pounds into cats caused high electroencephalographic synchronization and blocked the electroencephalographic arousal response. In mice, the mean intracerebroventricular convulsant doses of anisodamine and atropine were not affected by a number of neurotransmitter antagonists and ligands, except for diazepam which led to an increase, suggesting that central stimulation by these two drugs is GABAergic in nature [44]. The electroencephalographic responses to anisodine and scopolamine were antagonized by physostigmine or arecoline. The effects of anisodine appear to be similar to, but weaker than those of scopolamine [45, 46].

Both the synthesis and release of acetylcholine by rat brain cortex slices stimulated by high $K^+$ medium were enhanced by incubation with anisodine [47]. In addition, the release of acetylcholine from cat sensorimotor cortex was increased by anisodine [48]. Anisodine inhibited learning and the consolidation and retrieval of short-term memory in rats without affecting long-term memory. The action of anisodine could be blocked by the cholinergic agonist arecoline [49]. The effects of anisodine on learning and memory appeared to be specific. The results support the view that normal function of the cholinergic and especially of the muscarinic system is necessary for memory formation in mammalian brain [50].

In experimental studies, anisodamine was as potent as atropine sulfate in spasmolytic activity. In vitro, it antagonized the contraction of intestine and bladder smooth muscle induced by acetylcholine. In vivo, anisodamine reduced intestinal tension to the same extent as atropine. Anisodamine, however, had fewer side effects on salivary glands, pupils, and the central nervous system; its central action was only $^1/_6-^1/_{20}$ as potent as atropine [51, 52].

Anisodamine inhibited histamine-induced bronchial smooth muscle contractions in guinea pigs [53]. In chicken, i.v. injection of anisodamine did not cause any change in tracheal ciliary movement, but did so in animals treated with cigarette smoke. In young rats the change in serous tracheal exudation under cold stress was found to be inhibited by anisodamine. Acetylcholine content in trachea increased under cold stress without affecting that in brain. Apparently, the expectorant effect of anisodamine was mainly due to its peripheral action [54]. Phagocytosis by macrophages obtained by bronchoalveolar lavage from dogs with oleic acid-induced lung injury was increased by anisodamine treatment. The surface of the macrophages from anisodamine treated animals was smooth and the cells contained many lipid particles. The number of macrophages and polymorphonuclear leukocytes decreased after anisodamine treatment [55]. This effect may result from an inhibition of oleic acid-induced leukocyte accumulation in the microvasculature [56]. Anisodamine and anisodine promoted in vivo phagocytosis of i.v. injected colloidal $^{198}Au$ by mouse liver and spleen [57].

The $LD_{50}$ of anisodamine in mice was 350–430 mg/kg by i.p. and 123 mg/kg by i.v. administration. The minimal lethal dose by oral administration of anisodamine was 1600 mg/kg [51]. A pharmacokinetic study showed that the highest concentration of anisodamine, 15 min after i.v. injection into rats, was observed in the kidney. At 30 min, however, anisodamine concentration was highest in the pancreas followed by lung, heart, kidney, and spleen; the lowest concentration was observed in brain and plasma. Anisodamine had no significant accumulation effect in tissues. During the 24 h period after injection of anisodamine, urinary excretion was 39% in rats and 42%–49% in patients [58]. The biphasic half-lives of anisodamine injected i.v. into rats were 13 and 70 min [59].