NEURONAL COMPOSITION OF THE INTRAMURAL GANGLIA OF THE GASTRO-INTESTINAL TRACT

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The neuronal composition of the intramural plexuses of the gastro-intestinal tract in cats was studied by luminescence microscopy. The intramural ganglia were found to contain three populations of neurons. The neurons of the first population showed no specific catecholamine fluorescence. Those of the second population had fluorescence of the perikaryon and processes. The perikarya of the third neuron population were not fluorescent but adrenergic synapses were seen on their surface. Intraperitoneal injection of exogenous noradrenalin (1 mg) led to an increase in the number of neurons of the third population with specific fluorescence. On this basis it was concluded that as well as parasympathetic intramural neurons the intramural ganglia of the gastro-intestinal tract also contain sympathetic cells, most of which exhibit specific fluorescence only after their mediator content has been artificially increased.

Key words: gastro-intestinal tract; catecholamines; intramural nerve cells; luminescence.

Until recently the view has been held in the literature that intramural motor neurons, or Golgi type I cells, in the walls of the gastro-intestinal tract are mainly, if not entirely, parasympathetic in nature [3, 6, 7, 18, 21]. Meanwhile, a diametrically opposite view is held about the nature of these intramural intestinal plexuses. Van Campenhout [10, 11] showed by experimental morphological studies that neurons of the intestinal plexuses are sympathetic in origin and that the vagus nerves terminate in sympathetic structures only on some of the intramural ganglion cells.

Recent luminescence microscopic investigations have shown that in the intermuscular plexus of certain parts of the gastro-intestinal tract in mammals adrenergic neurons are present. Furness and Costa [13, 16] found adrenergic neurons in the wall of the small intestine of the guinea pig. Krokhina [4] detected specifically fluorescent neurons in the intermuscular plexus of the pyloric part of the stomach and in the duodenum in the cat.

As well as parasympathetic and sympathetic neurons, neurons whose perikarya are not fluorescent in intact animals but which possess adrenergic synapses have been found in the intermuscular plexus of the esophagus [8, 12, 17], stomach [9], and intestine [1, 4, 19, 22, 23]. The nature of these neurons is not yet clear. Some workers [18, 22] have considered that these nonluminescent neurons are parasympathetic and have concluded that inhibition of processes in the gastro-intestinal tract is brought about by parasympathetic neurons.

There is information in the literature that among adrenergic neurons in intact animals there are some that do not fluoresce, for they are distinguished by rapid metabolism of the mediator in the cytoplasm. Because of the low noradrenalin concentration it cannot be detected histochemically in the perikarya of these neurons [14]. By increasing the mediator concentration artificially in the cytoplasm of these neurons, they can then be demonstrated histochemically.

Because of these observations the writers postulated that neurons whose bodies do not fluoresce, but which have adrenergic synapses occurring in the gastro-intestinal tract, are also adrenergic neurons with

