sure and heart rate [29]. In rats, anisodamine lowered the mean arterial pressure, cardiac output, and contractility of the left ventricle. After coronary ligation, anisodamine also reduced the heart rate. It apparently reduced oxygen consumption by the heart and thus may protect the heart during insufficient blood oxygen supply [30]. The beneficial effect of anisodamine in dogs after ligation of the coronary artery was also proven by electrocardiography. Elevation of the ST segment of the electrocardiogram in experimental acute myocardial ischemia was markedly decreased by i.v. administration of anisodamine [31]. Intraperitoneal administration of anisodamine to rats immediately after ligation of the coronary artery decreased the ischemic damage as evidenced by an electron microscopic study. Ultrastructural changes in cell nuclei, mitochondria, sarcoplasmic reticulum, and capillary vessels following ischemic insult were markedly decreased by anisodamine treatment [32, 33].

Anisodamine also increased the arteriolar diameter and red cell flow velocity in the skeletal muscle of the anesthetized rat. Arteriolar flow was three to four times greater than under control conditions. Mean arterial blood pressure was reduced immediately by 15%–20% for 60 min before normalization [34].

Intraperitoneal injection of anisodamine into rats for 21 days increased the cAMP concentration and cAMP/cGMP ratio in liver, kidney, and plasma. In mice, i.p. injection of anisodamine for 28 days increased bone marrow colony-forming cells and nucleated cells in the bone marrow. Microcirculation of the bone marrow was also increased by anisodamine as detected by capillary hyperproliferation; however, in vitro addition of anisodamine to marrow cell cultures did not increase the colony-forming cells, suggesting that anisodamine increases bone marrow function by promoting microcirculation [35].

Anisodamine did not show obvious effects on pulmonary microcirculation kinetics and pulmonary water and protein transport in sheep with chronic lung fistulas. In sheep with pulmonary injury induced by air embolization, anisodamine treatment at the early stage reduced water exudation from pulmonary capillary vessels apparently by reducing pressure in the vessels [36].

The phase transition temperature of dipalmitoyl phosphatidylcholine liposomes was decreased in the presence of the tropane alkaloids anisodamine, anisodine, scopolamine, and atropine in a concentration dependent fashion, reflecting a concentration dependent increase in the fluidity of phospholipid liposomes. These observations offer an explanation of the molecular mechanism of drug action on biological membranes [37, 38]. Anisodamine displayed the highest activity followed by atropine, scopolamine, and finally anisodine [39]. The interaction of anisodamine with dipalmitoyl phosphatidylcholine and dipalmitoyl phosphatidic acid liposomes was interpreted as a trigger mechanism [40]. Raman spectroscopic studies showed that the interaction of anisodamine with the neutral phospholipid membrane is essentially a polar-polar interaction [41]. The increase in membrane fluidity appears to be a result of the interaction of anisodamine mainly with the polar head of the phospholipid, affecting the hydrophobic region of the bilayer as detected by electron spin resonance [42, 43] and fluorescence polarization studies [43].

Electroencephalographic studies in conscious rabbits after intracerebroventricular injection of anisodamine, atropine, anisodine, and scopolamine suggested that anisodine and scopolamine are mainly central nervous system depressants, whereas anisodamine and atropine are stimulants. Intravenous injection of the four com-