experimental brain-edema. We presume that the improvement of abnormal E.E.G., after use of urea, would be related to circulatory factor in addition to above two factors.

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**E5. The Medical Treatment of Cerebral Edema with Steroids, Urea and Hypothermia.**

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The dogs with intracerebral bleeding were made to produce cerebral edema and high cerebrospinal fluid pressure. Steroids, urea and hypothermia were used for the treatment of resulted cerebral edema and elevated cerebrospinal fluid pressure.

Results as follows:

1. Urea in 30% solution is very effective for the decrement of elevated CSFP, clinically and experimentally. The average value of it is about 50% of the initial pressure. From histological study the effect is also shown in reduction of cerebral edema.

2. From Fay hypothermia was introduced into neurosurgical treatment. Brain volume and high CSFP are reduced. Cerebral blood flow (CBF) and cerebral O₂ consumption (CMRO₂) are said to be also reduced. This is confirmed from my study in dogs with intracerebral bleeding. Below 32°-30°C, CBF and CMRO₂ line become rather horizontal. From this, I conclude that the lower limit of the cooling must be decided between these. In normal dogs this limit exist between 30°-28°C. Therefore it is important practically that in pathological states or head injury, brain tumor etc., lower limit appears 2°-4°C higher than in normal state.

3. Premedicated dogs with dexamethasone present moderately good values in above mentioned experiments, especially in CMRO₂ and cerebral arteries venous O₂ difference (A-VO₂). Systolic blood pressure is also hold at high level than injured dogs.

4. Histological examination of the 2 groups shows remarkable difference and the application of hypothermia and steroids have a good effect for the reduction of cerebral edema.
DISCUSSION:

Experimental studies on the production and absorption of Cerebrospinal fluid under administration of Hypertonic Urea

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Some experimental results on the production and absorption of cerebrospinal fluid under administration of hypotensors (hypertonic urea, polyvinylpyrolidon and hypertonic glucose) using radioisotopes were reported in the previous meeting.

In the previous report, in short, following suggestion was made from the experimental results. The hypotonic action of urea and PVP may be ascribed to facilitation of the absorption of CSF, on the basis of the fact that the remarkable increase of the turnover of isotopes ($^{32}\text{P}$ and RIHSA) from CSF to blood was observed when these agents were given. On the other hand, remarkable inhibition of the production of CSF may be essential to the hypotensive action of hypertonic glucose.

In the experiments using rabbit on acute intracranial hypertension by means of the balloon method, initial increase of the absorption rate was observed after urea, while after PVP gradually increased hypotensive effect was noted though its slight grade. However, in the cases being applied with 50% glucose, transient decrease of the production rate followed by tendency to secondary increase was observed.

In the recent study, same experiment was carried out on the rabbits being initiated brain edema by means of the placement of the swollen balloon for 24 hours in the extradural space. The following results were obtained by the comparative estimation of production and absorption rate of CSF on the treated and non-treated rabbits with 30% urea, in such three groups as normal, initial stadium of cerebral hypertension and brain edema. (1) The production rates were decreased slightly in last two groups compared with normal animals, and then no significant shift of the production rate in all groups was observed after urea infusion. (2) The absorption rates were also inhibited in the two non-treated pathological groups, on the other hand, after urea, remarkable increase of the absorption rate reached to the normal label was noted only in the group of brain edema.

From above results, it is interesting to suppose for the clinical use of urea that the hypotensive effect of hypertonic urea may be more excellent in the state developing brain edema than the initial stadium of acute intracranial hypertension.