ELECTRON-MICROSCOPIC INVESTIGATION OF THE LOCALIZATION OF PRIMARY AFFERENT FIBER SYNAPSES IN THE COCHLEAR NUCLEI OF THE BRAIN STEM

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The character of distribution of synapses in the cochlear nuclei of the cat brain stem was determined electron-microscopically by unilateral destruction of the spiral ganglion. Most degenerating nerve endings of the afferent fibers of the auditory nerve are located in the ventral cochlear nucleus, and a smaller number in the dorsal nucleus. The calycine terminals and small "boutons" containing spherical synaptic vesicles and making contact both with the body of neurons and with dendrites of different caliber, degenerated in the ventral cochlear nucleus; presynaptic terminals containing flattened vesicles remained unchanged. A small number of presynaptic terminals in the dorsal nucleus degenerated; the overwhelming majority of terminals containing both spherical and flattened vesicles and forming axo-somatic and axo-dendritic synapses remained unchanged.

The neurohistological methods of silver impregnation of degenerating nerve fibers and their endings used to study connections in the CNS do not reveal the precise localization of degenerating presynaptic terminals.

In the investigation described below an electron-microscopic study was therefore made of the distribution of synapses in the primary cochlear nuclei of the brain stem after destruction of the spiral ganglion in cats.

Previous investigations [1, 2] showed that presynaptic terminals of two types are found in the cochlear nuclei: some contained spherical synaptic vesicles 300-500 Å in diameter, others flattened synaptic vesicles, 400-600 Å in length and 150-250 Å in width. The overwhelming majority of calycine terminals, which are found only in the ventral cochlear nucleus (VCN) [4, 5, 11, 12, 13, 16], contain spherical synaptic vesicles, whereas some terminal "boutons" found in VCN and the dorsal cochlear nucleus (DCN) contain spherical and others contain flattened synaptic vesicles.

EXPERIMENTAL METHOD

The operation of destruction of the inner ear, including the spiral ganglion, was performed on 15 adult cats. In all experiments the spiral ganglion was destroyed only. The animals survived for 2, 3, 5, 7, and 11 days after the operation. The brain was fixed by perfusion with 2.5% glutaraldehyde solution and small pieces of the cochlear nuclei were subsequently fixed in 1% osmium tetroxide solution in phosphate buffer [9]. After dehydration, the material was embedded in Épon-812. Sections, 300-800 Å in thickness,

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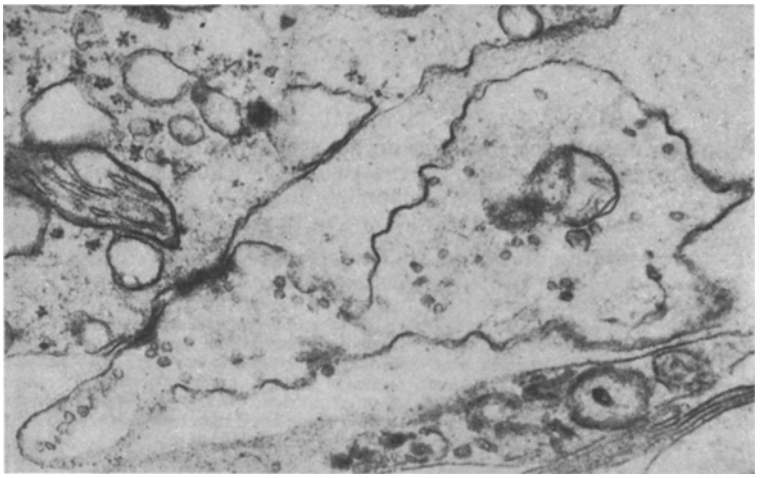

Fig. 1. Antero-ventral cochlear nucleus. Degenerating calycine terminal (CT). Third day of degeneration, 59,000×.

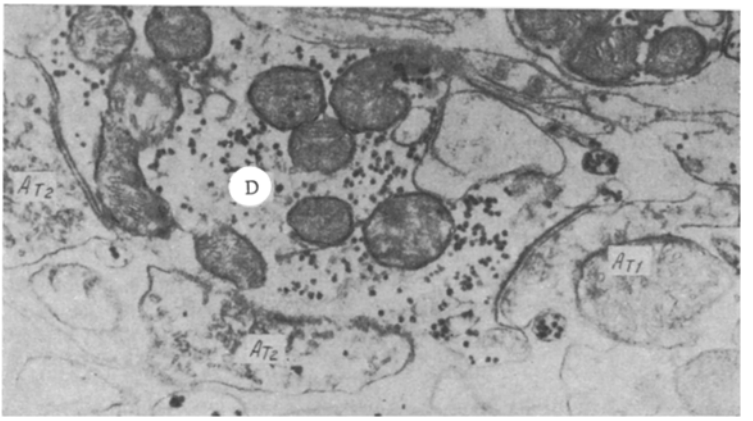

Fig. 2. Postero-ventral cochlear nucleus. Degenerating ending (A_{T1}) and unchanged small "boutons" (A_{T2}) containing flattened synaptic vesicles form contacts with the trunk of a dendrite (D); 42,000×.

were mounted on grids without a supporting film, stained by Reynolds' method [15], and examined in the electron microscope with an accelerating voltage of 80 kV.

