

DISCUSSION OF TREATISE ON NYSTAGMUS

By Dr. R. E. Walters, O.D.

In considering the problem of nystagmus and nystagmoid movements, we must bear in mind that we are dealing with fixation reactions and postures of the eyes that are the net results of several different factors. Following the scheme of Duke-Elder we may list these as follows:

1. Psycho-visual reflexes:
 - a. The primary shift of fixation to place the image, first seen peripherally, upon the macula of one or both eyes.
 - b. The finer, corrective movements to ensure exact, bifoveal, fixation – the “fusional” reaction.
2. Postural reflexes:
 - a. Static:
 1. Tonic reflexes from the otolith organs of the labyrinth. These are dependent upon the position of the head with respect to the gravitational field.
 2. Tonic neck reflexes from the proprioceptive nerve endings in the muscles of the neck. These reflexes depend upon the position of the head with respect to the trunk.

The function of this group is to maintain the visual field, as far as possible in its normal orientation in space, no matter what position the head may assume with respect to the body.
 - b. Stato-kinetic reflexes. These originate at the cristae ampullae of the labyrinth as the results of differences of pressure of the otolymph, the fluid within the semicircular canals, as the head is turned from one position to another. It is essentially a response to accelerated motion of the head – uniform motion appears to have little effect. The function, in this case, as in that of the static reactions, is to maintain the position of the visual fields constant during changes of motion, however, rather than changes of position.

The static reflexes – tonic labyrinthine and tonic neck reflexes are, therefore, response of the eyes to abnormal positions of the head, i.e., positions away from the primary position, whereas the kinetic, labyrinthine reflexes are responses to movements of the head.

Quoting Duke-Elder: “In the normal condition all these reflexes are summated, the one supplementing the other, with the result that there is an extremely well developed correlation of ocular, labyrinthine, and neck reflexes, by means of which, both at movement and at rest, and in the various physiologically possible positions of the head with respect to the body in space, the correct visual attitude and the suitable correlation of the two eyes are ensured”.

With the above basic physiology in mind we may easily conceive that disturbances in any one of the above reflex activities might result in improper coordination of the visual axes. So we find that nystagmus, as a symptom, may have several different causes.

We may have an interference in the first group of reflexes – the psycho-visual or, better, the cortico-ocular reflexes. Such is usually the case in the congenital types of nystagmus.

Here such conditions as albinism, ocular malformations, corneal leukemia following ophthalmia neonatorum, cataract, disease of the retina or choroid and total color blindness, may so lower the effectivity of the afferent side of the visual reflexes, that accurate fixation is impossible. The resulting nystagmus is a rolling, oscillating or random excessive movement of the eyeball.

We may have a similar type of nystagmoid movement following similar conditions that may be acquired in later life, i.e. retinitis, choroiditis, cataract, and rarely high refractive errors, such as aphakia.

If there be disease affecting any part of the paths of the reflexes of the postural group, we may have a nystagmus as one of the major symptoms. Such lesions may be located, on the one hand, in the labyrinth or along the course of the eighth nerve, or on the other hand, may be located within the brain stem, cerebellum, or even in the higher cerebral areas. In the first instance, we would be likely to find a typical aural nystagmus, with the quick and slow components distinctly in evidence. In the latter situations the nystagmus, although no less a manifestation of interference in postural reflexes, might take on a far less rhythmical character.

Objective observations of nystagmus, reveal several types, roughly classified as follows:

1. Pendular or oscillatory movements, wherein the swing is smooth and equal in both directions.
2. Random searching movements. This is typical of amaurotic conditions. These two types are usually found accompanying cortico-visual disturbances.
3. Rhythmic type, with the quick and slow components. This is characteristic of labyrinthine disturbances and is frequently referred to as the aural type. Sometimes this type is referred to as jerky, rather than rhythmic. In either case the quick and slow components are obvious. The slow component is due to an imbalance in the labyrinthine stimuli to the ocular motor nuclei. The quick component is a cerebellar reaction to correct the loss of fixation.
4. Dissociated types. Wherein the two eyes seem to move independently of each other. Frequently found in cerebellar disease.
5. A disjunctive form, wherein the eye movements are toward and away from each other alternately – a rare form.
6. Unilateral – affecting one eye only.
 - a. If it develops only when one eye is covered, it is termed a latent nystagmus.
 - b. If it develops only with an effort of fixation, it is called fixation nystagmus. This is found to a slight extent in normal cases at the extreme limit of lateral or supra-versions, however, a marked exaggeration of this fixation nystagmus is frequently found in diseases of the central nervous system.

