

A REVIEW AND FUTURE PROBLEMS OF POSITIONAL NYSTAGMUS

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Nystagmus which occurs when certain positions or postures are kept up and is absent in others is known as a positional nystagmus. It is usually accompanied by vertigo described as a sense of movement or whirling of an object or of the environment, or the subject. It is preferable to discuss the syndrome in terms of the objective sign, the positional nystagmus, because vertigo is a subjective sensation and therefore not always susceptible to exact evaluation.

Breuer¹⁾ in 1874 and Ewald²⁾ in 1892 reported their animal experiments on the inner ear and directed attention to the importance of the otoliths as a staticsreceptor organ. Valuable results of Magnus and de Kleyn's³⁾ animal experiments (1921) marked a new stage in the study of problems of function of the otoliths and actually stimulated various investigators to study more closely the otolithine mechanism, while Bárány⁴⁾ had earlier had in mind when he, on occasional cases, observed nystagmus in certain head positions.

Positional nystagmus was at first thought to indicate a disturbance of the otoliths, hence it was known as "Otolith nystagmus".⁵⁾⁶⁾ As clinical and experimental studies have advanced, however, it was soon made apparent that the positional nystagmus not only appeared in various affections of the labyrinth but also in retrolabyrinthine or central disturbances. It is possible that positional nystagmus due to labyrinthine affections may originate in the otoliths but it has so far not been possible to obtain evidence. The otoliths of the lower animals were proved to produce counter rolling movements of the eyes,⁷⁾⁸⁾ but the problem as to whether they may cause a positional nystagmus remains even now undecided.

A positional nystagmus of central origin has been produced experimentally after bilateral labyrinthectomy,⁹⁾ hence the phenomenon does not depend on the appearance of a functioning receptor organ.

In the present state of our information the primary significance lies in the fact that the positional nystagmus which accompanies the complaint affords proof of an organic disease of the vestibular mechanism.

Knowledge obtainable from characteristics of nystagmus as to localization may frequently be of definite value but the diagnostic implications of the various types have not yet been fully explored. It is of practical importance to bear in mind that a posture vertigo may escape detection unless specific questions are asked to elicit such information as bear on vertigo and a positive history is followed up by examination for positional nystagmus.

Among the European and American researchers beside Nylén¹⁰⁾ Lindsay,¹¹⁾ Ruttin,¹²⁾ Kobrak,¹³⁾ Seiferth,¹⁴⁾ Mygind,¹⁵⁾ Guttith,¹⁶⁾ and Frenzel¹⁶⁾ have published many articles on the subject.

Definition:—Positional nystagmus is a spontaneous nystagmus, appearance of which is influenced by head position; from the clinical point of view it is of especial importance for us to differentiate between a spontaneous and a positional nystagmus. A spontaneous nystagmus is present in normal erect position and continues to be present in the same intensity without regard to change in head positions.

Because of the many variations which appear, definition of positional nystagmus is far from being single. In the opinions of some writers, only that form of nystagmus which alters the direction of its quick component in certain head positions is recognizable as true positional nystagmus.

Nylén¹⁸⁾ stated "My opinion is, however, that it is more suitable—not least from the practical point of view—to differentiate between spontaneous and positional nystagmus. By spontaneous nystagmus is thus meant a nystagmus which is not influenced by the position of the head, but which constantly alike in all head positions, while positional nystagmus in one way or another is clearly altered when the head adopts another position."

On the other hand Lindsay¹⁹⁾ said, "For practical reasons it is considerably preferable to include as positional all cases in which the nystagmus appears when certain positions are assumed and is absent in other positions, and within this general group may be distinguished several sub-groups which will be referred to under classification."