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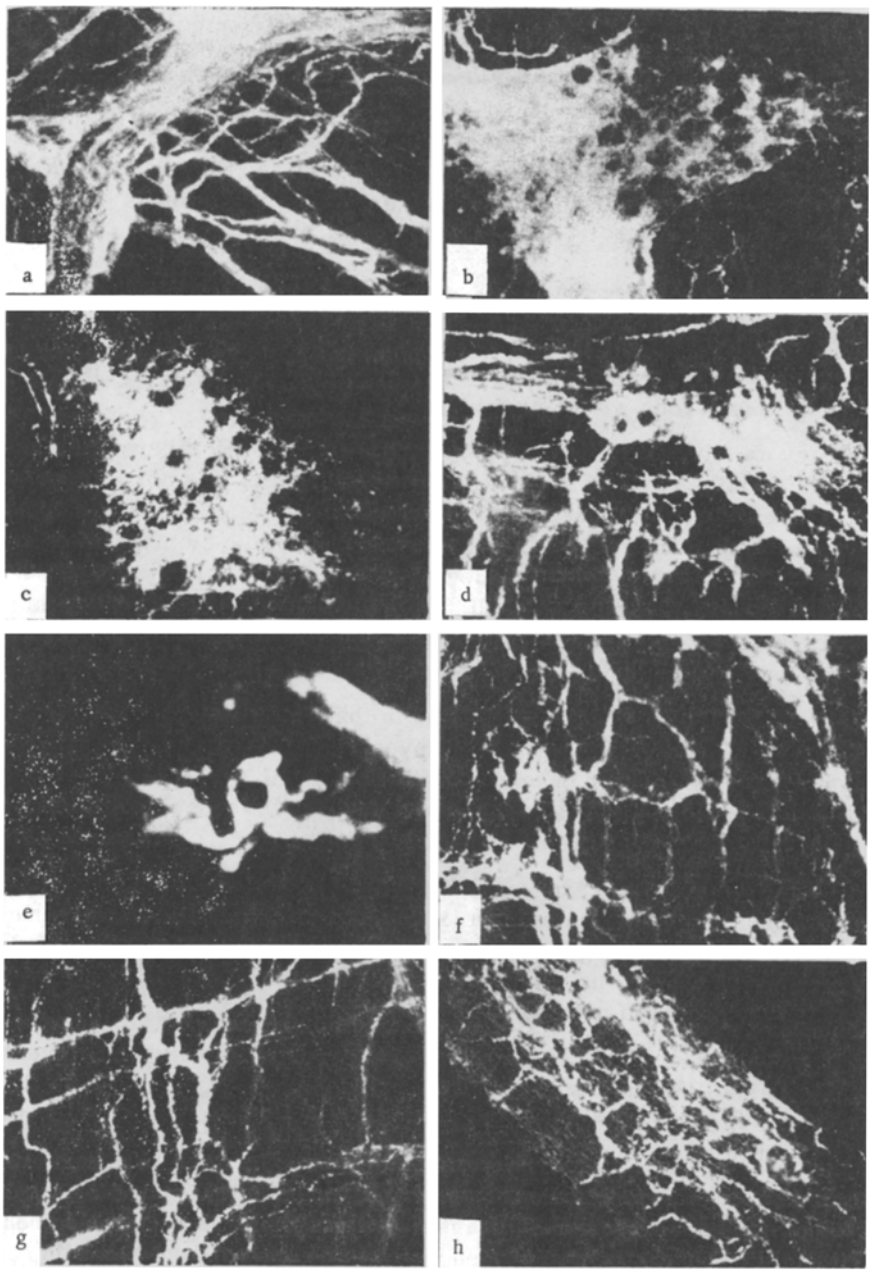

Fig. 1. Catecholamines in intramural adrenergic neurons and nerve fibers in walls of cat small and large intestine after intraperitoneal injection of exogenous noradrenalin: a) adrenergic fibers in connections between ganglia of Auerbach's plexus in small intestine (120×); b and c) adrenergic neurons in intramural ganglia of ileum and large intestine respectively, nuclei of neurons do not luminesce (360×); d and e) isolated adrenergic neurons in intermuscular plexus of small and large intestine respectively (360×); f and g) terminal adrenergic plexus in outer longitudinal layer of smooth muscle of large intestine (120×); h) adrenergic innervation of muscle coat of artery from wall of large intestine (120×).

a low mediator content. The object of the investigation described below was, by artificially increasing the noradrenalin concentration in the perikaryon of these neurons and by making use of the intrinsic properties of sympathetic nerve cells, to "assimilate" exogenous noradrenalin, to "compel" these neurons to fluoresce specifically, and in that way to clarify their nature.

EXPERIMENTAL METHOD

Noradrenalin (1 mg) was injected intraperitoneally into the cats 2 h before the material was taken. The animals were killed by exsanguination under inhalational anesthesia. Pieces of all parts of the small and large intestine were washed with physiological saline to remove the contents. Sections were cut for histochemical investigations [5] by the method of Falck and Owman [15]. Unembedded sections were studied and photographed with the ML-2 microscope. Autoluminescent structures were identified by heating the sections without paraformaldehyde.

EXPERIMENTAL RESULTS

As was stated previously [4], many varicose adrenergic fibers run "en passant" through Auerbach's plexus in intact animals, without touching nonluminescent nerve cells. Other adrenergic fibers terminate in synapses on some of the neurons with nonluminescent bodies.

After preliminary noradrenalin loading the number of adrenergic fibers passing through the ganglia of Auerbach's plexus and in the communications between the ganglia was not appreciably increased but the character of their luminescence was sharply altered. The adrenergic fibers lost their marked beading and their luminescence was sharply intensified (Fig. 1a). Whereas in the stomach and duodenum of intact animals single nerve cells with specific luminescence were found, preliminary noradrenalin loading led to the appearance of whole groups of neurons and small ganglia in the small and large intestine in which all the neurons gave specific luminescence, which was absent only in their nuclei (Fig. 1b, c). Isolated adrenergic nerve cells also were found (Fig. 1d, e).

