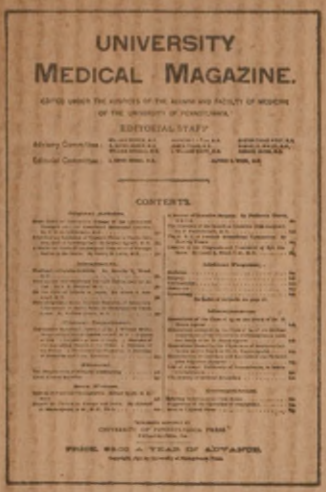
REPRINTED FROM



UNIVERSITY
MEDICAL MAGAZINE.

EDITORIAL STAFF

CONTENTS.

DECEMBER, 1896

THE PUPIL REFLEX IN ABSOLUTE BLINDNESS.

BY GEORGE C. HARLAN, M.D.

THE PUPIL REFLEX IN ABSOLUTE BLINDNESS.[1]

By George C. Harlan, M.D.

The pupil is dilated and fixed in so large a proportion of cases of absolute blindness, in which the ball is sound and the media are clear, that the impression is quite general among those whose attention has not been particularly called to the subject that reflex pupillary action is not compatible with loss of light-perception. This is less to be wondered at when we consider that few of the ophthalmological text-books contain anything to correct this misapprehension, and that many of the neurological works do not bring the point out clearly. These considerations may justify the report of the following cases :

W. G., a bright and well-developed boy, 16 years of age, recently admitted as a pupil to the Pennsylvania Institution for the Instruction of the Blind, had good health until about a year ago. He then had a severe illness, which his attending physicians attributed to intracranial tumor, and during which his vision failed, and at the end of three months was entirely lost. This was all that could be learned of the history of his case. He was represented as in good health at the time of his admission to the institution, but it was soon found that he was unequal to any mental exertion, and had frequent headaches with occasional attacks of vomiting, and that it was necessary to return him to his home in a distant town.

There was entire absence of light-perception in both eyes; repeated testing and close watching left no doubt on this point. The pupils were dilated—about seven millimetres—and the eyes had the vacant stare and objectless movements of blindness, but the external appearances were otherwise normal. The pupils responded sluggishly and partially, though very evidently, to diffused daylight from a northern window, but promptly and completely to direct sunlight. The ophthalmoscope showed typical post-neuritic atrophy of both disks, which, in the absence of other probable cause, might have been considered sufficient to account for the blindness.

Of course, a moment's reflection shows that there is nothing in the behavior of the pupil in this case that does not admit of ready explanation, and that it is rather remarkable that such cases are not more frequently observed. If the well known path of reflex pupillary action—from the retina through the optic nerve, optic tract, corpora quadrigemina, and Meynert's fibres, to the centre for pupillary con-

[1] Read before the American Ophthalmological Society.

traction in the aqueduct of Sylvius, and thence returning to the iris through the fibres of the third nerve—be unimpaired, there is no reason why a lesion situated in the cortical visual centre, or in the fibres connecting this centre with the corpora quadrigemina, should necessarily interfere with the movements of the pupil. Ferrier states that a flash of light before the eyes will cause the pupils to contract in animals deprived of their cerebral hemispheres. Perhaps the reason why pupil reaction in absolute blindness is so rare is that a bilateral lesion would be required to cause complete cortical blindness, even in one eye (bilateral hemianopsia), and that so extensive a disease would be likely to involve other portions of the visual apparatus. As in the case just reported, the optic atrophy, evidently consecutive to neuritis, observed with the ophthalmoscope, would appear to be sufficient of itself to obstruct light stimulus, but has done so only partially; the pupils acting sluggishly and incompletely to diffused daylight, though promptly and fully to direct sunlight. The Wernicke symptom—hemianopic pupil reaction—has been much discussed of late as a means of locating unilateral lesions; and in the Argyll-Robertson pupil we have loss of reflex without loss of sight.

I expected to find a number of cases for the observation of the pupil in absolute blindness in the Pennsylvania Institution for the Blind, and with the assistance of Dr. George Cross made a search there for such cases; but among the 175 inmates we could find only three with sound balls and clear media who were entirely without the perception of light. Those who were conscious of direct sunlight were excluded.

A. W., a girl, 18 years of age, in excellent health, blind for two and a half years after cerebro-spinal meningitis; complete optic atrophy. Disks bluish-white and somewhat stippled, with margins not sharply defined and slight choroidal changes about them; retinal arteries slightly contracted; no nystagmus; pupils regular and seven millimetres in diameter; no reaction to diffused light, but decided though slow contraction when exposed to direct sunlight, more noticeable after prolonged shading, which, however, does not cause increased dilatation.

J. V., a healthy man, 20 years old, lost sight at 7 years of age, also from cerebro-spinal meningitis. Complete white optic atrophy. Disks stippled and margins sharply defined; no decided choroidal changes; nystagmus; pupils 5.5 millimetres; no reaction by usual tests, but after the eyes had been bandaged for half an hour the pupils were found to have dilated to eight millimetres, from which they gradually and imperceptibly returned to their former size.

They contracted decidedly, though slowly, on exposure to direct sunlight.

