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Presentation Abstract

Program#/Poster#: 241.4/I11

Title: Parvalbumin immunoreactivity in epileptic brain: Comparison of SE and TBI as epileptogenic etiology

Location: South Hall A

Presentation Time: Sunday, Oct 18, 2009, 4:00 PM - 5:00 PM

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Abstract: Objective: Parvalbumin (PARV) labels about 20 % of GABAergic interneurons which are responsible for perisomatic and axo-axonic inhibition to principal cells. Not surprisingly, impaired function of GABAergic perisomatic inhibition is presumed to contribute to seizure susceptibility in experimental and human epilepsy. To address a question, whether the severity of damage to PARV positive neurons depends on the type of epileptogenic insult, we compared the numbers of PARV immunopositive cells after status epilepticus (SE) and traumatic brain injury (TBI) in rats.

Material and methods: SE was induced by electrical stimulation of the lateral nucleus of the amygdala (14 stimulated, 7 controls) and TBI by lateral fluid-percussion brain injury (9 TBI, 9 controls). After 7 months, rats were video-EEG monitored for 2 wk to detect spontaneous seizures. Rats with TBI also underwent testing of seizure threshold with pentylenetetrazol under video-EEG control. Rats were perfusion-fixed for histology, and total numbers of PARV positive neurons in different hippocampal subfields were estimated with unbiased stereology (Stereoinvestigator®).

Results: In SE group, 7 rats had spontaneous seizures. In TBI group none of the rats had spontaneous seizures but seizure threshold was lowered. As compared to controls, the total number of PARV positive neurons in the ipsilateral (left) hippocampus was 52 % of that in TBI ($p < 0.05$), and 77 % in rats with SE and seizures ($p < 0.01$). The most dramatic decrease in TBI animals was seen in the hilus (30 % remaining, $p < 0.001$). Contralaterally, 65 % of PARV positive neurons in TBI rats were remaining ($p < 0.01$). The numbers of PARV positive neurons did not correlate with the decrease in seizure threshold in TBI rats or the frequency of spontaneous seizures in

SE animals.

Conclusion: Decrease in PARV positive neurons depends on epileptogenic etiology being greatest in rats with TBI which show lowered seizure threshold but no spontaneous seizures at the time of cell counting. Unexpectedly, the loss of PARV neurons in spontaneously seizing rats with post-SE epilepsy is much milder than in the TBI group, and not associated with seizure frequency. These observations suggest that reduced perisomatic inhibition in the dentate gyrus can contribute to seizure susceptibility. However, even a dramatic loss is not necessarily associated with occurrence of spontaneous seizures.

Disclosures: **N. Huusko**, None; **C. Roemer**, None; **A. Pitkanen**, None.

Keyword(s): GABAergic neurotransmission

Epilepsy

Animal models

Support: EPICURE project (FP6 grant LSHM-CT-2006-037315)

The Sigrid Juselius Foundation

The Academy of Finland

[Authors]. [Abstract Title]. Program No. XXX.XX. 2009 Neuroscience Meeting Planner. Chicago, IL: Society for Neuroscience, 2009. Online.

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