Noradrenalin loading caused a marked increase in the intensity of luminescence also in the axons of the terminal plexus in the circular and longitudinal layers of smooth muscles (Fig. 1f, g). These figures clearly show an increase in the number of terminal fibers per unit of innervated area of the muscle. The luminescence of the adrenergic axons was intensified equally in the muscle coat of the intramural vessels in the walls of the small and large intestine (Fig. 1h).

Ability to take up and assimilate exogenous noradrenalin from the blood stream is a property restricted to neurons of the sympathetic nervous system [14, 20, 24]. On this basis it can thus be concluded that in Auerbach's plexus in the small and large intestine there are many neurons that become capable of luminescence after administration of exogenous noradrenalin to the animal; consequently, these neurons can justifiably be regarded as sympathetic in nature.

The observations described above confirm earlier views [4] that the intermuscular plexus of the gastro-intestinal tract contains three neuron populations: nonluminescent parasympathetic neurons; sympathetic neurons whose perikarya and processes are luminescent in intact animals; sympathetic neurons with a low concentration and rapid rate of metabolism of catecholamines in the cytoplasm, which become luminescent only as the result of an artificial increase in the mediator concentration in their cytoplasm.

The possibility cannot be ruled out that the degree of luminescence of the neurons depends on differences in their functional state, i.e., that the adrenergic neurons, like the others, do not function all at the same time. There is morphological evidence in support of this hypothesis [2, 5].

Since the neurons do not all function at the same time, some of them are observed at a time when they do not possess adequate reserves of mediator, but others, on the other hand, are observed when they are once again ready for function and they contain a sufficiently high concentration of mediator, capable of histochemical demonstration. This hypothesis regarding differences in the times of function of the neurons applies not only to their perikarya but also to their postganglionic fibers which form the terminal synaptic structures distributed among the smooth muscle and other tissues in the walls of the intestine.

In intact animals substantially fewer terminals are found in the smooth-muscle layer of the intestinal wall. Their number rises sharply after administration of exogenous noradrenalin.

Regardless of the truth or otherwise of these hypotheses, what is certain is that there are many adrenergic neurons in the intramural ganglia of the gastro-intestinal tract.

LITERATURE CITED

1. A. P. Amvros'ev, in: Structural and Functional Organization of Autonomic Ganglia [in Russian], Minsk (1973), p. 5.

2. Yu. M. Zhabotinskii, Normal and Pathological Morphology of Autonomic Ganglia [in Russian], Moscow (1953).
3. N. G. Kolosov, Innervation of the Human Digestive Tract [in Russian], Leningrad (1962).
4. E. M. Krokhina, Byull. Éksperim. Biol. i Med., No. 3, 105 (1973).
5. E. M. Krokhina, Functional Morphology and Histochemistry of the Autonomic Innervation of the Heart [in Russian], Moscow (1973).
6. B. I. Lavrent'ev, in: Morphology of the Autonomic Nervous System [in Russian], Moscow (1946), p. 13.
7. W. Abel, J. Anat. (London), 47, 35 (1912).
8. H. Baumgarten and W. Lange, Z. Zellforsch., 95, 589 (1969).
9. H. Baumgarten, Z. Zellforsch., 83, 133 (1967).
10. E. Van Campenhout, Bull. Acad. Roy. Med. Belg., 10, 256 (1945).
11. E. Van Campenhout, Arch. Biol. (Paris), 58, 1 (1947).
12. M. Costa and G. Gabella, C. R. Ass. Anat., 141, 752 (1968).
13. M. Costa, J. B. Furness, and G. Gabella, Histochemie, 25, 103 (1967).
14. B. Ehinger and B. Falck, Acta Physiol. Scand., 78, 132 (1970).
15. B. Falck and C. Owman, Acta Univ. Lund. Sect. II, 7, 5 (1965).
16. J. Furness and M. Costa, Z. Zellforsch., 120, 346 (1971).
17. G. Gabella, Rev. Histochem. Norm. Patol., 14, 155 (1968).
18. C. Hill, Phil. Trans. Roy. Soc. (London), 215, 355 (1927).
19. D. Jacobowitz, J. Pharmacol. Exp. Ther., 149, 358 (1965).
20. T. Malmfors, Acta Physiol. Scand., 64, 248 (1965).
21. E. Muller, Arch. Mikr. Anat., 94, 208 (1920).
22. K. Norberg, J. Neuropharmacol., 3, 379 (1964).
23. D. G. Silva, G. Ross, and L. W. Osborne, Am. J. Physiol., 220, 347 (1971).
24. L. Stjärne, Acta Physiol. Scand., 62, 228 (1964).