Classification:—Experimental studies by de Kleyn and Versteegh,²⁰⁾ Nylén,²¹⁾ Chilow,²²⁾ Formann and Nylén⁹⁾ and Speigel and Sclala²³⁾ have demonstrated that a positional nystagmus may be presented by several types of disturbance affecting either peripheral or central organ of the vestibular system. On the other hand clinical observations by Nylén²⁴⁾ on cases of brain tumor, by Seiferth¹⁴⁾ in other disease states, as well as by Ruttin,¹²⁾ Frenzel,²⁵⁾ Lindsay²⁶⁾ and others have demonstrated the frequency of syndrome in certain diseases of both central and peripheral localization.

It is clear from the researches by the above mentioned authors that a practical differentiation between various types of positional nystagmus is pos-

sible apart from with a few exceptions. Classifications of positional nystagmus were proposed by Ruttin,¹²⁾ Nylén,¹⁸⁾ Lindsay,¹⁹⁾ Seiferth¹⁴⁾ and Meyer.²⁷⁾

Of these there is some agreement on two types which seem to possess certain significance in differentiating a ~~central~~ from peripheral lesion. In succeeding paragraphs I shall give an outline of these types and their subtypes.

Type I. Direction-changing positional nystagmus (Nylén's type 1, Seiferth's type 1, Ruttin's type 1, Lindsay's type 1, Frenzel's true positional nystagmus).

The direction of quick component is altered in certain head positions, generally being reversed in the opposite head positions. For instance it may be horizontal in lateral head position or vertical or oblique in some other positions.

Using the heading of a direction-changing nystagmus it is possible to differentiate regular and irregular type.

Thus sub-group (1) might be called "regular" and sub-groups (2) (3) and (4) variations of irregular positional nystagmus.

Irregular positional nystagmus is classed as Type 3 by Nylén. Regarding this matter, Lindsay stated that in view of the fact that all of these sub-groups show the direction-changing characteristic either constantly or intermittently, they are all listed here as sub-groups of Type 1 positional nystagmus.

(1) Direction-changing positional nystagmus which is usually maintained constantly. A common form is that in which the direction reverses in opposite head position.

(2) Direction-changing but the nystagmus has a limited duration, usually less than one minute.

Another variation of this type is that in which the appearance of the nystagmus depends on the rate of alteration in position. For instance, if the alteration from the supine to a lateral position, or from supine to an upright position is carried out slowly the nystagmus may not occur but if the alteration of position is repeated a little faster, the nystagmus may present itself in its usual intensity and for this usual duration.

Inasmuch as many clinical cases such as those due to head trauma or to vasomotor insufficiency may correspond to this type, the clinical significance of repeating the test is plain.

On repeated tests this type may sometimes be proved to be a minor variation in its characteristics.

(3) Positional nystagmus may make its appearance before the customary change of ninety degrees in position is completed or when the change

has progressed to a certain point in one direction. Hence it may then be sustained or only temporary in duration.

(4) Another variation of positional nystagmus which is usually of the direction changing type is that in which the positional nystagmus may appear only when the previously held position has been maintained for a considerable period of time. For example, a positional nystagmus that occurs on turning to a side position from the supine may fail to appear unless the supine position has been held for some minutes before turning to the side position. Such an irregularity is not necessarily confined to type I.

Type II. Direction-fixed nystagmus. In this type the nystagmus may arise only in certain positions, but it is constant in its direction (Nylén's type II, Frenzel's spontaneous nystagmus released by position stimulus).

This type may be divided into two sub-groups.

(1) There is no spontaneous nystagmus but a positional nystagmus appears in one or more positions and is constant as to direction of quick component.

(2) A spontaneous nystagmus arises usually in all head position, but an increase in its intensity appears in certain positions, the direction remaining the same.

Method of observation:—In recent years in Europe and America posture test has gradually been considered as a method of nystagmus test among other clinical vestibular examinations but it has not yet received similar attention in our country.

This test, however, does not yet seem to be universally accepted in spite of the fact that it is considered by its supporters as indispensable to vestibular diagnosis. One of the reasons for its failure to be recognized universally, is the fact that positional nystagmus is a greatly varying phenomenon.

