TRANSMISSION AND BLOCK IN SYMPATHETIC GANGLIA

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It has been shown by two distinct approaches that the cells in sympathetic ganglia are not a homogeneous population, at least pharmacologically. In the first approach, it has been shown that those cells which give rise to postganglionic fibres with vasoconstrictor properties are stimulated by both nicotine and acetyl choline and are inhibited by nicotine (larger doses), tetraethylammonium iodide and curare, whilst those cells giving rise to fibres with vaso-dilator activity are stimulated only by acetylcholine and are not paralysed by the above paralysants except in very large doses (2).

In the second case, five ganglionic blocking agents, (*d*-tubocurarine and its dimethyl derivative, pentamethonium iodide, tetraethylammonium bromide and "Pendiomide") showed different results quantitatively when tested for blocking activity on the superior cervical ganglion. The two groups of ganglionic cells tested in this case were those innervating the blood vessels of the ear and those supplying the nictitating membrane. For example with dimethyl-tubocurarine, the duration of paralysis on stimulation of the preganglionic fibres in the presence of the drug was 12½ minutes for the vasoconstriction of the ear vessels and 3½ minutes for the nictitating membrane, whilst the time to complete recovery was 30 minutes and 12 minutes respectively. Similar differences were obtained with the other agents (3). Thus we see that there are at least two pharmacologically distinct groups of cells in the superior cervical ganglion. Difficulty of penetration to the two groups cannot explain the results, for with pentamethonium iodide the relative duration of paralysis (and time to recovery) of the cells giving rise to the vasoconstrictor fibres and to those passing to the nictitating membrane is just the reverse of that with dimethyl-tubocurarine.

It now becomes necessary to consider this differentiation of ganglion cells in the light of the transmission mechanism within the ganglion. In view of Eccles (1) abandonment of the theory of electrical transmission for synaptic bridging in the cord it can be said with some certainty that transmission in the ganglion is humoral. It remains to name the transmitter. Let it be assumed that it is acetylcholine, and further that the discharge of the ganglionic cell is due to a depolarisation of the soma below a certain critical level. If the ganglionic cells are differentiated pharmacologically, then perhaps one of three mechanisms may be possible. a) Acetylcholine is not the only transmitter. This may seem surprising at first, till one remembers that in all probability acetylcholine is not the transmitter in the cord and a new humoral substance must be sought in this situation (1). b) Transmission is not via acetylcholine alone but due to the combined effect of this ester plus some other substance, *e.g.*, potassium ions or epinephrine. There is a great deal of supporting evidence for this suggestion. c) There is only one transmitter but the critical level of depolarisation ("local response") varies

in each group of cells. At present there is little evidence to support this mechanism and further work with internal microelectrodes is necessary.

In conclusion, the hypothesis that there exist in ganglia distinct groups of cells, each group innervating a specific region of the body, opens up new problems in humoral transmission.

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