J. McK., aged 15 years, a well developed muscular boy, large for his age, and of fair intelligence, has never seen; is not conscious of the shadow of the hand, or of a black screen passed before the eyes in direct sunlight. No history of blindness in any of his relatives, except a brother, who was born blind. He has two sisters with perfect vision. The brother was an inmate of the institution some years ago, and the cause of his blindness is recorded as "congenital, probably optic atrophy." There is constant nystagmus; a rather slow movement of the balls, chiefly horizontal, but occasionally slightly oblique. Ophthalmoscopic examination of the right eye shows a hypermetropia of seven or eight D. The disk is scarcely distinguishable, except by the emergence of the retinal vessels, which are somewhat narrow. It is a little raised above the level of the fundus, and its margin is obscured by coarse striations. In the left eye the disk margins are less obscure. There is no suggestion in either of the usual picture of optic atrophy. The constant nystagmus makes a satisfactory examination impossible.

The pupil in each eye, in the diffused light of a room, is regular and 4.5 millimetres in diameter. There is perceptible variation with the movements of the ball, particularly with that down and in, but no response to the light from a northern window. When, however, the eyes are bandaged, for awhile the pupils dilate a little more than a millimetre, and when again exposed to light, though no motion is perceptible, they return to their former size in a few seconds. When exposed to direct sunlight, even without previous occlusion, the pupils contract down to two millimetres with a movement that is easily perceptible, but deliberate and very different from the prompt action of a normal iris.

None of these pupils responded to artificial light from the ophthalmoscopic mirror.

It is probable that in these cases there is complete cortical blindness with slow and very imperfect transmission of light excitation to the centre for pupil contraction. In the congenital case we may suppose absence of cortical visual centres and imperfect development of the optic nerve, or, perhaps, intra-uterine cerebral disease. It is remarkable that two members of a family should be thus affected, without heredity or other recognizable cause. Dr. Gould[1] has reported a case of complete white optic atrophy, consecutive to a neuritis occurring in connection with obscure spinal and cerebral

[1] Eighth International Ophthalmological Congress.

disease, in which the dilated pupils, although they failed to respond to the usual tests, contracted gradually and imperceptibly to the normal size when the patient was seated before a window. They would probably dilate if the eyes were bandaged and contract visibly if exposed to direct sunlight.

I commenced to suspect that direct sunlight might cause contraction in all cases, and that perhaps the heat might have some effect on the iris; but Dr. Risley has removed all doubt on that point by showing me a patient whose pupils are absolutely indifferent to sunlight. There is wide mydriasis and absolute optic atrophy of unknown cause. Her optic nerves have completely lost their function, or there is lesion in some other part of the reflex path.

In all these cases the pupil contracted with a forced effort to close the lids. Gifford,[1] who has recently called attention to this reaction, thinks that it is a result of an overflow of stimulus from the orbicularis centre to the centre for pupil contraction, and Jackson suggests that it may be "due to an inhibition of the action of the dilator of the pupil accompanying an inhibition of the palpebral muscle of Müller which acts in opposition to the orbicularis." A simpler explanation, and I believe the true one, is that the sphincter pupillæ acts in association with the superior rectus, which always contracts when a forcible effort is made to close the lids. This association is well known to obtain during sleep.

Some interest attaches to the condition of the pupil in hysterical blindness as indicating the probable seat of the morbid process. Gowers thinks that this morbid process is in the cortex, and says that the pupillary action should decide. Leber also maintains that there is a central disturbance, and retains the expression "retinal anesthesia," not as implying that the retina is necessarily involved, but merely as a convenient name for a condition that is not understood.

Priestley Smith,[2] on the other hand, thinks that "hysterical amblyopia, neurasthenic amblyopia, and sympathetic amblyopia are conditions of peripheral anesthesia, due to reflex contraction of the vessels which nourish the retina," and maintains his view chiefly by a study of the visual fields. He does not mention the pupil. I have usually found the pupil normal in cases of hysterical blindness, of which I have chanced to meet with a considerable number, and believe that this is the rule; but in one case of complete monocular blindness in a typically hysterical subject that I reported to the

[1] Archives of Ophthalmology, July, 1895.

[2] Ophthalmic Review, Vol. III, No. 31.

society a few years ago,[1] a pupil dilated *ad maximum* and absolutely fixed was promptly restored to normal size and complete mobility by mental impression (the application of a wooden imitation of a magnet). My experience would seem to indicate, so far as the evidence of the pupil is concerned, that the morbid process in hysterical blindness may be either central or peripheral, or both, but that it is more frequently central. It must be remembered, however, that the mydriasis in the case just referred to may not have been due to any obstruction in the reflex path of pupillary contraction, but that it may have been, and in all probability was, the result of excitation of the pupil-dilating centre in the medulla, which is known to be influenced by the emotions. In other words, there is very little doubt that it was not a paralytic but an irritation mydriasis, such as is met with in, for instance, the psychical excitement of acute mania; a mydriasis which is more easily referred to muscular action than to elastic contraction, as simple elasticity can act only by relaxation of its opponent.

This case of hysterical monocular blindness has seemed to me to be of special interest, because it was not merely a more or less dubious subjective phenomenon, but presented the definite objective symptom of a dilated and immobile pupil. It gives little encouragement, however, to localization of the morbid process, if even that term is not too materialistic to be applied to such cases as this, which, perhaps, in the present state of our knowledge, can scarcely be rescued from the domain of metaphysics. Still, it is easier to conceive of a retinal cause, though the ophthalmoscope may show no sign of it, than to understand how a cause acting in the cortex, or anywhere behind the commissure, could account for complete blindness in one eye, the other remaining unaffected, without doing violence to what is known of the anatomy and physiology of the brain.

[1] Transactions of the American Ophthalmological Society, Vol. III, p. 649.