Available evidence indicates that status epilepticus or even a single seizure result in neuronal damage. Neurodegeneration may be also encountered in other neurological conditions – stroke, head injury and chronic neurodegenerative diseases. Among conventional antiepileptic drugs (AEDs), valproate exerted potent neuroprotective effects to the hippocampus in a rat model of status epilepticus. Diazepam was effective in various rodent models of status epilepticus, preventing also the aberrant mossy fiber sprouting, thus inhibiting epileptogenesis. In some experimental models phenobarbital proved also a neuroprotective agent. Newer AEDs, felbamate, gabapentin, tiagabine and topiramate produced potent neuroprotection against seizure- or ischemia-generated neuronal death. The comparable profile of activity was shared by a potential AED, talampanel. Vigabatrin and levetiracetam were effective against ischemia-induced neurodegeneration whilst their protective effects in seizure models were variable. Some newer AEDs, for instance topiramate and lamotrigine, seem to possess also antiepileptogenic activity.

Neuroprotection exerted by some conventional or newer AEDs is evident but an important question arises whether this process has any positive impact on epileptogenesis and cognitive performance. The existing data are not consistent and the final outcome of AEDs is dependent upon the seizure model and the timing of their administration after the induction of status epilepticus. So far, experimental analysis of the synergistic effects of AEDs combinations has been available and a possibility arises that the synergistic combinations could provide not only potent neuroprotection but antiepileptogenic effects as well.