

4. The rate and shape of the slide do not appear to be influenced by the nature of the injury nor the condition of the underlying substantia propria.

5. The sliding of a pigmented limbus after trauma can be distinguished from pigment proliferation after chemical stimuli and from pigment migration without epithelial loss in vitamin A deficiency.

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## ATAXIC NYSTAGMUS: A PATHOGNOMONIC SIGN IN DISSEMINATED SCLEROSIS

BY

WILFRED HARRIS

LONDON

FOR many years I have observed this peculiar form of nystagmus, and have demonstrated it to numbers of students and to my clinical assistants as being diagnostic of disseminated sclerosis. In my experience it is not met with in any other condition. On June 28, 1943, I showed a case illustrating this condition at the clinical meeting of the Medical Society of London, held in the wards of St. Mary's Hospital. As this aroused a certain amount of interest, and no-one who saw the case appeared to have observed this form of nystagmus previously, I feel that a description of its peculiarity should be put on record.

When the eyes are turned laterally, the conjugate action appears weak, so that the inner eye, *e.g.*, the right eye when looking

towards the left, does not reach the inner canthus, but the outer eye shows coarse nystagmus, the eye reaching the outer canthus with a quick movement, and a slower return, as though the outward movement could not be maintained, owing to weakness. Usually this nystagmus of the outer eye continues as long as the patient looks in that direction, the inner eye meanwhile remaining stationary, not having quite reached the inner canthus. It is this discrepancy between the movements of the two eyes which suggests to me the term "Ataxic Nystagmus."

Usually this peculiar nystagmus will be present when looking either to the right or left, though it may be more marked in one lateral movement than in the other, or it may be seen only in one lateral direction.

In one case only I have seen the outer eye give only one flick as far as the outer canthus, returning to a position parallel to the inner eye and remaining stationary, neither eye reaching quite to the outer canthus. A further injunction to the patient to look to the right or left reproduced the single flick of nystagmus of the outer eye, illustrating the weakness of the outward conjugate movement.

I have notes of seven cases of disseminated sclerosis with this type of nystagmus that I have seen since January, 1942, in all of which there could be no doubt of the clinical diagnosis.

The three following cases are typical:—

Mrs. C., aged 44 years, seen June 4, 1942. Two years weakness in walking and standing, unsteady on her feet, and cannot walk alone. Precipitancy of bladder. Somewhat anaemic, but blood film normal. K. J. + +, Plantars extensor, vibration sense in legs lost. Well marked ataxic nystagmus in both lateral directions. W. R. negative in blood and C. S. F. 4 lymphocytes per c. mm. Lange 2222111000. Protein content normal. She was shown at the Medical Society of London meeting on June 28, 1943.

P. F., a man, aged 50 years, seen October 2, 1942. Unsteady gait for nine years, getting gradually worse, can now only drag himself about by holding on to furniture. Legs spastic, plantars extensor, pallor of discs. Slow ataxic nystagmus in both lateral directions. For the past twelve months spasmodic neuralgia in right upper and lower jaws, especially in tongue, brought on by eating and handling the face. Had several injections at other hospitals, but no anaesthesia produced and no improvement. Sensory root resection also attempted, with failure to produce anaesthesia or benefit. Sent to me for alcohol injection. Injection of the right Gasserian ganglion by the lateral route produced total trigeminal anaesthesia and complete relief of pain.

This case is an instance of the not uncommon association of disseminated sclerosis and true trigeminal tic.

*B.M.*, a man, aged 43 years, seen January 8, 1942. Sixteen months previously fell heavily down four steps of an air raid shelter, was taken to hospital where he remained four weeks. Has limped ever since on the right leg, and his gait has become unsteady, and the arms also. Slight bladder incontinence, marked pallor of discs. K. J., + +, and R. plantar typically extensor. All abdominal reflexes absent.

Left pupil "tonic"; at first the pupil appeared to be of Argyll Robertson type, but after being kept in a dark room for ten minutes the pupil, now larger, contracted slowly to light. C. S. F. pressure and Queckenstedt tests normal, W.R. negative, 8 lymphocytes, protein 20 mg. Lange 4333332330. Definite coarse ataxic nystagmus was present to the right side only. This is a case of disseminated sclerosis in which the question of injury to the back might be arguable medico-legally as a cause or aggravation of his disease.

The two latter cases I have chosen in order to show an unusual symptom of disseminated sclerosis in each, one with trigeminal tic, the other with a tonic pupil and a history of injury immediately preceding the commencement of symptoms.

The only reference that I have met in the literature to nystagmus resembling what I have described above occurs in Kinnier Wilson's "Neurology," 1940, vol. 1, p. 161, where he quotes Paton as describing "jelly" nystagmus as pathognomonic of disseminated sclerosis. This form may, however, certainly be met with in other conditions, and is quite different from ataxic nystagmus. Wilson goes on to say "One-sided nystagmus is by no means rare; with impaired conjugate lateral movement the outer eye sometimes shows it, or it may be more evident in that one of the two." Here I think Wilson is undoubtedly referring to ataxic nystagmus, but he evidently had not realised that it is pathognomonic of disseminated sclerosis.

I feel sure that many physicians and ophthalmic surgeons must have seen this type of nystagmus when examining cases of disseminated sclerosis, without paying attention to its peculiarity or to its special association with this form of nervous disease.

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