REVIEW

CALCIFEROL MODULATION IN EPILEPSY

Wojciech Ku³ak1, Wojciech Sobaniec1, Katarzyna Wojtal2, Stanis³aw J. Czuczwar2,3

1Department of Pediatric Neurology and Rehabilitation, Medical University of Białystok, Warszynioka 17, PL 15-274 Bia³ystok, Poland, 2Department of Pathophysiology, Medical University, Jazewskiego 8, PL 20-060 Lublin, 3Isotope Laboratory, Institute of Agricultural Medicine, Jarzembowskiego 2, PL 20-000 Lublin, Poland


The ideal antiepileptic drug (AED) should correct the aberrant pathophysiology of epileptogenesis without interfering with normal neurotransmission. A new group of drugs with antiepileptic efficacy, without sedative properties, would be an exciting prospect. Theoretical considerations and results from experimental animal models of epilepsy have put forward the possibility that calcium (Ca2+) antagonists may form such a group. The initiation of epileptogenic activity in the neuron is thought to be connected with the phenomenon known as “intrinsic burst firing”, which is activated by an inward Ca2+ current. Ca2+ is described as the primary mediator of “excitotoxic” neuronal damage. Both necrotic and apoptotic cell death is associated with Ca2+ entry into the cells during status epilepticus. The Ca2+ channel blockers depressed epileptic depolarizations of neurons. In this review, we present anticonvulsant effects of cinnarizine, flunarizine, nifedipine, nimodipine, nicardipine, amloidpine, isradipine, niguldipine, diltiazem, verapamil and dantrolene in animal models of seizures. Also, a detailed analysis of interactions between Ca2+ blockers and AEDs was performed. Clinical trials in intractable epilepsy support to a certain degree antiepileptic properties of Ca2+ antagonists.

Key words: antiepileptic drugs, calcium channel blockers, seizures

correspondence; e-mail: kneur2@wp.pl