DAY-BLINDNESS *

BY

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DAY-BLINDNESS is a term generally applied to the syndrome of impaired vision in good illumination, associated with almost normal vision in the dark. The terms hemeranopia and heliophobia are synonymous with day-blindness. As the disease shows characteristics opposite to those of night-blindness, the term nyctalopia, as opposed to hemeralopia, is suitable. On the other hand Bourdier (1939) used nyctalopia to describe the state of retinal hypersensitivity characterized by normal day-light vision and supernormal night vision.

Day-blindness can be divided into three groups:

(1) Associated with central opacities of the cornea and the lens (the dioptric nyctalopia of Axenfeld, 1914). Improvement of vision results from dilatation of the pupil at dusk.

(2) Associated with either toxic amblyopia or macular degeneration.

(3) Congenital day-blindness.

Our case is an example of the third group.

Case History

Mr. N. E., 31 years old, came to the clinic with the complaint of bad day-light vision. From his earliest years he had kept his eyes half-shut in daylight. When he went to school he noticed a visual defect which was not improved with glasses. He was treated with miotics for a period, and 15 years ago he noticed for the first time the difference between his day and night vision. Though he was examined by many physicians and was treated with vitamin A, injections of mercury cyanide, and iontophoresis, the condition failed to improve. His father and mother were first cousins but neither they nor three brothers and the remaining relatives showed evidence of ocular defects. He was slightly pale and swarthy. Apart from a malarial infection in childhood he did not remember having suffered from any disease.

The patient wore dark glasses continuously, had always kept his eyes half-shut, and was obliged to close them further in order clearly to recognize objects. Externally both eyes were normal and there was no nystagmus. Skiascopy disclosed in the right eye a mixed astigmatism of $-0{,}50$ D in the vertical and $+1{,}50$ in the horizontal meridian, and in the left eye a simple hypermetropic astigmatism $+1{,}0$ D in the horizontal meridian. Biomicroscopic examinations of the cornea and lens revealed no abnormality. Both pupils showed miosis and sluggish movements; light and convergence reflexes were present. Ophthalmoscopic examination showed in both eyes slightly pale disks and dark red tigroid retinæ, which were considered to be within normal limits in view of the patient's dark complexion. A few round pigmented dots and one or two atrophic patches were scattered over the peripheral areas of the retina.

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In the right eye in the upper external quadrant near the ora serrata, an old patch of chorioiditis was found. No pathological change could be found in the macular area with the binocular ophthalmoscope.

Ocular tension was 22 mm. Hg (Schiötz) in both eyes. Provocative tests gave negative results. In normal day-light, V.A. was C.F. at one metre, while in reduced light, V.A.R. was 6/60, V.A.L. was 6/36. Experimental miosis with

