POSSIBLE MEDIATION BY ENDOGENOUS PROSTAGLANDIN OF THE SIALAGOCIC EFFECT INDUCED BY BRADYKININ IN DOGS (*)

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The increase of the dog submaxillary gland secretion induced by bradykinin (3 μg/kg) by intracarotideal route is low, shortlasting and tachyphylatic. It is unaffected by hexamethonium (2 mg/kg, i.v.) and is still present in chronically denervated glands. The effect is abolished by atropine (1 mg/kg, i.v.), morphine (10 μg/kg, i.v.) or following the ganglionic block by depolarization induced by nicotine (1 mg) by intracarotideal route.

A possible adrenergic mediation of bradykinin effect was excluded, since: a) the polypeptide does not increase the secretion when injected into the artery leading towards the superior cervical ganglion; b) isoproterenol, but not bradykinin, elicits secretion in the atropinized animals; c) even though bradykinin release catecholamines, from the adrenal gland, there is no secretion in the right submandibular gland when the injection is made on the left side. The last observation also makes improbable the participation of the central nervous system in bradykinin effects.

Our results indicate that bradykinin interacts with receptors sensitive to blockade by morphine which are probably located in the postsynaptic membrane of cells of the parasympathetic ganglion of the submaxillary gland. The mechanism of action of McN-A-343 and pilocarpine resembles that of bradykinin;they differ in their susceptibility to blockade by indomethacin. This nonsteroidal antinflammatory agent was more specific in blocking bradykinin and the sialagogic effect induced by chorda stimulation (Grellet, 1968; Corrado, 1974). The correlation of these results with the finding that Prostaglandin PGF 2 alpha, but not PGE, is a very potent sialagogic agent in the dog (Corrado, 1974), suggests that the effect of the polypeptide could be mediated by endogenous prostaglandins which probably have an important role in salivary secretion physiology.

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