Experimental Cerebral Infarction

Part 2: Electroencephalographic Changes Produced by Experimental Thalamic Infarction in Dogs

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SUMMARY Previously, one of the authors developed a reliable experimental model in dogs for producing cerebral infarction. The

EEG increased detection of experimental cerebral infarction, and was useful in predicting ischemic regions or infarction.

A PROEDURE recently developed by one of the authors produced a localized infarction confined to the thalamus with a high rate of success.

Methods

Adult mongrel dogs were used in these experiments. Intravenous pentobarbital (35 mg/kg) was used for general anesthesia. The airway was secured with an oral endotracheal tube with spontaneous respiration. After fixing the head of the dog on a stereotaxic apparatus, we exposed the underlying skull with a midline sagittal incision. Holes were drilled through the skull and depth electrodes inserted at approximately 100 μm and 10 Hz.

Small holes were drilled through the frontal and parietal bones bilaterally. Screws, (φ 2 mm) were inserted as epidural electrodes, and a reference electrode was placed in the paranasal sinus (fig. 1). These were immobilized with dental cement. The animal was then removed from the stereotaxic apparatus and an intracranial vascular occlusion was produced surgically as described elsewhere.

The EEG tracings were directly observed on the oscillograph and simultaneously recorded on a data recorder which fed them to a signal processor (San-ei Sokki, 7T07) for power spectrum analysis, and recording on an X-Y recorder.

Experiment 1. Repeated Short Term Occlusion

We occluded 4 cerebral arteries simultaneously (internal carotid, middle cerebral, anterior cerebral, and posterior cerebral) with clamps and released them after 5 minutes. This procedure was repeated several times at 20 minute intervals.

Experiment 2. 7 Day Follow Up

In Experiment 2 we used only those animals whose depth EEG recording from the thalamus showed diminution of fast waves with an attenuation of voltage on the side of the surgical occlusion. The 4 arteries were occluded for 2 hours. Serial EEGs were recorded for approximately 10 minutes following an intravenous dose of pentobarbital (15 mg/kg) on days 3, 5, and 7 after the surgery.

Autopsy and histological examination were made on the 7th day after surgery. The sizes of the infarctions in the thalamus were estimated microscopically, and each infarction was graded according to a 4-point scale: grade 0: no infarction in the thalamus, grade 1: small infarction foci (less
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than half) of the thalamus, grade 2: infarction affecting about half of the thalamus, grade 3: infarction affecting more than two-thirds of the thalamus.

Results

Experiment 1. Repeated Short Term Occlusion (fig. 2)

Following temporary 4 vessel occlusion, a significant diminution of fast wave components of about 10 Hz occurred with attenuation of voltage in the ipsilateral thalamus. The contralateral thalamic EEG showed no appreciable change when compared to that obtained before occlusion. When the clamp was released 5 minutes after its application, the ipsilateral thalamic EEG showed fast wave components with restoration of potentials to the preocclusion level. The result was reproducible in the repeated experiments.

Experiment 2. 7 Day Follow Up (figs. 3, 4)

After 2 hours of occlusion, the thalamic EEG on the occluded side showed a pattern characterized by diminution of fast wave activity and attenuation of voltage. These changes persisted throughout the occlusion. Following release of the occlusion, fast wave components of about 10 Hz returned although they were fewer than observed prior to the occlusion. On the 3rd day, only slow waves of 4 Hz or lower were seen. These waves diminished on the 5th and 7th days; and the EEG became virtually flat. In the non-occluded side there was no difference in the EEG before, and 7 days after, contralateral vascular 4 vessel clamping.

Cortical EEG tracings were observed to be unchanged on both the ipsi- and contralateral cerebral hemispheres. Microscopic examination revealed infarcts localized to the thalamus on the occluded side in all 4 animals, and were grade 3 in 2 dogs and grade 2 in 2 dogs.

Discussion

For an adequate experimental model of cerebral infarction, the procedure must produce a constant lesion topographically and chronologically with high predictability. Chronic experiments must be possible.

The infarction produced by the occlusion of the middle cerebral artery varied markedly among investigators, using a variety of animals. The dog has complex and numerous cerebral collaterals, so that permanent occlusion does not regularly induce infarction.

In a previous study, temporary occlusion of internal carotid, middle cerebral, anterior cerebral arteries and the distal portion of the posterior communicating artery in dogs in the vicinity of the circle of Willis produced infarction. The area of infarction involved the entire thalamus in a frontal section at a site 5 mm posterior to the posterior margin of the optic chiasma. Two hour temporary occlusion produced infarction in 13 of 20 dogs.

When EEG recordings were made from the anterior part of the thalamus (N. Ventralis) fast wave components diminished significantly with attenuation of voltage in the ipsilateral thalamic lead following the temporary cerebrovascular occlusion.

These changes were reproducible by repeated vascular occlusion and release.

The EEG pattern is influenced by vascular occlusion so

![Figure 1. Diagrammatic representation of the EEG recording system. F — Frontal epidural electrode. P — Parietal epidural electrode. N — Reference electrode. Th — Thalamic depth electrodes.](image1)

![Figure 2. Changes in the thalamic depth EEG pattern and power spectrum following repeated temporary occlusions of the right side. L-Th — depth EEG from non-occluded side (upper) and its power spectrum (lower); R-Th — depth EEG from occluded side (upper) and its power spectrum (lower). Abscissa of power spectrum indicates frequency, graduated at 2-Hz intervals.](image2)
the cessation of cerebral blood flow\textsuperscript{7-9} and flattening of the EEG suggests ischemia of cerebral tissue.

The reasons for these changes are several and include synaptic depolarization following release of cellular potassium into the extracellular space, changes in neurotransmitter metabolism, tissue lactic acidosis and changes in ATP metabolism.\textsuperscript{11, 12}

The electrophysiological recordings in this animal model, with constant infarction in all dogs, showed diminution of the fast wave component and attenuation of voltage in the ipsilateral thalamic lead. Using this model the relationship between EEG change and cerebral blood flow may be clarified.

References

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