

Acta Persica Pathophysiological

Pathophysiology of epilepsy

Azam Asgari^{1*}

¹Shahrekord University of Medicine Sciences, Shahrekord, Iran

ARTICLE INFO

Article type:
Epidemiology and Prevention

Article history:
Received: 8 February 2016
Accepted: 28 March 2016
Published online: 3 April 2016

Keywords:
Epilepsy, Morbidity, Mortality,
Diet

ABSTRACT

Core tip: The physiopathology of the epilepsies is still incomplete. A significant number of patients with epilepsy, especially temporal lobe epilepsy, are pharmacoresistant. Surgical incision as a treatment choice for these patients is associated with serious side effects and may be effective for a small percent of patients. Therefore, many researchers hope to find new therapeutic ways for treatment of these drug resistant epileptic patients.

Please cite this paper as: Asgari A. Pathophysiology of epilepsy. Acta Persica Pathophysiol. 2016;1:e07.

Introduction

Epilepsy is one of the most popular neurological diseases, involving people of different ages that result from a hyper-synchronous neuronal discharge. Dysregulation of the balance of excitatory and inhibitory neurotransmission due to various causes such as inherited or acquired gene mutations has been implicated in a wide range of brain disorders, including different type of epilepsy syndromes (1,2). The property of all epileptic syndromes is a persistent enhancement of neuronal excitability. However underlying causes and physiopathological mechanisms are not clearly understood but neuronal abnormality likely associated with causative factors such as brain injury, metabolic disorder, brain tumors, infection, stroke, inflammation and hypoxia (1).

The most developmental brain disorders (for example tuberous sclerosis, Angelman syndrome and Rett syndrome) that lead to epilepsy are derangement of neuronal migration that may have genetic causes. About one-third of epileptic patients have a genetic problem that contributes to the etiology of epilepsy (2).

Different factors are involved in creating epilepsy but functional, structural and electrophysiological alterations occur in all epileptic brain areas. The functional changes involve quantity of cations and anions, metabolic changes, and changes in neurotransmitter levels. The structural alteration includes of both neurons and glial cells. In abnormal conditions amount of potassium in extracellular fluid will increase and depolarizes neurons so leads to discharge activity. Glia is more numerous than nerve cells in the mammalian brain and able to clear neurotransmitters from the extracellular environment and correcting the extracellular fluid potassium concentrations that increased during seizures, in normal range. Proliferation of glia in epileptic brain area will

affect glial potassium regulating capacity and contribute to seizure generation (3,4).

Clinical and experimental evidence indicates that in some types of epilepsy excitability is due in part to impaired gamma-aminobutyric acid (GABA)ergic inhibitory. One form that this can take is impaired excitatory synaptic input to GABAergic interneurons. As the main inhibitory neurotransmitter, GABA can effectively control the excitability of neurons through activation of its receptors in the central nervous system. A-type GABA receptors are the major Cl⁻ permeable ion channels activated by GABA and the most abundant rapid inhibitory neurotransmitter receptors in the brain. The GABA receptor controls basal information processing in the central nervous system, therefore permitting it to affect a wide variety of physiological and pathophysiological processes (5,6). Drugs that enhance GABA-mediated inhibition are anticonvulsant. Mechanisms through which this occurs include direct interaction with the GABA/benzodiazepine receptor (benzodiazepines, barbiturates, chlormethiazole), inhibition of GABA-transaminase (vigabatrin) and blocking GABA uptake (tiagabine) (7). Anticonvulsant medication is still the most common method for treating individuals with epilepsy, although more than 10 new antiepileptic drugs have been established in the past decade, epilepsy stays resistant to drug therapy in about 70% of patients. However, the rest persist to suffer from seizures, illness and some risk of mortality (8-10).

Conclusion

The pathophysiology of the epilepsies is still incomplete. A significant number of patients with epilepsy, especially temporal lobe epilepsy, are pharmacoresistant. Surgical incision as a treatment choice for these patients is

*Corresponding author: Azam Asgari,
Email: aazam.asgari1@gmail.com

associated with serious side effects and may be effective for a small percent of patients. Therefore, many researchers hope to find new therapeutic ways for treatment of these drug resistant epileptic patients.

Authors' contribution

AA is the single author of the paper.

Conflicts of interest

The author declared no competing interests.

Ethical considerations

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the author.

Funding/Support

None.

References

1. Lees GV, Jones EG, Mcnamara J. Epilepsy. *Neurobiol Dis.* 2000;7:549-51.
2. Ure JA, Perassolo M. Update on the pathophysiology of the epilepsies. *J Neurol Sci.* 2000;177:1-17.
3. Bordey A, Sontheimer H. Properties of human glial cells associated with epileptic foci. *Epilepsy Res.* 1998;32:286-303.
4. Cloix JF, Hévor T. Epilepsy, regulation of brain energy metabolism and neurotransmission. *Curr Med Chem.* 2009;16:841-853.
5. Brickley SG, Mody I. Extrasynaptic GABA (A) receptors: their function in the CNS and implications for disease. *Neuron.* 2012;73:23-34.
6. Delorey TM, Olsen RW. Gamma-aminobutyric acid receptor structure and function. *J Biol Chem.* 1992; 267:16747-50.
7. Macdonald RL, Bianchi MT, Feng H. Mutations linked to generalized epilepsy in humans reduce GABA (A) receptor current. *Exp Neurol.* 2003;184 Suppl 1:S58-67.
8. Olsen RW. Phenobarbital and other barbiturates: mechanism of action. In: Levy RH, ed. *Antiepileptic Drugs*. Philadelphia: Lippincott Williams and Wilkins; 2002:489-95.
9. Schmidt D, Loscher W. New developments in antiepileptic drug resistance: an integrative view. *Epilepsy Curr.* 2009;9:47-52.
10. Sander JW. The use of antiepileptic drugs--principles and practice. *Epilepsia.* 2004;45 Suppl 6:28-34.

Copyright © 2016 The Author(s); Published by Nickan Research Institute. This is an open-access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.