NOTE ON THE PATHOLOGY OF TOXIC
AMBLYOPIAS.By J. HERBERT PARSONS, B.S., B.Sc., F.R.C.S.,
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It has been known since 1880¹ that the papillo-macular bundles of the optic nerves are degenerated in cases of tobacco amblyopia. These fibres are the axis-cylinder processes of the ganglion cells of the macular region of the retina, and hence changes in these cells are probably the cause of the degeneration. Sachs and de Schweinitz failed to find any microscopic evidences of such changes; but their observations were made before the introduction of Nissl's method of staining the intracellular granules. No case of tobacco amblyopia has yet been submitted to this test; but, on the one hand, the effect of nicotine upon the central nervous system has been investigated, and, on the other hand, changes have been described in the ganglion cells of the retina in other forms of toxic amblyopia.

To the second group belong those cases of experimental amblyopia produced by the administration of quinine, flin mas, carbon disulphide, etc., to animals. Distinct changes are found in the ganglion cells and nuclear layers of the retina, which Ward Holden² attributes largely to diminution in the blood supply due to vaso-constriction of the vessels, produced in the cases recorded by him by the administration of large doses of quinine to dogs. This and other forms of toxic amblyopia have been very carefully reinvestigated recently by Birch-Hirschfeld,³ who confirms the general results, though with some modifications.

The effect of nicotine upon nerve cells has received much attention from the experimental point of view, since Langley⁴ discovered that it paralysed them. The effect of the administration of nicotine to animals has also been investigated histologically in the cells of the central nervous system, with positive results, by Vas and Pándi.⁵

The paralyzing effect of the drug is obtained whether it is injected into the circulation or whether applied to the cells locally. It seemed to me to be worth while to investigate any changes produced in the cells by local application.

For this purpose the superior cervical ganglia of cats, which had been anaesthetised with ether, were removed immediately after death, and immersed for various periods from 1 to 3 hours in aqueous or normal saline solutions of nicotine of 1 per cent., 2.5 per cent., or 5 per cent. strength. Thin sections of the ganglia were compared with others of normal ganglia prepared and stained by identically the same process—namely, fixation in saturated corrosive sublimate solution, imbedding in paraffin, and staining with toluidine blue.

After being immersed for one hour in 1 per cent. solution of nicotine, which would be fully sufficient to paralyse the cells, they cannot be distinguished from normal cells. After more prolonged action or immersion in stronger solutions various changes are brought about which need not be referred to here.⁶

This supports the conclusion arrived at by Langley⁷ that the drug acts, not essentially upon the nerve cell, but upon the branchings (synapse) of the pre-ganglionic fibres around the cell. That there may be changes in the cell itself, which the delicate Nissl method is not sufficiently delicate to show, is, of course, possible.

The results are of some interest from the point of view of toxic amblyopia, for it would seem that if there are changes in the ganglion cells of the macula, they are a secondary result of changes in the fibres forming networks around them, that is, in the terminal fibrils of the inner granules, which form a network in the inner molecular layer. These, in turn, may be the result of changes in the outer molecular layer, in the fibrils of the cone fibres.

Warrington⁸ has shown that cutting off the afferent impulses to a nerve cell is an even more potent cause of degeneration of the Nissl granules than cutting its own axis-cylinder process.

The action of nicotine may reasonably be supposed to be twofold: (1) Vascular, causing vaso-constriction of the arterioles, which would explain the selection of the sparsely-supplied macular region; (2) paralytic, upon the synapses either of the cone fibres, or of the inner granules, or of both.

BIBLIOGRAPHY.

- ¹ Samelsohn, *Graef's Archiv*, 1882, Bd. xxviii, s. 1. ² Ward A. Holden, *Trans. Amer. Ophth. Soc.*, 1898. ³ Birch-Hirschfeld, *Graef's Archiv*, 1900, Bd. 1. ⁴ Langley and Dickinson, *Proc. Roy. Soc.*, 1880, xli, p. 425. ⁵ Pándi, *Ungar Archiv. f. Med.*, ii, 1893, s. 257. ⁶ See *Proc. Phys. Soc., Journ. of Phys.*, xxvi, p. 38. ⁷ Langley, in Schäfer's *Textbook of Physiology*, ii, p. 621. ⁸ Warrington, *Journ. of Phys.*, xxiii, p. 114.

PARALYSIS OF THE CERVICAL SYMPATHETIC.

With Special Plate

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LESIONS of the cervical sympathetic are probably not so infrequent as the comparatively scanty references to them in current literature might lead us to expect. Many cases escape recognition because their signs and symptoms are not so obvious as in the paralysis of an ordinary cranial or spinal nerve.

The following case is unique, not so much because it exemplifies to a marked degree nearly all the classical symptoms of paralysis of the cervical sympathetic, but because it also demonstrates with great clearness the precise limits of one symptom in particular—anidrosis—with reference to which a considerable indefiniteness of statement has hitherto prevailed.

Fig. 1.—Area of anidrosis=right side of head, neck, and upper part of thorax, together with right upper extremity, bounded by thick black line. Area of cutaneous analgesia=shaded area of right upper extremity, back and front.

For the opportunity of studying the case I am indebted to the courtesy of Dr. W. H. Farmer, who, like myself, was a civilian medical officer *en route* home from South Africa on board the hospital ship *Simla*. Since our departure from the Cape the weather had been of tropical heat, and it had been observed that a certain patient sweated only on one side of his face.

History.

The patient, a man 30 years of age, is a soldier who was wounded in the left side of his neck two months and a-half previously by a Mauser bullet. At the time he was hit he was lying on his face, firing at the enemy. The bullet entered the left side of his neck an inch and a-half below the mastoid process, and came out through the seventh right interspace in the posterior axillary line, lodging in his bandolier. The bullet track thus passed in front of the vertebral column. At the time of the injury he felt an "electric shock" all over his body, but especially in his right upper extremity. The latter became at once totally paralysed. He also commenced to spit blood, and the hæmoptysis continued fairly copiously for three days, together with some difficulty in swallowing, the later being apparently referable to an injury of the œsophagus. On the fourth day his temperature became normal, and in three weeks the hæmoptysis entirely ceased.

The right upper limb, which was at first totally paralysed,