Physiology and Pharmacology, 15 (1), 1-15
Spring 2011 [Article in Persian]

Physiology and Pharmacology

Electrophysiological characteristics of hippocampal CA1 neurons after spreading depression-triggered epileptic activity in brain slices

Parviz Shahabi[1], Ali Gorji[2]*, Yaghoub Fathollahi[1], Mahyar Janahmadi[3], Javad Mirnajafi-zadeh[1], Jalil Arabkheradmand[4], Marius Alexander Eickhoff[2]

1. Dept. Physiology, Faculty of Medical Sciences, Tarbiat Modares University, Tehran, Iran
2. Westfälische Wilhelms-Universität Münster, Institut für Physiologie I, Münster, Germany
3. Neuroscience Research Centre and Dept. Physiology, Medical School, Shahid Beheshti Medical Sciences University,Tehran, Iran.
4. Shefa Neuroscience Center, Tehran, Iran

Received: 22 Oct 2010 **Accepted: 4 Dec 2010**

Abstract

Introduction: A close link between spreading depression (SD) and several neurological diseases such as epilepsy could be demonstrated in many experimental studies. Epilepsy is among the most common brain disorders. Despite a large number of investigations, its mechanisms have not been yet well elucidated. Hippocampus is one of the important structures involved in seizures. The aim of this study is to get an insight into the patho-physiological processes induced by SD that lead to the generation of epileptiform field potentials.

Methods: The horizontal amygdala-hippocampus-neocortex slices of rat brain in which SD was induced by KCl application in each brain structure were used. Following $GABA_A$ receptor antagonist bicuculline superfusion (1.25 µmol/L, for 45 min), SD induced epileptic activity in all tested slices was monitored.

Results: The induction of SD in the hippocampus resulted in interictal and ictal epileptiform field potentials and intracellular paroxysmal depolarization shifts (PDS). After SD, RMP slightly depolarized and the threshold of AP decreased, while the frequency of AP significantly increased. Amplitude of depolarization and also amplitude of discharges were also significantly increased. ISI significantly decreased and the most of neurons shifted from FA to SA indicating an enhanced excitability.

Conclusion: SD may cause pathological changes in brain structures such as increased excitation and/or decreased inhibition. Propagation of SD over epileptogenic areas may trigger seizure attacks in some patients and our findings provide evidence on the role of SD in temporal lobe epilepsy.

Key words: Epilepsy, Horizontal slices, Spreading depression, Ictal burst activity, Bicuculline

* Corresponding author e-mail: gorjial@uni-uenster.de
Available online at: www.phypha.ir/ppj