In rare cases the postural nystagmus or vertigo is indicated in patient's own description of his symptoms but a few specific questions will bring out this characteristic in detail since patients become aware of those position which they must avoid.

In performing the ear functional examination, postural tests are best preceded by the routine otoscopic examination, hearing tests, examination for spontaneous nystagmus exclusion of a labyrinthine fistula and the head shaking test.

According to practical demands, the posture test is performed in various ways. One can simply let the patient sit or stand and from there incline his head to the right or to the left shoulder, or bend his head forward and back.

This method, however, has obvious weakness. The patient cannot be

examined in the very important position, namely hanging position, and the stimulus provided here does not always seen to be sufficiently powerful to produce nystagmus. This method is rapid and simple, but it is dependable only for use in a large daily practice, as pointed out by Eckel.

From a practical point of view the posture test should be so carried out that the patient on an ordinary hench adopts the position which are considered to be necessary. These should be:

1. In the sagittal plane, sitting or standing, supine and hanging position.
2. In the frontal plain; right and left lateral positions respectively with the supine position as starting-point.

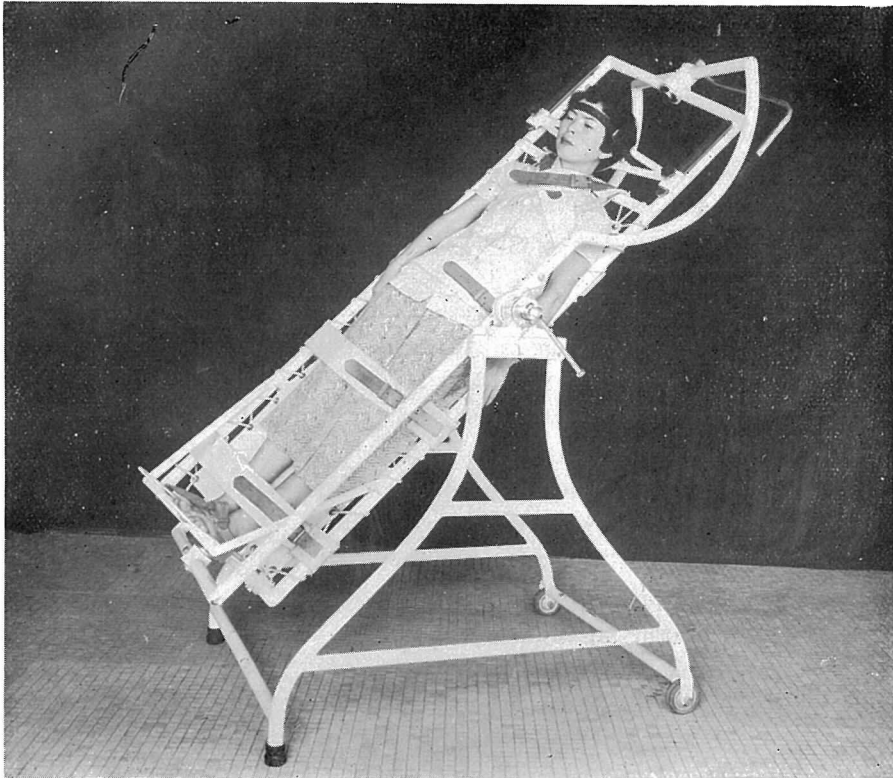


Fig. 1. Posture table being used at our clinic. Change of patient's head position is smoothly made in three dimensions.

If no nystagmus appear in other of the lateral positions the patient should at once be transferred to the side position first adopted, so that a latent nystagmus may possibly make its appearance (V. Devivere, de Kleyn²⁸),

In order to exclude neck reflexes the patient's head and body should

be moved together in changing the position. This is generally easy to carry out, except in the very important hanging position particularly the test in which the head is turned to the right and left sides respectively. In a human subject, however, the neck reflexes have a negligible effect on eye movements and for this reason do not complicate the tests.

