Epilepsy and epileptic syndrome as concomitants of diseases such as brain tumors, infection, trauma, developmental anomalies, and cerebrovascular disturbances are characterized by the periodic appearance of convulsions (status epilepticus). World statistics provide evidence that 1–2% of the population suffers from this affliction [20]. Such a wide distribution of convulsive states requires deep investigations of their origins and pathogenesis, as well as a search for therapeutic agents able to prevent and cure them. Efforts to achieve both of these aims are made using models of convulsive states, generally in rats and mice. Many such models have been described to date, because of the complexity and multifarious pathogenetic origins of epileptic syndrome. Comparison of models and the efficacies of test therapeutic agents identifies a sequence of stages in the development of convulsions and their cellular and molecular mechanisms. One obligate stage in generating convulsions is imbalance between inhibitory (GABAergic) and excitatory (glutamatergic) influences on neurons in different parts of the brain. This is indicated by the ability of glutamate and its agonists (kainate, AMPA, and NMDA) to elicit convulsive syndromes, which can be prevented by competitive and non-competitive blockers of glutamate receptors [3, 15, 18, 19, 24–26]. GABA receptor modulators, generally benzodiazepines, also weaken the manifestations of convulsions [11, 14, 16].

KEY WORDS: tremor, glutamate receptors, mice.
can be prevented not only by atropine, but also by glutamate antagonists or GABA receptor modulators [10, 23].

We report here our studies of tremor induced by another muscarinic cholinoreceptor agonist – arecoline. This tremor can be regarded as a model for the tremorous form of Parkinson’s syndrome [1]. The abilities of glutamate antagonists, i.e., selective blockers of open NMDA or AMPA receptor ion channels, to prevent arecoline tremors were studied. We have previously demonstrated the efficacy of some of these blockers in convulsive syndrome induced by intraventricular administration of NMDA and kainate, systemic administration of corasol, and corasol kindling [3–5], in haloperidol catalepsy [8], and in motor disorders induced by revesive rotation [2]. Preliminary results from the present study of arecoline tremor have been discussed previously [7].

**METHODS**

Experiments used white mongrel mice weighing 20–24 g from the Rappolovo supplier. Animals were kept in an animal house with free access to water and food. Mice received arecoline s.c. at a dose of 6 mg/kg. This dose induced tremor in 90–100% of animals. The following parameters were recorded: the latent period of onset of tremor, the duration of tremor, and the number of animals in which tremor appeared. In addition, the intensity of tremor was assessed, on a scale from 0 to 3: 0 identified the complete absence of signs of tremor, 0.5 identified minor twitches of the head muscles, 1 corresponded to periodic minor tremor of all muscle groups, 2 identified persistent marked tremor, and 3 corresponded to constant tremor with high-amplitude movements approaching convulsions, with...