effect of anisodamine was probably partly due to a lysosome stabilizing action [14]. In hemorrhage shocked rats, anisodamine increased arterial blood pressure following retransfusion of blood and prolonged shock compensation time. Cathepsin D activity in plasma and liver lysosomes was also markedly decreased by treatment with anisodamine [15].

Anisodamine significantly increased the survival time of traumatically shocked rats but had no effect on cathepsin D activity; however, plasma myocardial depressant factor accumulation was decreased by anisodamine, indicating that prevention of the formation of this factor may be one of the protective mechanisms of the drug [16].

The antishock effect of anisodamine was studied in rabbits shocked by experimental superior mesenteric artery occlusion after i.v. and direct intraenteral infusion. Shock was reversed in a number of treated animals, the mortality rate decreased, and blood pressure increased. Ventral vagotomy did not prevent shock induced by superior mesenteric artery occlusion. Thus, the antishock effect of anisodamine may be due to the prevention of production of a shock inducing intestinal factor and not via blockade of the ventral vagal cholinergic system. The elevation of serum acid phosphatase observed in rabbits with superior mesenteric artery occlusion did not occur in anisodamine pretreated animals, indicating prevention of release of lysosomal enzymes induced by intestinal hypoxia [17, 18].

In *Escherichia coli* endotoxin shocked dogs, abnormalities in pulmonary circulation and function were markedly improved and the average survival time was prolonged by injection of anisodamine after the endotoxin injection [19]. The hemodynamic parameters in endotoxin shocked dogs were normalized or improved following i.v. administration of anisodamine; these improvements included restoration of systemic arterial pressure and total systemic peripheral resistance as well as increases in cardiac output [20, 21], indicating that the effects of anisodamine are not confined to vasodilatation and may be relatively complex.

In rabbits, infused i.v. anisodamine antagonized the decrease in arterial blood pressure and heart rate observed during induction of shock by *Escherichia coli* endotoxin. A slight decrease in renal sympathetic nerve discharge was observed in anisodamine treated rabbits under endotoxin shock in contrast to an increase in untreated shocked rabbits. No such effects were observed in endotoxin shocked rabbits when a bolus injection of anisodamine was given intracerebroventricularly. Anisodamine given by i.v. infusion also decreased renal sympathetic nerve discharge in normal rabbits but did not affect blood pressure or heart rate [22].

Anisodamine induced the release of serotonin from platelets in correlation with a protective effect against endotoxic shock in dogs [23]. Intravenous injection of endotoxin induced a significant increase in plasma levels of cAMP and cGMP in dogs. Anisodamine reduced the plasma levels of cGMP in control dogs and apparently prevented the rise in plasma levels of cGMP in shocked dogs whose survival times were prolonged [24, 25].

Plasma levels of 6-keto-$PGF_{1\alpha}$, a stable metabolite of prostacyclin, were markedly elevated in dogs administered *Escherichia coli* endotoxin; blood pressure decreased simultaneously. Intravenous injection of anisodamine countered these effects [26–28].

Anisodamine injected i.v. into anesthetized dogs at doses higher than those causing cholinergic blockade induced a dose dependent decrease in arterial blood pres-