CHANGES IN SYNAPTIC FORMATIONS IN INTOXICATION BY OCCUPATIONAL POISONS

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The present work is devoted to the study of the changes in the axomatic interneuronal connections of the efferent neurons of the cortex and motor neurons of the ventral horns of the spinal cord in the presence of poisoning by some occupational poisons.

Much work of Russian authors has been devoted to the study of the synaptic architectonics of the axomatic interneuronal connections (V. M. Bekhterev, S. A. Sukhanov, V. A. Bets [1], N. M. Yakubovich, B. I. Lavrentyev [5, 6, 7] A. D. Zurabashvili [4]). The investigation of the axomatic synapses on sectioned material present great difficulties, since the synapses undergo postmortem changes very quickly. It is known from the work of B. I. Lavrentyev [5, 6, 7] that the synapses of the axo-synaptic connections appear at late stages of phylo- and ontogenesis and gradually grow more complicated. It has been proved that the axomatic synapses or end-feet (boutons terminaux) on the body of the neuron are the ends of one or several axons and change when the latter are cut, and degeneration of the end of the corresponding axon extends to the synapses but does not extend to the neuron which is innervated by these endings.

Usually the cell body and its dendrites are covered extensively with synaptic end-feet or buttons. In addition transient synapses exist in the form of thin, so-called transit fibrils which pass over the body of the neuron and do not adjoin it.

The histological pathology of the axomatic synapses was studied by B. I. Lavrentyev, L. I. Smirnov, A. D. Zurabashvili [4, 5, 6, 7, 9, 10], who came to the conclusion that the synapses are the most easily wounded and most vulnerable part of the neuron. L. I. Smirnov described a number of pathological changes in the synapses: wrinkling of the end-feet swelling of the end fibers and the presence of granules of argentophilic disintegration in the pericellular interstices. Ioffe and Dzhibson authors cited according to A. L. Shabadash [12]) describe 5 stages in the degeneration of the synapses: elongation, swelling, elongation of the end-feet and their dark discoloration, degeneration of the end-feet into granules and their disappearance.

At the present time a number of clinical data exist [2] which indicate that functional changes in the nervous system in the form of an asthenic state occurs at the initial stages of lead and arsenic poisoning. Indications [3] also exist regarding the slight decreases in sensitivity during the initial stages of the above poisonings, which disappear with the administration of prozerine, which acts on the synaptic connections.

Based on the above, we undertook the study of the changes on the synaptic axomatic connections between the efferent neurons of the brain cortex and the motor neurons of the ventral horns of the spinal cord during occupational poisoning.

EXPERIMENTAL METHODS

Experiments were carried out on 34 animals (18 rats, 4 dogs and 12 rabbits), which were subjected to short and long-term intoxication with aniline (subcutaneously in doses from 0.003 to 0.9 g per 1 kg of weight) (14 animals), lead acetate (subcutaneously in doses from 0.03 to 0.04 g per 1 kg of weight and by mouth in doses from 0.007 to 0.008 g per 1 kg of weight) and sodium arsenite (subcutaneously in doses from 0.0004 to 0.012 g per 1 kg of weight). The duration of the short experiment was from 5 to 10 days, of the long 1–3 months. The brains of healthy animals, which lived under the same conditions as the experimental ones, served as the controls. The brain was prepared by the methods of Gliss, Nissl and Ramon y Cajal.

EXPERIMENTAL RESULTS

In studying the spinal cord of the control animals, it was observed that the motor cells of the ventral horns of the spinal cord were surrounded by a pericellular plexus whose terminal fibers ended on the body of the neurons as multitudinous loops, rings, or buttons (Fig. 1). There were considerably fewer synaptic end-feet around the efferent neurons of the cortex than around the motor neurons of the spinal cord (as is known, other synaptic connections predominate in the cortex – the axodendritic ones).

The changes which arose in the synaptic formations of the axomatic interneuronal connections of the efferent neurons of the cortex and motor neurons of the spinal cord were of one type and similar to each other in all the above intoxications (aniline, etc.). This gave us a basis for summarazing the materials obtained.

The changes which arose in the synaptic axomatic connections during long-and short-term lead, aniline and arsenic poisoning did not represent anything specific for these poisonings and were similar to the changes described for various other injurious influences on the organism.

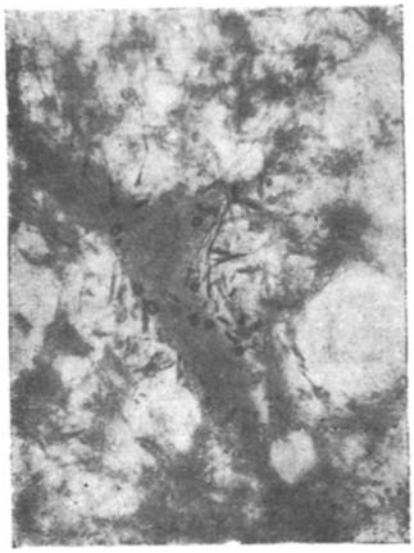

Fig. 1. Multitudinous end-feet on the body of a motor neuron from the spinal cord of a rabbit. (Prepared according to Ramon y Cajal).

