (900–2,000 mmH₂O), the dye appears in the cortical arteries without delay. However, when the pressure reaches to the high level, it delays markedly or sometimes does not appear at all. When the moderate degree of pressure (400–700 mmH₂O) is maintained longer than 6 hours, the dye appears in the venule after it completely disappears from the arterioles. Disappearance of the dye from the veins of above mentioned size is delayed whether the pressure is markedly elevated or after the moderate pressure is maintained certain period of time. No circulatory change is seen when the pressure is below 400 mmH₂O. We feel it is important to note that inflow into the cerebral arterial system is not influenced by moderately increased intracranial pressure but it mainly dependent to the pressure difference between the intracranial pressure and systemic blood pressure. We also like to stress that the circulatory disturbance does not become prominent right after the pressure is elevated but it progressively become prominent as the same pressure is maintained, and this circulatory change seems to occur mainly either at capillary level or on the venous side of the cerebral circulation.

Thus, the retardation of intracranial blood flow causes increase in cerebral blood volume and as the result, the intracranial pressure is further elevated.

g-9. Cerebral Blood Flow and Acute Increased Intracranial Pressure, especially on Luxurious Cerebral Perfusion after Decompression

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g-10. Effect of Increased Intracranial Pressure on Cerebral Hemodynamics

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In order to investigate the mechanisms whereby the increased intracranial pressure produces arterial hypertension, that is, Cushing phenomenon, studies from the cerebral hemodynamic point of view were carried out on adult mongrel dogs. Serial recordings of respiration, systemic blood pressure, cerebral blood flow through the cerebral cortex and brain stem, intracranial pressure and EEG

were performed on a polygraph. A balloon was placed and expanded gradually either in the supratentorial space or in some instances in the posterior fossa where the effects of pressure more directly influenced the brain stem. Cushing phenomenon associated with the inhibitory response of respiration, was more clearly observed at the earlier stage in the case of infratentorial compression, and it started with a sudden decrease in cerebral blood flow of the brain stem.

Several mechanisms have been proposed to explain the etiology of Cushing phenomenon; namely, the medullary anoxia, mechanical baroreceptor and axial distortion of the brain stem. It was concluded from the results of this experiment that the decrease in cerebral blood flow of the brain stem was one of the most responsible factors for the production of Cushing phenomenon.

g-11. Effects on Cerebral Circulation and Cerebral Metabolism of Various Kinds of Treatment for Head Injury

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At this meeting last year we reported the results of our experimental investigation on the effects on cerebral circulation (CBF) and cerebrospinal fluid (CSF) pressure of normal and brain edema dogs, of various drugs currently used for treatment of head injury, including 20% mannitol, thiamine tetrahydrofurfuryl disulfide (T. T. F. D.), cytochrome C, cytidine diphosphate choline (CDP-choline) and prednisolone.

This time we reported the results of our investigation on the effects of these drugs on CBF, cerebral oxygen consumption ($CMRO_2$) and CSF pressure of brain edema dogs. It was found that 20% mannitol, among those drugs, was most potent in promoting CBF and $CMRO_2$ and reducing CSF pressure. Then we tried to find the effects on CBF, etc., of the cerebral metabolism stimulating drugs after CSF pressure of brain edema dogs had been rendered approximately normal by previous infusion of 20% mannitol.

When 7 cc/kg of 20% mannitol was administered intravenously during 30 minutes, CBF and $CMRO_2$ showed a marked rise (by about 150%), together with a fall in CSF pressure during the infusion, but on finishing the infusion CBF and $CMRO_2$ showed a rapid fall for the first 15 minutes, continuing to fall gradually thereafter. Ten minutes after ending the mannitol infusion either 4 mg/kg of T. T. F. D., 0.5 mg/kg of cytochrome C, or 10 mg/kg of CDP-choline was given intravenously. Administration of T. T. F. D. caused prolongation of the period of increased CBF and $CMRO_2$, but the prolongation was only