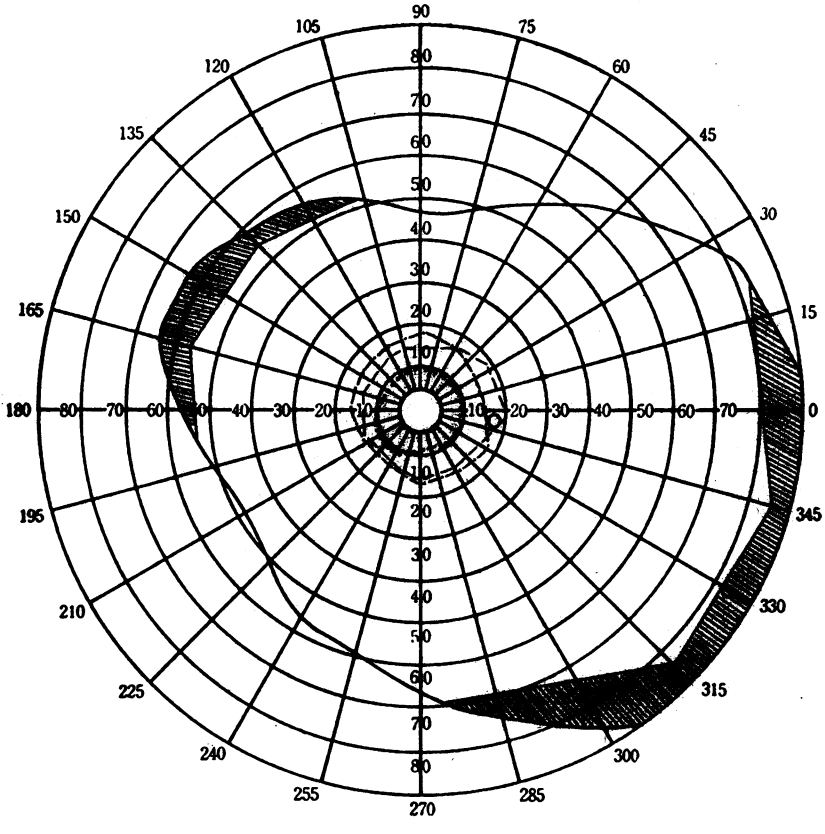

FIG. 1.—Visual field, right eye.

——— 3/330 White — · — 5/330 Blue

. . . . 5/330 Red — — — 5/330 Green

eserine lowered the visual acuity, whereas mydriatics disturbed near vision without improving distant vision.

The light threshold and the dark adaptation tests performed with the Birch-Hirschfeld apparatus revealed nothing pathological. The concentric narrowing of the visual field for white, which was seen in normal day-light examinations, disappeared on reducing the illumination. The colour field examination in normal day-light was impossible, for the coloured test objects of 5 mm. diameter were not seen by the patient. The same examination repeated in reduced light revealed a marked contraction for all colours, especially for red (Figs 1 and 2). As the patient could not distinguish colours properly, the limits of the colour fields were determined by the points at which the patient first perceived slightly coloured

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objects, rather than by those at which he recognized them in full colour and saturation.

Holmgren's yarn test gave the following results: The patient recognized the red skein only when he brought it near his eyes. He called yellow, green, and blue, yellow. He matched the red test skein with orange, brown, and yellow; the violet test skein with violet and grey. He also matched the light green test

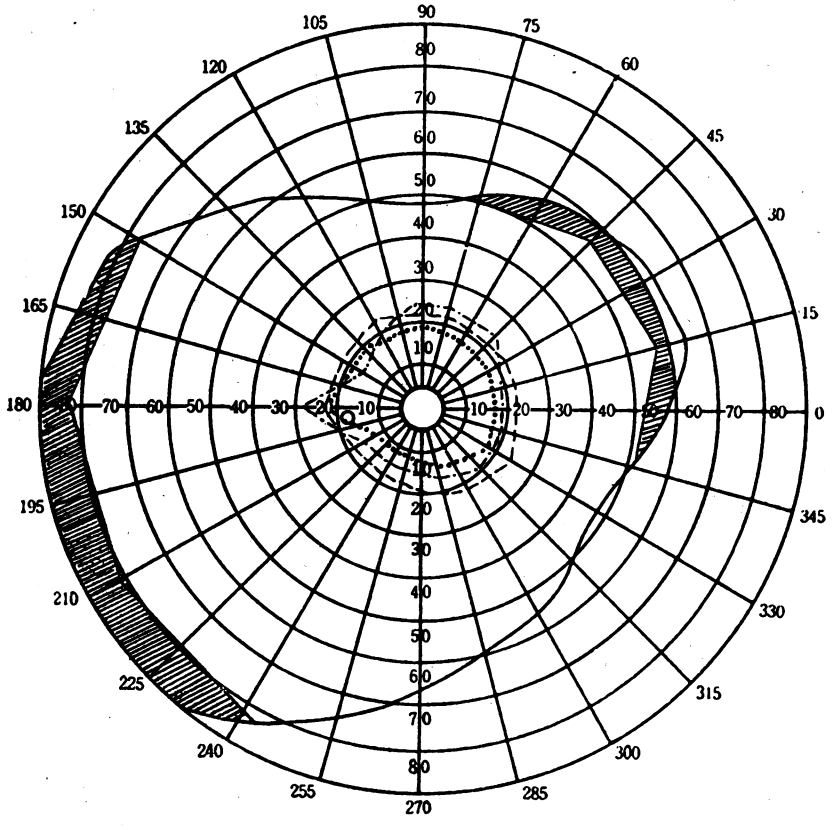

FIG. 2.—Visual field, left eye.

