

## SUMMARY

This thesis deals with multiple aspects of changes in excitability of brain tissue that can be observed during development of cortical ischemic lesion and epilepsy.

Firstly, we thoroughly explored experimental models of cerebral ischemia and we introduced and adapted a model of a cortical photothrombotic ischemic lesion in a rat. This model was morphologically verified on coronal slices incubated in TTC.

Next, we researched changes in somatosensory evoked potentials recorded on cortex during development of cortical ischemic photothrombotic lesion. The observed decrease in amplitude of components of the somatosensory evoked potential is congruent with the current state of knowledge. Unexpectedly, less pronounced changes were apparent in the control group and in the contralateral hemisphere. They can be attributed to the influence of the laser light alone on the cortex. Similar effect can be expected to cause shortening of latency in both groups and both hemispheres.

Thirdly, we confirmed that acute changes of single unit activity recorded in the vicinity of a developing ischemic focus resemble expected changes of local perfusion. This also confirms efficacy of the model of cortical photothrombotic ischemic lesion. We also observed similar, but less conspicuous and transitory changes of single unit activity in the control group, which we attribute to the effect of the laser light.

In the fourth part, we described the implementation of semi-interactive detection of components of somatosensory evoked potential in time, that was necessary for the evaluation of our aforementioned experiments. We deliberately employed several programming techniques (nearest neighbour, simulated annealing with multidimensional optimization criterion). The output of the program was verified by comparison with the recording in a three dimensional chart.

The fifth part of the work involved the design and implementation of an algorithm for detection and evaluation of single unit activity recorded in the third part of this work. The program we designed met our requirements. We used and adapted several common programming techniques and data mining methods (data filtration, neuronal network, k-means algorithm, principal component analysis). In comparison with other methods, the level of detection achieved with our program was excellent.

In the light of our previous findings, we researched the influence of laser light in the visible spectrum on excitability of brain tissue. These changes were compared with findings in previous experiments as well. We assume, that changes in excitability (changes of latency, amplitude of components of evoked potentials and single unit activity) are with regard to the latency of their onset caused by changes in concentration of intracellular ions induced by exposure to laser light. This is also the suggested

mechanism responsible for shift of frequency maximum after cessation of rhythmic cortical stimulation with laser or during repeated stimulation of cortex by short laser pulses. Similar changes in response to electric cortical stimulation or electric stimulation of peripheral nerve observed in the contralateral hemisphere can be explained by the influence of ambient light or by diaschisis.

Finally, in the last part of the research work, we showed that a burst of four electrical pulses can unmask persistently increased excitability overshadowed by massive inhibition even in the interictal period when postictal inhibition sets in. We believe that repeated stimulation of the same or increased intensity may paradoxically stimulate inhibitory systems and ultimately lead to increased refractivity in the postictal inhibition period. In comparison, with the same stimulation scheme and laser instead of electric pulses, we were unable to provoke any epileptic phenomena.