

Hypoxic - ischemic CNS lesions and epileptic seizures share many common pathophysiologic features. Basic mechanisms, either detrimental (excitotoxicity, free radicals, inflammation, changes in permeability of the blood brain barrier, particularly necrotic and apoptotic cell death), or reparative (gliosis, neuroplasticity, neurogenesis, vascular proliferation) occur in

Both types of damage, but may be expressed in varying degrees, and may take place in different time term.

Hypoxia, but also global and focal ischemia, CNS are in humans and in experimental models under certain conditions accompanied by epileptic seizures. Epileptic seizures vice versa

acutely and chronically affect cerebral blood flow (CBF). CBF and cerebral metabolism is significantly

affected during status epilepticus (SE). CBF changes can significantly contribute to pathophysiological

consequences of seizures and may have links with some of their clinical manifestations (eg

Todd's postparoxysmal hemiparesis (MS Mathews et al., 2008; Yarnell PR 1975)).

Focal ischemia, CNS has often consequences in distant, but functionally linked areas (diaschisis). There is a remote influence not only the blood flow and metabolism, but also

excitability (Andrews, RJ, 1991 Buchkremer - Ratzmann I. et al. 1996b). Also in the focal

epilepsie often affecting the metabolism and perfusion in remote areas (Stubgen JP 1995).

Secondary epileptogenesis in areas remote primary epileptogennímu bearing is also known (eg the mirror focus (Morrell and F. de Toledo- Morrell L., 1999 N. Tsuru, 1981 ; Wilder

B. J. and Morrell, F., 1967)).

Common phenomena can also be found in studying the effects of repeated exposure epileptic seizures or hypoxia / ischemia. When repeating insults may be resistance following exposure increase or decrease the contrast . Epileptic tolerance (increasing resistance to repeated seizures) has been documented as the model bikukulinových convulsions (Sasahira M. et al. 1966)

1 International classification of epilepsies and epileptic syndromes (ICEES , 1989) distinguishes between epilepsy and epileptic syndromes partial (1) , generalized (2) , without the possibility of determining whether partial or generalized (3) and special syndromes (4).

The term focal epilepsy (in English. Focal epilepsy) is as synonymous with partial epilepsy commonly used.

International classification of epileptic seizures (ICES , 1981) seizures generally divided into partial (1)

generalized (2) and unclassifiable (3) . Proposal of a new classification of epileptic seizures and epilepsy (ILAE , 2001

2006) criticizes the term partial (in English. Partial = partial , incomplete) and recommended it to replace the term focal (focal)

which better reflects the idea about generated in a distinct part of the brain. In this dissertation are

terms focal and partial used as synonyms . The trend towards one or the other term (particularly verbal

connections) is elected according to traditional practices (eg, focal epilepsy and no partial epilepsy partial complex

focal seizures and not complex seizures , etc.)

al . , 1995) or in intrahipokampální application of kainate (Lere C. et al. , 2002). The phenomenon of kindling , ie

gradual mongering epileptic answer (and its generalization) repeated induction of focal

seizures is also well known (Racine RJ , 1972). Ischemic or hypoxic tolerance , better known

Today , the term ischemic or hypoxic preconditioning is known about since the middle of last

century . Ischemic preconditioning has been extensively studied mainly in the heart , but from about 90 flight

the last century, whether in the CNS (K Kitagawa et al. , 1990). Epileptic and ischemic tolerance can be

in addition cause cross , as was observed mitigate the consequences of focal ischemia due to previous

exposure seizures (Plamondon H. et al. , 1999) , or attenuation of consequences experimental assistance of hypoxic preconditioning (Emerson MR et al . , 1999).

The study of common mechanisms of hypoxia / ischemia and seizures has undoubted importance for clinical practice. It can contribute among others to understand the pathophysiology

symptomatic epilepsy, cerebrovascular disease (stroke) , posthypoxických Epilepsy children postparoxysmálních neurological deficits or late consequences . In addition, however, leads

to a deeper understanding of the general principles of neural tissue response to various damaging goes on and on

allows you to search for new therapeutic strategies .

Attempting to capture in a single work all the interrelationships between hypoxia / ischemia and

epileptic seizures would be very unsubmitive , and certainly doomed to failure.

Consequently, it focuses

on some selected functional consequences of epileptic seizures and hypoxia / ischemia in acute

experiments and their possible influence . The common denominator of this thesis is the thesis that i

stimuli and insults of relatively low intensity can , despite the absence of major structural changes (cell death) can lead to significant and often long-lasting consequences.

In the theoretical section summarizes the main findings relating to the remote consequences of focal lesions , hypoxic / ischemic preconditioning , and provide an overview of models of acute hypoxia , global and focal ischemia and generalized seizures .