estimated from the existence of spontaneous impulses discharged without chemical stimulation. That is, stimulation with K-benzoate may shift the depolarized level of the postsynaptic membrane to a hyperpolarizing direction. This corresponds to the phenomenon of the so-called disfacilitatory hyperpolarization found in some cells[3,6,7]. The K-benzoate-induced hyperpolarization at the postsynaptic axon membrane in the surrounding papillae might spread out electrotonically up to branching point of a gustatory nerve fibre, as has been suggested by Miller[2]. The hyperpolarization spread will add, at the branch point, to the depolarization elicited by NaCl stimulation of the other taste bud, so that the resultant initiation of impulses to NaCl may be reduced.

1 We thank Miss M. Oguro for her help in preparing the manuscript. Present address of L.M. B. is: Department of Biological Science, Florida State University, Tallahassee (Florida 32306, USA).
2 I.J. Miller, Jr, J. gen. Physiol. *57*, 1 (1971).
3 T. Sato and L.M. Beidler, J. gen. Physiol. *66*, 735 (1975).
4 L.M. Beidler, in: Progress in Biophysics and Biophysical Chemistry, vol. 12, p. 107. Ed. J.A.V. Butler, H.E. Huxley and R.E. Zirkle. Pergamon Press, Oxford 1961.
5 I.J. Miller, Jr, in: Olfaction and Taste. Ed. D.A. Denton and J.P. Coghlan, vol. 5, p. 217. Academic Press, New York 1975.
6 J. Toyoda, H. Nosaki and T. Tomita, Vision Res. *9*, 453 (1969).
7 R. Llinás, J. Neurophysiol. *27*, 1117 (1964).

The effects of dopamine on renin release in the isolated perfused rat kidney

T. Quesada, L. Garcia-Torres, F. Alba and C. Garcia del Rio

Departamento de Fisiologia y Bioquimica, Facultad de Medicina, Granada (Spain), 1 November 1978

Summary. In the isolated and perfused kidney of the rat, the stimulant effect of dopamine on renin release is blocked by propanolol and not by haloperidol. This suggests that the release of renin induced by dopamine is due to the activation of β-receptors.

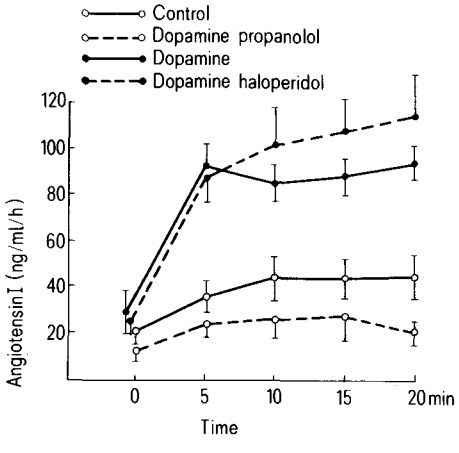
It has been shown that renin secretion is increased by dopamine[1-4]. However, the mode of action of dopamine on renin release is still not clear. It has been suggested that dopamine could act through dopamine[3] or β-adrenergic receptors[4]. Using the isolated perfused kidney of the rat, we have studied the direct effect of dopamine on renin release and the influence on this effect of haloperidol, a dopamine receptor blocker, and propanolol, a β-adrenergic blocking agent.

Materials and method. The method of isolation and perfusion of kidney has previously been described[5] elsewhere. The perfusion fluid was Krebs-Ringer dextran saline equilibrated with 95% O_2 and 5% CO_2 at 37 °C and was delivered as pulsatile flow at a constant rate (8 ml/min).

Perfusion pressure was monitored (Desvices M2). Samples were collected at 0, 5, 10, 15 and 20 min. Drugs were administered from 1 to 20 min. Renin was measured in the perfusion fluid by incubating with rat renin substrate and radioimmunoassay of angiotensin I, and the results are expressed as ng/ml/h of angiotensin I generated. The drugs used were: dopamine (Merck) at doses of $4.7\times$ (10^{-8} 10^{-7} and 10^{-5} M) dissolved in perfusion buffer with 6×10^{-4} M of ascorbic acid, propanolol (ICI) (2×10^{-4} M) and haloperidol (Latino SA) (5×10^{-5} M).

Results. The perfusion pressure was not altered by any of the drugs used in the present study. Neither propanolol (n = 7) nor haloperidol (n = 7) caused significant changes on renin secretion compared with the control kidney (n = 14). Dopamine was administered in 3 doses: 4.7×10^{-8} M and 4.7×10^{-7} M did not alter the renin release, but the 3rd doses 4.7×10^{-5} M produced a significant increase ($p < 0.01$) of renin release. The elevation of the renin secretion obtained with this dose of dopamine was not altered by haloperidol but was inhibited significantly ($p < 0.01$) by propanolol (figure).

Discussion. It is clear from the present results that dopamine (4.7×10^{-5} M) causes a significant increase of renin release in the isolated perfused kidney of the rat. Low doses of dopamine (10^{-8} M and 10^{-7} M) have been shown to have a stimulant effect on renin 'in vitro', but the monoamine oxidase inhibitor, pheniprazine, was used[4]. The possibility of an intrarenal conversion of dopamine into noradrenaline could be discarded, since there were no changes in the perfusion pressure during any experiment. Therefore, the action of dopamine on renin secretion described here could be attributed to its direct effect on the juxtaglomerular cells through an activation of β-receptors. The increase in renin release mediated by dopamine was blocked completely by propanolol and not by haloperidol.



The effect of dopamine (n = 6), dopamine plus propanolol (n = 7) and dopamine plus haloperidol (n = 9) on renin secretion on the isolated perfused kidney. Values given are means±SEM. For significances see text.

1 C.B. Ayers, R.H. Harris and L.G. Lefer, Circulation Res. *103*, suppl. 1, 24 (1969).
2 C.S. Wilcox, M.J. Aminoff, A.B. Kurtz and J.D.H. Slater, Clin. Sci. molec. Med. *46*, 481 (1974).
3 J.L. Imbs, M. Schmidt and J. Schwartz, Eur. J. Pharmac. *33*, 151 (1975).
4 D.P. Henry, W. Aoi and M.H. Weinberger, Endocrinology *101*, 279 (1977).
5 W.S. Peart, T. Quesada and I. Tenyi, Br. J. Pharmac. *54*, 55 (1975).