AMBLYOPIA DUE TO METHYL ALCOHOL.

By J. W. STIRLING, M.D., Montreal.

The comparative rarity of this condition and its gravity render the publication of case reports of some value.

The minimum quantity of wood spirit necessary to produce the ocular symptoms is as yet uncertain, but the onset is very rapid and is associated with much general disturbance. There is marked loss of vision, going on quickly to total blindness, which may, after a varying length of time, improve to a greater or less extent, but which always ends in a certain degree of atrophy of the optic nerve with corresponding limitation of the visual field. Optic neuritis has been noted at the onset.

The pathological change must affect the whole optic nerve and not be limited to any particular nerve bundles. The visual field suffers a peripheric contraction of irregular shape and degree, but is never hemianopic, and it is rare even in the mild cases that a central scotoma has been observed. My case report is as follows.

H.E., rather thin nervous man of forty-one years of age, was brought to me by Dr. Winter of Algonquin, Ontario, on account of poor vision. The patient stated that he had had poor sight for over a year. The onset of his trouble was very sudden, and dated from a severe drinking bout about thirteen months ago. At that time he drank six ounces of wood spirit and two ounces of brandy in one hour. He felt stupid but managed to eat his supper, went to bed and slept well. The next morning felt unwell but ate his breakfast and later his dinner. About 3 p.m. (22 hours after drinking the alcohol) he vomited severely, and then went to bed two hours later, sleeping twenty-four
hours continuously. Upon awaking he found that his vision had entirely gone. The patient remained totally blind for thirty-six hours, but at the expiration of that time he began to notice shadows of objects with the left eye; this vision slowly improved for six months, but has remained at a standstill ever since. He began to appreciate shadows with the right eye about a month after the left eye first noticed them. The vision of the right eye likewise improved slowly for a few months, but this improvement has also ceased for the past six months.

His present condition is as follows: Vision = R.E. $\frac{1}{20}$; L.E. $\frac{1}{10}$; no lens further improves vision. The pupils barely oscillate to light, and are 3 to 4 millimetres in diameter. There is total colour blindness.

The ophthalmoscope shows both optic discs to be chalk white, the edges sharply defined, and the retinal vessels only somewhat diminished in diameter. The visual fields are greatly contracted, especially the right one, only a small portion upwards and outwards being retained.

The right field extends upwards $35^\circ$, up and out $40^\circ$, but ceases $10^\circ$ short of the horizontal meridian; in no direction does it reach below the fixation point; finally, it extends $15^\circ$ to the upper inner side of the vertical meridian. The left field is much larger than the right, extending to between $40$ and $50^\circ$ up and out, down and out, and horizontally inwards with deep re-entering angles, as is the rule in cases of optic atrophy.

It is now over a year since I last saw him, but from the latest accounts his condition remains unchanged.

Ward Holden considers the methyl alcohol amblyopia as similar in nature to that caused by quinine poisoning or that following severe haemorrhages. In his experiments with methyl alcohol on animals (in 1899) he found marked
changes in the ganglion cells of the retina and in the optic nerve trunk.

Birch-Hirschfeld has noted similar changes (v. Graefe's Archiv. f. Ophth., lxi., Heft 2).

Rymowitsch, in similar experiments on rabbits, found fatty degeneration, etc., of the ganglion cells of the retina, with varicose hypertrophy of the nerve fibres, and oedema of the granular layers, but without changes in the optic nerve itself.

Birch-Hirschfeld considers the changes in the ganglion cells to be the primary lesion, and the optic nerve degeneration to be secondary thereto.