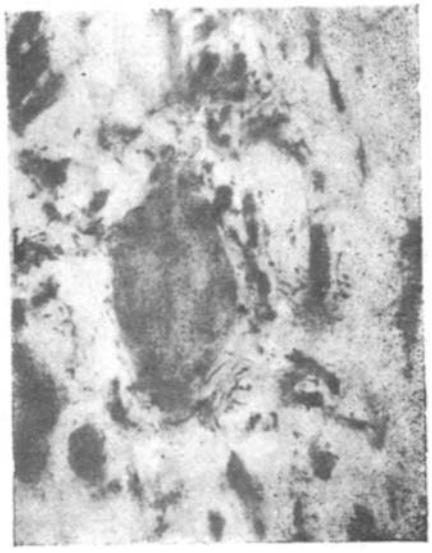

Fig. 2. Long-term arsenic poisoning of a rabbit. The marked thickening and overimpregnation of the end-feet, their scaling from the body of the motor neuron from the spinal cord and granular degeneration are evident in the illustration. (Prepared according to Ramon y Cajal).

In long-term intoxication with very small, gradually increasing doses of lead, arsenic and aniline, swelling, club-shaped bulging and darkening of the end-feet and their separation from the cell body were noted [Fig. 2]. Some of the end-feet moved away from the cell body, became deformed and thickened, while others continued to keep their normal structure. This, apparently, is explained by the fact that the synaptic formations of the body of one neuron can belong to the

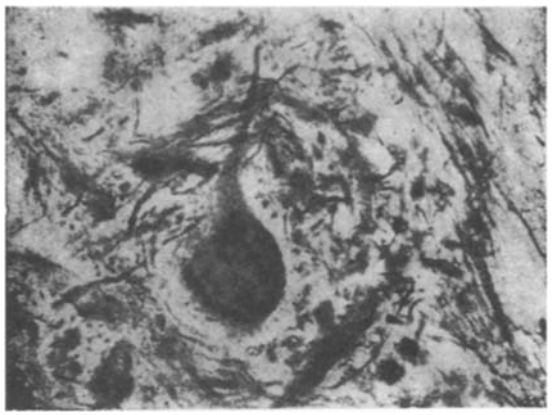

Fig. 3. Long-term lead poisoning of a rabbit. Granular degeneration and lysis of the end-feet, coarsening of the preterminal fibrils around the motor neuron from the spinal cord are visible in the illustration. (Prepared according to Ramon y Cajal).

axon endings of several different neurons. It was always possible to find neurons with normal synaptic formations next to a neuron with changed synaptic formations. No changes were found in the neurons with Nissl's stain.

Changes similar to the above were not found among animals of the same group which were killed 1–2 months after the poisoning ended. This gave us the basis for the conclusion that the above changes are reversible and serve as a manifestation of the initial, reversible reaction of the nervous system. These changes were found before the changes which originated in the body of the neurons. Apparently, the synapses of the neurons, which are very fine formations, are very sensitive and are the first to react to various poisonings.

In more prolonged and more evident chronic poisoning by the same toxic substances, we observed acute degenerative changes in the synaptic formations: degeneration of the synaptic end-feet into grains or their complete lysis Fig. 3 . When Nissl's stain was used, considerable dystrophic processes were observed in the body of the neurons in these cases.

In short-term poisoning by the above substances, marked changes were observed in the synaptic axomatic formations: the end-feet scaled off and partially dissolved; a great number of neurons with changed synapses was observed, although the destruction of the synapses around the individual neurons was not as great as in long-term poisoning. Markedly evident vascular disturbances with perivascular and pericellular edema and acute swelling of the protoplasm of many neurons was found in these cases with Nissl's stain. Apparently, as a result of the great sensitivity of the synaptic formations, the synapses between the individual neurons reacted very quickly, before the neurons.

SUMMARY

Changes in synaptical axomatic junctions in the efferent neurons of the cerebral cortex and in the motor neurons of the ventral horns of the spinal cord were studied on 34 animals exposed to acute and chronic intoxication with aniline, lead and arsenic. The study showed synaptic formations to possess high sensibility and to respond to the small doses of toxic agents earlier than do nervous cells. Small doses of the mentioned toxic agents bring about such initial reversible changes in synaptic formations as superimpregnation and club-like thickening of the synaptic rings along with their shifting from the body of the nervous cell. In the case of expressed chronic or heavy acute intoxication, destructive changes take place, e.g., degeneration of synaptic end-feet to clubs, or their complete dissolution.

* In Russian.

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