the endothelial cells did not penetrate the membrane of the neurons. Instead, we believe that the neurosecretory cells ensheath these capillaries. These observations, however, must await confirmation at the ultrastructural level.

Capillaries with similar characteristics have been described in the caudal neurosecretory system in other species of fishes[8,12]. However, in those species this studied phenomenon was reported as an occasional occurrence. The most intriquing finding in the present study of the bluefish was that the majority of the neurons in the caudal neurosecretory system contained capillary networks in close association with the nucleus and cytoplasm.

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Influence of Thalamic Stimulation on Cortical Epileptogenic Focus

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Summary. Single stimuli applied to the non-specific thalamic nuclei do not change the activity of a cortical epileptogenic focus whereas rhythmic stimulation of these structures transforms the interictal activity into an ictal one.

Discharges of an experimental cortical epileptogenic focus could be triggered by various sensory stimuli[1,2], or by electrical stimulation of the nervous system[3–10]. The only structure from which cortical focal discharges could not be triggered was non-specific thalamus[6,7,9]. But rhythmic stimulation of this structure induced generalized epileptic phenomena[11,12]. For this reason we decided to study the influence of stimulation of non-specific thalamic nuclei on a cortical focus.

Experiments were performed on 14 rabbits aged from 2 to 3 months. Animals were without general anaesthesia, locally anaesthetized with Procaine and immobilized by Flaxedil. The concentric stimulation electrode (of 0.4 mm outer diameter) was introduced stereotaxically into the thalamus at coordinates AP 3–3.5, L 3.5, V 6 mm[13]. Localization of the electrode tip was controlled electrophysiologically as well as histologically. An epileptogenic focus was elicited by K-salt of penicillin (PNC) applied on the undamaged dura mater, covering the somatosensory cortical area at the same side at which the thalamic electrode was placed. Activity of somatosensory areas of both hemispheres was registered in reference connections (an indifferent electrode on the stereotaxic frame) using an 8-channel EEG apparatus. The thalamic stimulation started after PNC focus had been stabilized (usually 10 to 20 min). Rectangular pulses of 0.1–0.2 msec duration, frequencies from 0.2 to 40 Hz were applied. Stimulation series of frequencies higher than 2 Hz lasted for 30 sec; minimal interval between 2 stimulations was 3 min. Intensity of stimulation was lightly suprathreshold for the thalamocortical evoked potential.

Results obtained in 10 animals with stimulation electrodes localized in the non-specific thalamic nuclei (lateralis anterior, lateralis posterior, anteroventralis) did not differ and the description relates to all the cases. Stimulation of these nuclei never triggered focal discharges at the 1:1 rate (Figures 1 and 2). Rhythmic stimulation

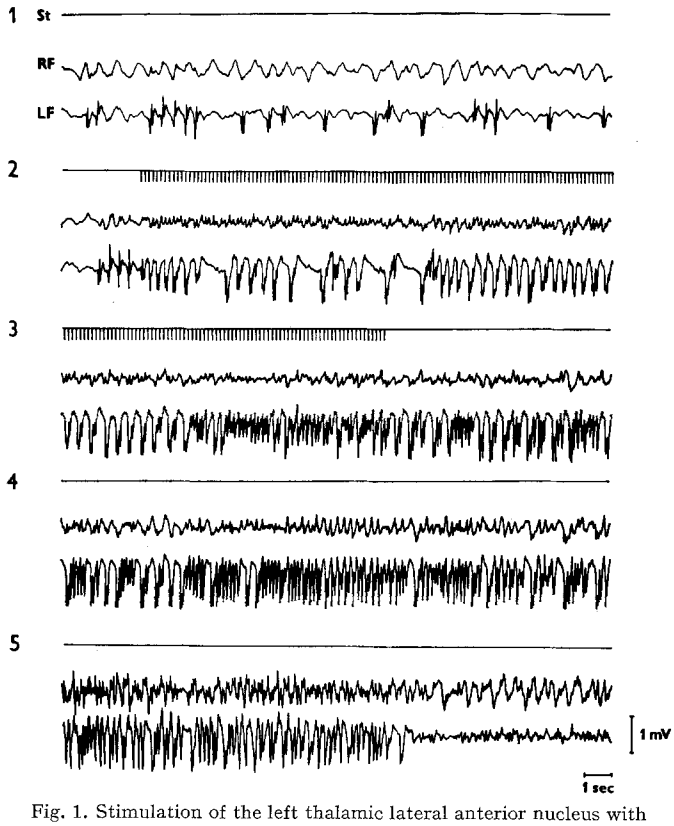

Fig. 1. Stimulation of the left thalamic lateral anterior nucleus with frequency of 7 Hz. From top to bottom: stimulation marks, activity of right and left somatosensory cortical area. 1, 2, 3, 4 and 5 – continuous recordings. Time mark 1 sec, amplitude mark 1 mV.

