Cognitive impairment occurs more frequently in patients with epilepsy than in healthy controls (Binnie, 2003). There are many probable explanations for that, such as detrimental effects of the underlying disease, frequent seizures, subclinical epileptiform discharges, adverse effects of antiepileptic drugs, disturbances of sleep, comorbid diseases, or educational deprivation (Hirsch et. al. 2003; Aldenkamp et al. 2005).

Both repeated seizures and antiepileptic drugs may influence cognitive function. Antiepileptic drugs have a detrimental effect on the central nervous system and may affect cognitive function both in healthy controls and in patients with epilepsy. This is the crucial controversy in epileptology because the application of antiepileptic drugs is the main therapeutic principle.

The interplay between seizures and antiepileptic drugs is complex and might influence cognitive function either favorably or adversely. However, the influence depends on variety of factors. For instance, patients with syndrome of mesial temporal epilepsy caused by hippocampal sclerosis are highly susceptible to cognitive impairment especially if there is exposure to other detrimental factors (Mula et al. 2003).

The potential of antiepileptic drugs to impair cognition differs between drugs. Based on the experiments in healthy controls, it is believed that the impact of lamotrigine on cognitive impairment is negligible, while the effect of topiramate may lead to significant impairment of memory, speech fluency, and attention in as many as 20-40% of exposed persons (Kockelman et al. 2004). However, this effect is highly dependent on seizure activity. In patients with frequent seizures, irrespectively of epilepsy types, cognitive impairment may be the principal cause of disability that leads to low scores in quality-of-life inventories (Meador et al. 2005). It seems that cognitive impairments induced by seizures are reversible for most seizure types when seizures are controlled adequately. If the drug that impairs cognition, like topiramate stops all seizures, the net effect will be improvement in cognitive capacity because benefit from the improvement in seizure control will offset the potential of topiramate to impair cognitive functions. Additionally, at least for some seizure types there may be a kind of time window within which impairments are reversible. Exceeding the time window may result in irreversible impairment. This emphasizes the need to achieve complete and early seizure control. However, if therapy is ineffective, any degree of cognitive impairment produced by a drug augments the cognitive impairment produced by all other factors (Baeta et al. 2002).

We are conducting a study aimed to define factors influencing cognitive impairment in patients with pharmaco-resistant mesial temporal lobe epilepsy treated with topiramate. We will correlate scores on selected tests of memory, attention, verbal fluency and depression before and 6 months after the topiramate therapy with EEG and MRI findings in temporal lobes. We expect to find a subgroup of patients more prone to cognitive impairment when treated with topiramate with recognizable pattern of clinical/MRI/EEG/neuropsychological characteristics. We hope we could find hints suggesting that patients with certain characteristics should not be treated with particular drug because the risk of cognitive impairment might be increased compared with other options.

References: