44. The Effects of Hypothermia of Cession of Postencephalitic Epilepsy

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The patient suffered from encephalitis japonica 4 years ago. The epileptic convulsion began about 3 years ago and increased gradually in frequency. After convulsion, the patient always complained of headache, diplopia and numbness of the left side of the body. Various kinds of anticonvulsant drugs were given to the patient during 3 years, but there were no effects on the prevention of an attack of epilepsy. About 6 months ago the patient affected with a status epilepticus continuing for 4 days.

The physical and neurologic findings during the intervals of convulsions were normal but the right oculomotorius paresis. The bilateral ventricles were slightly but diffusely enlarged after carotid angiography and lumbar pneumoencephalography. Dysrhythmia was noted bilaterally after electroencephalography. The patient was anesthetized with GOF by intubation for 2 hours to disrupt convulsion. After waking up from GOF anesthesia, the patient affected with epilepsy again. The body cooling was then, carried out for 3.5 hours in the patient under intubations anesthesia with GOF. The temperature of the rectum was 29°-30°C. No epileptic convulsion occurred for these 6 months after once of the body cooling. At present, no neurological sequelae are found. The anticonvulsant drugs have been continuously given to the patient.

From the results of this case, it is thought that hypothermia would effect on the prevention of the epileptic attack.

45. Subcortical or Brainstem Lesion Effect on Subcorticogenic Seizure with Special Reference to the Forel-H Field

—experimental analysis—

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1. Chronic experiment: Utilizing unanesthetized cats, an attempt was done to make an electroencephalographical analysis of the chronic effect of the bilateral

subcortical or brainstem lesions (2-2.5 mm in maximal diameter of football shape) on the threshold of Metrazol induced major seizure. It has been reported that the seizure discharge is originated at subcortical level of motor system. Threshold increment was obtained when the lesion were made bilaterally at putamen of the subcortical ganglia, mediodorsal nucleus of the thalamus, the posterior half of the hypothalamus, the Forel-H field, red nucleus and the medial and ventral parts of mesencephalic reticular formation. Threshold decrement was, in contrast, obtained when the lesion was put bilaterally at the anterior half of the hypothalamus, ventral nuclei and CM of the thalamus and the dorsal and lateral parts of the mesencephalic reticular formation. The increment was ranged with 93% over the control threshold, and the decrement was within 59% of the control threshold.

2. Acute experiment: Utilizing unanesthetized cats, an attempt was done to make a physiological analysis of the threshold increment mechanism of the Fore-H field lesion to the cortico-subcortical seizures in correlation with hypothalamic and brainstem activation system by means of electrocoagulation or total transection at various levels. The after-discharge threshold increment caused by H field both at motor cortex and lenticular nucleus but not at ventrolateral nucleus of the thalamus nor amygdaloid structures obviously to the interception of the ascending activation impulses originating from the mesencephalic reticular formation both to the motor cortex and lenticular nucleus.

3. Conclusion and Summary: a) Threshold variation was obtained at bilateral lesioning of a certain part of subcortex, thalamus and midbrain reticular formation. The increment and decrement areas were arranged at the ventral and dorsal parts respectively of the diencephalic and mesencephalic border level. b) The H-field, medial part of the subthalamus appears to be the main axis of the pathways between cortico-subcortex and midbrain both for the descending epileptogenic convulsive impulses originating at the former and for the ascending seizure maintaining impulses originating at the latter.

46. Experimental Study on Effects of Hyperventilation on Cerebrospinal Fluid Pressure and Oxygen Tension in the Cerebral Cortex

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It is known that hyperventilation is one of the effective methods of reducing intracranial pressure. It is also known, however, that hyperventilation may cause cerebral anoxia. The present study was undertaken for the purpose of investigating