CENTRAL AND SENSORY TRANSMISSION

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Attempts to analyze the distribution of cholinergic neurones from the gross determination of acetylcholine, cholinacetylase and cholinesterase in the nervous system are open to several sources of error. Some of these problems can be investigated by histochemistry, which is unfortunately restricted as yet to the distribution of cholinesterase.

In the first place, it has been assumed as a working hypothesis that all the cells in a functional group have similar enzyme contents. However, in the spinal posterior root ganglia, whereas the majority of the ganglion cells contain very little cholinesterase I (true, specific, etc.) some of the cells contain quite respectable amounts. This suggests that whilst most of the posterior root afferents may have non-cholinergic terminals, some afferents, possibly those subserving one particular sensory modality may have cholinergic terminals.

A second assumption made about cholinergic neurones is that the acetylcholine system is distributed either uniformly throughout the neurone or is specifically concentrated at the synaptic terminals. Histochemical localization of cholinesterase I in the dog central nervous system using the 5-bromoindoxyl acetate method (3) has shown that in cells containing large amounts of enzyme (e.g., anterior horn cells) the highest concentration of enzyme is found in the somatic cytoplasm, and that the concentration of enzyme falls off in the dendrites. There is no enzyme in either the nucleus or nucleolus and no evidence that the enzyme is specifically concentrated at the cell surface. Bearing in mind the pitfalls of histochemical methods, this suggests that the high concentration of cholinesterase found in grey matter is largely due to the presence of cell bodies rather than to fine unmyelinated fibres and their synaptic terminals. The significance of the high concentration of enzyme in the somatic cytoplasm is puzzling. One possibility suggested by Weiss (4) is that many enzymes may be formed in the soma and carried down the axon by axoplasmic flow. MacIntosh and Work (2) have attempted to test this point by treating cats with near lethal doses of DFP and then following the regeneration of cholinesterase in the anterior spinal roots. The enzyme appeared to recover first in the proximal part of the roots as predicted by Weiss' hypothesis. On the other hand it may be that the somatic cholinesterase is concerned with the control of the excitability of the postsynaptic membrane indirectly, through the internal acetylcholine. As recently pointed out by Coombs *et al.* (1), the properties of the somatic membrane may differ significantly from those of peripheral axons.

Finally, it should be noted that as far as cholinesterase content is concerned, cells in the central nervous system do not fall into two clear-cut categories of being with or without the enzyme. Rather, among different cell types there is a continuous spectrum of enzyme concentrations.

REFERENCES

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