——— 3/330 White — · — 5/330 Blue
. . . . 5/330 Red — — — 5/330 Green

skein with light blue, and the yellow test skein with light blue. Some darker or lighter tones of the test skeins were omitted. Examinations with Stilling's pseudo-isochromatic diagrams (18th edition) revealed that the patient could read the first and second plates very well; from the third to the fifteenth some were recognized with difficulty, some were misinterpreted and others not recognized at all. In Plate XV_1 the number 92 was read as 45; and in Plate XV_2 the numbers 45 and 48 were read as 92 and 67 respectively. Hertel (1929) considered this symptom to be characteristic of yellow/blue blindness (tritanopia).

When he looked through the hand spectroscope in day-light, he saw a colourless spectrum with dark extremities. When the light was gradually reduced, the

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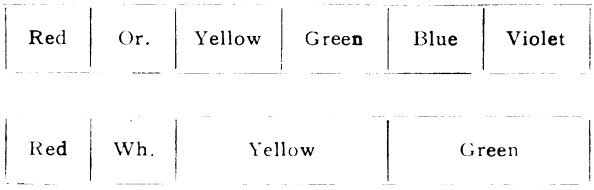
patient first distinguished colour at the red end of the spectrum. In the most suitable illumination for the patient the spectrum was composed of the following colours: red, a light colour similar to white, yellow, and green. Their places according to the normal spectrum were as follows:



Arterial blood pressure was 13/8,5 Vaquez; R.B.C. count: 3,200,000, Hb: 60 per cent., W.B.C. count: 3,400; blood picture was: stab: 1 per cent., polymorphnuclear: 57 per cent., eosinophils: 4 per cent, monocytes: 5 per cent., lymphocytes: 33 per cent., sedimentation rate: 3 mm. in the first hour, blood sugar, nitrogen and cholesterin within normal limits; Wassermann and Mantoux tests negative. Radiological examination of the lungs, nasal sinuses, and teeth for a focus of infection, showed no pathological change; the sella turcica was normal.

Discussion

According to Nettleship (1909) day-blindness is associated with colour blindness, amblyopia, and often with nystagmus. There is no evidence of optic atrophy to explain the impairment of vision. The fundus is usually normal, but sometimes the disks and the maculæ show changes, which are too slight to be considered the cause of such a grave functional impairment. The disease is hereditary and recessive; it may be seen in several brothers of the same family, but the parents are always healthy although an uncle may be affected.

Incidence is greater among males than among females, and in most cases consanguinity is found. The disease is congenital and remains stationary. As mental disorders are frequently associated with the syndrome, this disease presents a clinical picture similar to retinitis pigmentosa; and in some cases typical retinal pigmentation has been found. It is, however, a rare disease; including his own and some solitary cases showing no familial incidence, Nettleship (1909) was only able to collect 84 cases in the literature.

Day-blindness is generally supposed to be due to impairment of cone function. Venneman (1906) attributed it to defective nourishment. Goldmann (1936), observing the symptoms of day-blindness in a case which showed temporary occlusion of the central retinal artery, suggested the presence of a lesion in the inner layers of the retina. Thus a disturbance in function of the amacrine and horizontal cells, impairing the contrast function of

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the retina, might be the cause. Since histological examinations have not been made in cases of day-blindness, no definite anatomical changes have been described, and some writers suggest that the condition is due to a subtle physiological disturbance.

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