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of 2 Hz frequency and even higher ones resulted in potentiation of late components of the focal discharges (i.e. after-discharges; Figure 1), a transition from the interictal to ictal phase of activity was often seen at stimulation frequencies of 4 to 10 Hz. The ictal activity usually started during stimulation series and continued after cessation of stimulation. It was often restricted to the hemisphere where the PNC was applied and spread to the opposite hemisphere when seizures reappeared. Once started, the ictal activity could be observed recurrently, also without any stimulation. In a seizure it was possible to distinguish 2 phases: the 'tonic' one with rhythmic spike activity, which transgressed into the following 'clonic' one with spike-and-wave or polyspike-and-wave electrocorticographic pattern. Stimulation during this second phase could revoke the first 'tonic' pattern.

Stimulation of the specific relay nuclei (in 2 rabbits) never elicited transition into the ictal phase. Low frequencies of stimulation triggered focal discharges at 1:1 rate, 'cyclical spike driving'[14], was also observed.

Hippocampal stimulation (2 cases) did not trigger individual focal discharges. Transition into ictal activity with higher stimulation frequencies was possible, but repeated stimulations were necessary; the seizure was sometimes found to start only after the stimulation was over.

Our results confirm the conclusions of previous authors[6,7,9] that the non-specific thalamic stimulation cannot trigger individual discharges of the cortical epileptogenic focus. The rhythmic stimulation in our experiments was found to transform the interictal activity into ictal phase. The results of MIHAILOVIĆ[15], with two independent cortical foci synchronized during non-specific thalamic stimulation, are based on the same mechanism – the synchronization of cortical bioelectric activity with a rhythmic stimulation of these thalamic nuclei creates favourable conditions for the spread of epileptic activity[16,17].

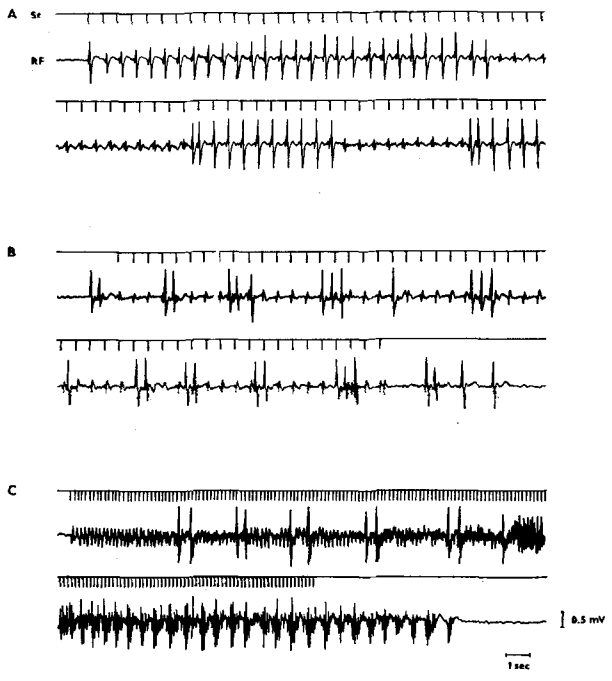

Fig. 2. (A) Stimulation of the right ventral posterolateral thalamic nucleus with frequency of 1.6 Hz; (B) stimulation of the right lateral anterior nucleus with frequency of 1.6 Hz; (C) Stimulation of the right lateral anterior nucleus with frequency of 7 Hz. In all pairs the upper row – stimulation marks, the lower row – activity of the right somatosensory cortical area. Both pairs in each section represent continuous recordings. Time mark 1 sec, amplitude mark 0.5 mV.

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Influence of the Visual Cortex upon Collicular Evoked Responses in the Rabbit

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Summary. In rabbit, the depression of the visual cortex by KCl showed an enhancement of the collicular evoked potentials. This enhancement revealed that the visual cortex had exerted a tonic type influence upon the superior colliculus.

Electrophysiological studies have shown that receptive field properties of the superior colliculus (SC) cells depend to a considerable extent on cortical input in cat[1,2] and monkey[3]. Particularly the visual cortex seems to play a prominent role in controlling the responsiveness of deeper layers of the SC. In contrast to the cat, the ablation of visual cortex in the rabbit does not seem to affect the receptive field characteristics of superior colliculus neurons for either chronic or acute preparations[4,5]. However, the rabbit's SC receives inputs from visual area I and II[6]. Thus it seems that the role of the visual cortex upon the SC in rabbit is not yet established. The present results suggest that the visual cortex may have some tonic effect upon collicular responsiveness as revealed by an increase of evoked responses following a topical application of KCl upon visual cortex as a means for interrupting the cortical functions.

Methods. The studies were conducted on adult rabbits of either sex, anesthetized with pentobarbital sodium (30 mg/kg, i.v.), paralyzed with Gallamine triethiodide and kept under artificial respiration. The responses were evoked by applying, every 2 sec, a 900 msec pulse of light (45 F.C.) which provide a ON stimulus at its onset and a OFF stimulus at its offset. The optic nerve was electrically stimulated (ONS) through a fine platinum curved needle inserted in close proximity to the nerve at its exit