Modified sensory processing in the barrel cortex of the Rat model of Alzheimer’s disease

Goshadrou F (PhD)

Assistant Professor, Department of Physiology, Paramedical Sciences Faculty, Neuroscience Research Center, Shahid Beheshti University of Medical Sciences, Tehran, Iran.

Abstract

Background and Objective: Alzheimer’s disease is characterized by brain degenerative alterations with subsequent learning and memory loss. Learning and memory is closely associated with brain cholinergic system. Cholinergic fibers originated from eminent basal nucleus which is extended to cortex and hippocampus. This study was conducted to investigate sensory processing in the barrel cortex neurons of Rat model of Alzheimer’s disease.

Materials and Methods: In this experimental study, 14 male Wistar Rats weighing 250-350g randomly divided into control and experimental groups. Alzheimer’s disease in Rats induced, by infusion of ibotenic acid (5 µg/µl in each site) into nucleus basalis of Meynert (NBM) using Hamilton syringe and stereotaxic apparatus. The control group was non-lesion Rats with vehicle treatment. Two weeks after NBM-lesion, each animal was tested by passive avoidance learning (PAL), then neural response assessed by extracellular recording.

Results: In cases, ibotenic acid infusion into NBM, significantly reduced memory (P<0.05). The results evoked by multiple whisker stimulation in extracellular single unit recording showed that in Alzheimer’s disease model of animals excitatory receptive field (RF) of neurons were extended but inhibitory RF was decreased (P<0.05). In addition the magnitude of neural response following principal whisker deflection decreased in cases (P<0.05).

Conclusion: This study indicated that in animal model of Alzheimer’s disease possibly reduce sensory processing and contact discrimination.

Keywords: Alzheimer’s disease, Sensory processing, Barrel cortex, Ibotenic acid, Rat

* Corresponding Author: Goshadrou F (PhD), E-mail: fgoshadrou@yahoo.com

Received 12 September 2010 Revised 14 March 2011 Accepted 17 April 2011