While the above discussion has been concerned, chiefly, with pathological forms of nystagmus, it should be born in mind that nystagmus may manifested under experimental conditions as a result of normal physiologic activity. In fact, such tests form the principal methods of investigation of the integrity of the vestibular reflexes. So we find nystagmus manifested in the following clinical tests:

1. Barany's revolving chair test. In this test rotation of the entire body about an axis perpendicular to the plane of the horizontal semi-circular canals will establish momentum of the endolymph within the canals. A sudden stopping of the motion of the body does not immediately stop the acquired motion of the endolymph, which then persists for a short period, causing an exaggerated reflex originating in the cristae ampullae of horizontal canals and reflexly inducing a slow drift of the visual axes away from the primary position and in the direction of the induced flow of the endolymph. The visual axes are then brought sharply back to the primary position by a cerebellar reflex, probably assisted, if the eyes are open, by cerebral reflexes. The cycle is rapidly repeated with diminishing intensity as long as the motion of the endolymph persists – a period of 20 to 60 seconds in the normal individual. With irritative lesions affecting the vestibular pathways a more violent and more persistent nystagmus may result, and conversely, in depressive types of lesions the intensity and duration of the nystagmus is decreased or abolished.
2. A similar reaction occurs when the labyrinths are stimulated by hot or cold douches to the ear, or
3. By increasing or decreasing pressure in the middle ear, or
4. By galvanic stimulation.

The cases submitted by Dr. Elmgren are most interesting. It is unfortunate that one of the original ophthalmograph records is missing. If it showed a type of activity similar to these shown in the subsequent graphs, the writer would consider it to be a case of visual interference rather than labyrinthine. In the booklet by the American Optical Company entitled "Reading in the Class Room", 1936, on page 14, is shown the graph of a case of nystagmus with much larger lateral sweeps than those which appear in Dr. Elmgren's cases. The nystagmoid sweeps in the case of the boy, first graph, top. Are quite evident and appear to be somewhat of the dissociated type. In the A.O. graph the lateral sweeps are of the order of 6 mm or 24 prism diopters, while in Dr. Elmgren's first graph of the boy the excursions are more sinusoidal in character and the extent of the oscillation is from 1 to 3 mm or 2 to 6 prism diopters. Obviously the two types are different. The A.O. case is probably of labyrinthine, and Dr. Elmgren's case is of visual origin.

It is interesting to note the unusual head position assumed by the older patient. Might it not be possible that he was calling upon some tonic neck reflexes to assist in stabilizing a fixation ability that was impaired from some other cause.

Most of our optometric tests in the past have dealt solely with the psycho-visual reflexes of the class 1B cited above, e.g. fusional movements to correct inaccuracies of fixation. Excepting in overt cases of nystagmus, we have given little thought to the fact that all of the above reflexes are continually active during normal use of the eyes. Perhaps we have been too optimistic in assuming that all is well with the labyrinthine and tonic neck reflexes in many of our cases.

In this connection it would be well to consider that one very common symptom encountered in our daily practice is a tenseness or aching of the neck muscles. Another is the head tilt, frequently encountered. To what extent do the tonic neck reflexes and labyrinthine impulses enter this clinical picture? Are the hyperphorias associated with the head tilts causes or effects – or are both symptoms the result of

imbalances within the proprioceptive system of which we know too little? A fertile field for research is here indicated.

Dr. Elmgren is to be congratulated upon his courage in tackling cases of this type. His work points the way to further study in this field.

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DR. ELMGREN'S DISCUSSION OF DR. WALTER'S DISCUSSION ON NYSTAGMUS

Dr. Walters has been very kind to me in the discussion of my paper on Nystagmus. I expected a severe critical discussion, especially so an account of not presenting a "manifest" or "labyrinthine" case. One reason for not doing so was, that I had shortly before completed a paper for the Year Book of Optometry, showing such case.

Several Ophthalmographs were made of the cases presented, a few of which were attached to the manuscript for record, which cannot be printed in the Syntonogram, but they should be on file at the College.

On page 122, "Controlled Reading", by Earle A. Taylor, Nystagmus graphs are shown.

Page 321 and 323, Year Book of Optometry 1938, is a case presented by Dr. Bondelid where prisms were prescribed for a Nystagmus patient.

In the above mentioned Year Book, pages 325 to 335, there is a paper on reading habits and several ophthalmographs by me. The graphs on page 335 is such case as referred to by Dr. Walters and found on page 14 in the A.O. company booklet. On page 332, graph B where I use the phrase "bad Eye-habits", it is nystagmus, but I did not use the word nystagmus for a special reason. Nystagmus is used on other graphs.

In conclusion I wish to emphasize the fact that Nystagmus is far more prevalent than what is generally believed to be. The ophthalmograph opened the pathway to research on many ocular functions.