rostral to and the lower margin of 2 or 3 mm caudal to the obex in the medulla oblongata, and that
the superficial sagittal section of this region of about 1 to 1.5 mm depth abolished only the third
component of response to the vagus nerve stimulation (2). From these experiments, it was presumed
that some specific pathways existed in the medulla for the vago-vagal, splanchnic-vagal and sciatic-
vagal reflexes.

This communication reports the effects of some drugs on these reflex responses recorded in the
right cervical vagus nerve in cats.

After operation under ether animals were anesthetized with chloralose (50 mg/kg, intravenously),
immobilized by repeated doses of gallamine triethiodide and artificially ventilated. Drugs were ad-
ministered into the saphenous vein.

Strychnine (100 µg/kg) augmented mainly the first component of response to the stimulation of
the sciatic nerve (Fig. 1-A). This effect lasted over sixty minutes. On the other hand, mephenesin
(30 mg/kg) reduced this component for about thirty minutes (Fig. 1-B). These two drugs had much
less effects on the responses to the vagus and the great splanchnic nerve stimulation. The effect
of mephenesin was decreased by the preceding administration of strychnine.

Pentobarbital (15 mg/kg) reduced or abolished all of these reflex responses. Among them, the
first two components of response to the vagus nerve stimulation and the first component of response
to the sciatic nerve stimulation were greatly affected, but the third component of response to the
vagus nerve stimulation and the first component of response to the great splanchnic nerve stimula-
tion were fairly resistant to this dose of the drug (Fig. 1-C).

Relatively large dose of GABA (200 mg/kg) reduced only the response to the vagus nerve
stimulation, particularly the third component. In most cases this effect of GABA disappeared within
about fifteen minutes. Responses to the great splanchnic and the sciatic nerve stimulation were
not affected by this drug (Fig. 1-D).

REFERENCES
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TRYPAMINE RECEPTORS AND TYRAMINE

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Considerable evidence has accumulated to support hypothesis of Burn and Rand (1) that tyramine
acts by releasing catecholamine from the tissue stores (2-5). Vane (6) observed that tyramine pro-
duced a stimulant effect on rabbit duodenum and a biphasic effect on rat stomach strip. This effect
was inhibited by bromlysergic acid diethylamide (BOL) and phenoxybenzamine. On this basis, he
suggested that the action of tyramine on these preparations was due to an action on tryptamine
receptors. Since BOL and phenoxybenzamine block 'D' type of tryptamine receptors (7) it may be
presumed that tyramine acts on 'D' receptors. To test this presumption we have studied the effect
of tyramine on rat uterus which contains only 'D' type of tryptamine receptors.
Experiments were performed on isolated uteri obtained from rats pretreated with diethyl stilbestrol (10 μg/100 g) for two days. Some of these rats were also pretreated with reserpine (15 mg/100 g) for two days. The uteri were suspended in 10 ml bath containing aerated de Jalon's fluid. The contractions were recorded on the smoked kymograph paper with the help of a lever having frontal writing point. The drugs used were tyramine hydrochloride and 5-hydroxytryptamine creatinine-sulphate.

Tyramine did not produce any effect on rat uterus in concentrations ranging from 1 μg/ml to 1000 μg/ml. The effect of tyramine was also studied on uteri obtained from reserpinised rats to rule out the possibility of released catecholamines having masked the stimulant action of tyramine. However, in this series of experiments also tyramine failed to produce a contraction. In both the series the uteri were sensitive to concentrations of 5-HT as low as 0.025 μg/ml. Considering the work of Vane (6) tyramine should have produced a stimulant effect on rat uterus if it was acting on 'D' type of tryptamine receptors. Since tyramine did not cause a contraction of rat uterus we conclude that it does not act on 'D' type of receptors.

No substance is yet known which activates only one type of tryptamine receptor. There may be a theoretical possibility that tyramine acts on 'M' type of tryptamine receptors only, since one of the test objects taken by Vane (6) i.e., rabbit duodenum contains both 'M' and 'D' type of tryptamine receptors. Dhawan, Gupta and Dhawan (8) reported the presence of tryptamine receptors in the brain which are responsible for the central hypotensive response of 5-HT (9). If tyramine was acting on the tryptamine receptors, it should have, like 5-HT, produced hypotension on intracerebroventricular administration. But on the contrary, it produces a pressor response (10). This rules out an action of tyramine on tryptamine receptors in central nervous system as well. Moreover, it has been shown that the central pressor action of tyramine is due to the release of acetylcholine (10). It thus appears that tyramine does not act on tryptamine receptors and that the results obtained by Vane (6) can be explained on the basis that tyramine releases acetylcholine (11).

It could be argued that tyramine should have contracted the rat uterus if it releases acetylcholine. The failure of tyramine to cause the contraction may be due to a minimal content of acetylcholine in the uterine tissue—a fact supported by the observation that physostigmine also fails to stimulate the uterus (12). Thus it appears that tyramine produces acetylcholine-like effects only in tissues rich in acetylcholine e.g. brain (10) and rabbit atrium and ileum (11).

REFERENCES