proternenol was also given.

From these facts, it is concluded that beta receptors in the injured skeletal muscles develop over-excitability to catecholamine (particulary adrenaline) when the muscles are injured mechanically or chemically, and that the resulting changes in the excitability of the beta receptors mentioned above may be responsible for the formation of vertigo of cervical-origin which is intimately correlated with over-excitment of the cervical proprioceptors.

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## 74. Craniocervical Injury—Impact Dynamics and the Pathological Findings

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Last year authors reported the crash experiments of monkeys and dummies. The sled, equipped with two seats for the monkey and the dummy, was dashed against the concrete barrier. At the moment of impact, the forehead of the monkey and/or dummy ran against the target on the sled.

In this subsequent report, pathological findings of the monkeys were discussed in relation to the intracranial and intraspinal pressure change.

Among the 5 monkeys (macaca fuscata), the most severely injured monkey died 40 hours after the trauma. Necreopsy revealed a coup contusion in the left frontal lobe and a contre-coup contusion in the right occipital lobe. The mechanical data of the impact were: sled velocity 31.1 km/h, head acceleration 284G, peak of occipital pressure $-1$ kg/cm$^2$ and the duration of the impact 10 msec.

Whereas no contusions were noticed in other 4 monkeys and their peak values of occiptal pressure were less than $-0.53$ kg/cm$^2$. These results should be the expemental support to the hypothesis that the cavitation occurs when the negative ripressure arrives $-1$ kg/cm$^2$, and makes brain injury.

On the other hand, there existed some concurrent pathological findings, such as "intermediary contusion" along the impact line and the brain stem hemorrhage, which must be considered to be due to the shear strain. Actually the rotational acceleration of 7500–57500 rad/sec$^2$ was recorded at the impact.

Concerning about the spinal cord, pathological findings of cervical cord were very slight. Even the cervical cord of the most severely injured monkey was affected only with edema. This findings is accordant with the result of the theoretical analysis, which shows the pressure change of cervical canal being very small at the head

impact. As the pressures of the cervical canal was not measured in the monkey, further experiments were performed using the human head dummies and the dogs with the pressure sensors. The pressure changes of the cervical canal were confirmed to be much smaller than those of the intracranial pressure changes in the head impact. This might support the fact that the cervical cord injury accompanied by the head impact was relatively slight.

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## 75. Clinico-Pathoanatomical Studies on the Genesis of the Chronic Subdural Hematoma

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The chronic subdural hematoma here concerned is limited to a cystic type which consists of the outer and inner membrances. The classic osmotic theory of Gardner for the genesis of the hematoma has been questioned by various points. Recently, We clearly showed the absence of osmotic difference between the hematoma content and blood or CSF. To elucidate the genesis of the hematoma, we attempted clinico-pathoanatomical studies on the hematoma and its membrane from 20 operated and 17 autopsied cases (average 55 years, 92% in male). The hematoma occurred unilaterally in 88% of the total cases. Anamnesis of trauma was obtained in 100% of the operated cases and in 62% of the autopsied cases. There was no detectable morphological difference in the hematoma membrane among these two groups.

1) By pathoanatomical analysis of the available cases, we propose that the chronic subdural hematoma may be formed by the following process. An old traumatic hemorrhage widely spread in the subdural space is organized from the dural side resulting in formation of a thin fibrous membrane on the dural surface (primary membrane). Multiple petecheal bleedings occur within the primary membrane probably due to breakage of highly congested venules in the membrane. These small bleeding foci confluent together leading splitting of the entire membrane. During this process, many intact blood vessels may be teared, creating a hematoma in the splitted membrane. The process may be progressive untill it forms an efficient size of a hematoma.

2) In nearly total cases examined, signs of intracranial hypertension, slight or severe, were recognized after post-traumatic latent intervals of 4–12 weeks. Signs of mental disturbances may be followed. To determine the significance of the post-traumatic latent interval in relation to hematoma formation, we performed comparative studies between clinical course and morphological analyses on the hematoma