EXPERIMENTAL RESULTS

Investigation of the character of distribution of the degenerating axon terminals in the cochlear nuclei at different times after destruction of the spiral ganglion showed that the degeneration was most intensive in VCN (especially in the antero-ventral nucleus). Degenerating axon terminals in VCN were in contact both with the perikaryon and with dendrites of different diameter. After destruction of the spiral ganglion all calycine endings containing both spherical and flattened synaptic vesicles degenerated (Fig. 1) while only solitary calycine terminals, containing greatly flattened vesicles, remained unchanged. So far as small axon terminals were concerned, in this case all "boutons" with spherical vesicles had degenerated, whereas, "boutons" containing flattened vesicles remained intact (Fig. 2); however, it cannot be asserted categorically that all terminals with flattened vesicles without exception remained unchanged. Rough counting (not paying attention to calycine terminals) showed, admittedly, that whereas the ratio between the number of presynaptic terminals with spherical synaptic vesicles and the number of terminals with flattened vesicles in the VCN of animals with an intact spiral ganglion was 3:4, in experimental material the ratio

between the number of degenerating terminals and the number of intact terminals with flattened vesicles was 3.2 :4, i.e., 4 :5.

The order of distribution of both degenerating and unchanged presynaptic terminals in VCN may be of any kind. In some cases several degenerating terminals were located on the cell bodies, while in others degenerating and intact endings alternated with each other; cases were observed in which several "normal" terminals were side by side.

No relationship could be found between the number of degenerating synapses and their localization; degenerated endings were found just as often on the dendrites as on the perikaryon.

Damage to the spiral ganglion caused degeneration of most terminals participating in the formation of glomerular complexes both with the central dendrite and with the central axon as described by the writers previously [2].

A different picture was observed in DCN; in this case, by contrast with the changes in VCN, only a few presynaptic terminals degenerated after destruction of the spiral ganglion and most of them, containing both spherical and flattened synaptic vesicles, remained unchaged. The unchanged presynaptic terminals formed synaptic contacts with the body of the nerve cells and with dendrites of different calibers. The character of distribution of the terminals containing synaptic vesicles of different shapes was variable.

In both VCN and DCN degenerating presynaptic terminals made contact both with the body and with the dendrites.

It can be concluded from the results of this investigation that most afferent fibers of the cochlear nerve terminate in VCN and a smaller number in DCN. Endings of fibers of peripheral neurons of the auditory system consist of small "boutons" and calycine terminals containing spherical synaptic vesicles. These endings form both axo-somatic and axo-dendritic synapses. Endings of axons reaching the cochlear nuclei from higher levels of the brain are represented in VCN by terminals containing flattened synaptic vesicles and in DCN by terminals with both spherical and with flattened vesicles. These endings take part in the formation of axo-somatic and axo-dendritic synapses.

Some investigators [3, 8, 10, 17] attach importance to the shape of the synaptic vesicles from the point of view of functional differentiation of synapses. It follows from the conclusions given above and from the view that afferent fibers of the cochlear nerve always transmit impulses of excitation, whereas endings of axons coming from the higher auditory centers have an inhibitory effect on the VCN neurons [6, 7, 14], that presynaptic terminals containing spherical synaptic vesicles form excitatory synapses in VCN, whereas terminals containing flattened synaptic vesicles form inhibitory synapses.

LITERATURE CITED

1. A. L. Mikeladze and I. L. Lazriev, Soobshch. Akad. Nauk Gruz. SSR, 58, No. 2, 457 (1970).
2. A. L. Mikeladze and I. L. Lazriev, Arkh. Anat., No. 11, 18 (1970).
3. D. Bodian, Science, 151, 1093 (1966).
4. S. Ramon y Cajal, Histologie du Systeme Nerveux de l'Homme et des Vertebres, Vol. 1, Madrid (1952).
5. M. L. Feldman and J. M. Harrison, J. Comp. Neurol., 137, 267 (1969).
6. J. Fex, Acta Physiol. Scand., 64, 43 (1965).
7. R. Galambos, J. Neurophysiol., 19, 424 (1956).
8. E. G. Gray, Prog. Brain Res., 31, 141 (1969).
9. U. Karlsson and R. L. Schultz, J. Utrastruct. Res., 12, 160 (1965).
10. F. I. Khattab, Experientia, 24, 690 (1968).
11. N. J. Lenn and T. S. Reese, Am. J. Anat., 118, 375 (1966).
12. R. Lorente de No, Laryngoscope, 43, 1 (1933).
13. K. K. Osen, Arch. Ital. Biol., 108, 21 (1970).
14. R. K. Pfalz, J. Acoust. Soc. Amer., 34, 1472 (1962).
15. E. S. Reynolds, J. Cell Biol., 17, 208 (1963).
16. W. A. Stotler, Anat. Rec., 103, 561 (1949).
17. K. Uchizono, Exp. Brain Res., 4, 97 (1967).