during the functional loadings the magnitude of positive shifts of DC-potentials in temporal and frontal area was increased. These changes reflect more pronounced than in healthy controls lowering of cerebral pH probably caused by increased glycolysis.

It is supposed that in persons at genetic risk for AD the abnormal electrophysiological patterns of brain activation in response to memory task and hyperventilation can be related to the alteration of brain energy metabolism.

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P.5.102 Change of cerebral acid–base balance with chronic brain ischemic disease in response to vinpotropil therapy

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In patients with chronic brain ischemic disease (CBID) the disturbances of cerebral blood circulation lead to neurodegeneration. Since the decrease of cerebral blood flow is often accompanied by a shift of acid–base balance (ABB), we estimated the ABB changes using brain DC-potential method. DC-potentials recorded from the head reflect the electrical potentials of the brain–blood barrier, which depend mainly on the intravessel concentration of $[H^+]$ ions (Lehmenkuhler et al., 1999; Fokin and Ponomareva, 2003). 29 subjects with CBID (13 men and 16 women, mean age 54.6±3.1 years) were examined before and after one month administration of vinpotropil, which has nootropic properties and improves the glucose metabolism in the brain.

Before treatment, DC-potential levels were significantly higher in the patients with CBID than in healthy controls, indicating moderate cerebral acidosis. After a month of vinpotropil therapy a correction of DC-potentials was observed. The pathological symptoms were reduced. Transcranial Doppler sonography showed an increase of blood flow in the hemispheres. It is supposed that the correction of acid–base balance after vinpotropil therapy is due to the increase of cerebral aerobic glucose metabolism.

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P.5.103 Neuroprotective effect of a synthetic peptide mimetic of neural cell adhesion molecule, P2, in an in vitro model of ischemic damage

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Neural cell adhesion molecule (NCAM) is known to be involved in molecular mechanisms modulating neuronal survival and functioning in normal and pathological conditions, and it is of crucial importance in regulating neuronal differentiation and synaptogenesis. NCAM functions depend on its adhesive and signaling properties. The homophilic and heterophilic binding sites of NCAM are currently identified and synthetic peptides mimicking physiological binding interaction have been prepared.

In the present study, we used a synthetic NCAM peptide mimetic derived from a homophilic heterophilic binding site of NCAM, termed P2, which corresponds to a 12 amino acid sequence in the second NCAM Ig-module. The effects of P2 were estimated using organotypic hippocampal slice cultures in normal conditions and after the exposure to short-term (10 min) combined oxygen and glucose deprivation (OGD), which was used to induce ischemic cell damage in vitro. P2 was added to the culture medium 3–4 h before OGD. The cultures were tested immediately after OGD and at 1 h and 4 h of reoxygenation. The viability of hippocampal CA1 neurons (by propidium iodide or trypan blue staining) and the metabolic activity of the slices (by MTS/formazan-assay) were assessed. After fixation and embedding of the cultures in epoxy resin (EPON), light and electron microscopy analysis were carried out. 4 h after OGD, an increase in the number of damaged neurons in the CA1 area and a decrease of the total metabolic activity of the slices were observed. In most cases, we noticed a cytoplasmic condensation of the neurons in OGD-exposed cultures as compared to control. P2 was found to abolish