If one wishes to be reassured that the presenting phenomenon should be entitled positional nystagmus it becomes necessary to employ a specially constructed the so-called "posture-table" (See Fig. 1), as the above listed sources of error can then be avoided.

Change of patient's position should be made very slowly as advised by Williams. Usually a position can be changed 90 in about 5 seconds. In accurate tests the position change should commence at a low speed of 1-2 per second and very gradually speeded up the usual rate as mentioned above. In these test an electrically driven posture table is desirable. Special tables have been constructed by Grahe²⁹⁾ and Nylèn⁸⁾ to permit the patient to be passively shifted into all postures and to allow photography of eye-movements during the examinations.

Fig. 1 shows the author's modification of the posture table being used at the author's Clinic.

Observations of the eyes for positional nystagmus have heretofore been generally done with or without the use of 20 diopter lenses to removing fixation. Lateral gaze is not necessary because of a complication arising in those cases presenting a spontaneous nystagmus in the direction of gaze. While the patient's history indicates which positions are likely to yield positive results the completion of the whole series of positions is advisable since further information may thus be obtained.

The order in which the tests were found to be convenient is as follow:
1) Sitting on the table in erect posture, 2) Shifting from erect to supine, 3) from supine to right lateral, 4) from right lateral to supine, 5) from supine to left lateral, 6) from left lateral to supine, 7) head extending over the end of the table, 8) supine position, 9) shifting from supine to erect, 10) Feet over the side of the table and stooping forwards the headhanging position, and from stooping over to the erect position (Lindsay).¹⁹⁾

The above positions and movements cited are generally in common everyday use with the exception of head extended over the end of the table. This position introduces possible factors of tension on neck muscles and circulatory interference.

A factor which is of special significance is the rate at which the positions are changed. The positional nystagmus is independent of the rate of change in position in certain clinical conditions. The nystagmus may be

temporary or may be maintained while in that position. In other clinical conditions the syndrome may be dependent on the rate at which the new position has been reached although it may appear either before or after the new position was completely attained. In Lindsay's¹¹⁾ report this latter type is one of the varieties most frequently seen in office practice and includes many cases where the underlying etiology is related to vascular or vasomotor insufficiency as well as many cases of vertigo following head trauma. In such cases it may be necessary to change the position at a certain rate in order to bring on the vertigo and nystagmus. Objection has been raised to the speedy changing of position since the reaction may then be due to the movement factor rather than position. It must, however, be pointed out that although a slow change of position may fail to elicit the symptom in certain cases, these subjects may experience severe vertigo if the movements are carried out at the rate which is customary and necessary in their daily routine activities.

The first objective of the postural tests is to reproduce the syndrome. Therefore if a slow change of position fails in spite of the presence of a positive history, the test must be repeated more rapidly the position may be changed slowly on the first test and if negative may be repeated at a more rapid rate.

A continuous recording of the eye movements has been made using a special position table with picture camera attached (Nylén)¹⁸⁾; it can also be achieved by recording the corneo-retinal potentials during the tests.

Contact lens shown Fig. 2 was employed to observe the eye movement at the author's clinic.

Clinical significance:—The presence of positional nystagmus makes it apparent that there is a disturbance in the vestibular system, in which are included the vestibular organ in the inner ear, nervus vestibularis with its centres in the brain. As many physicians have an erroneous opinion that positional nystagmus appears only in central affections, it is very important to point out that the phenomenon can appear in diseases in widely different spheres affecting the balance system. The proof of this assertion is given in the following line: Positional nystagmus may be present wherever the inner ear is involved in, for example, inflammation, haemorrhage, thrombosis, emboli, circulatory and secretory disturbance, degeneration and toxic conditions; in case of nervus vestibularis: in neuritis, meningitis, tumors, vascular anomalies, degeneration, atrophy, etc.; in connection with the brain in encephalitis of different types, tumors, abscesses, increased intracranial pressure, syphilis, arteriosclerosis, hypertension, hypotension, allergic and toxic conditions, cranial trauma, haemorrhage, emboli or thrombosis.

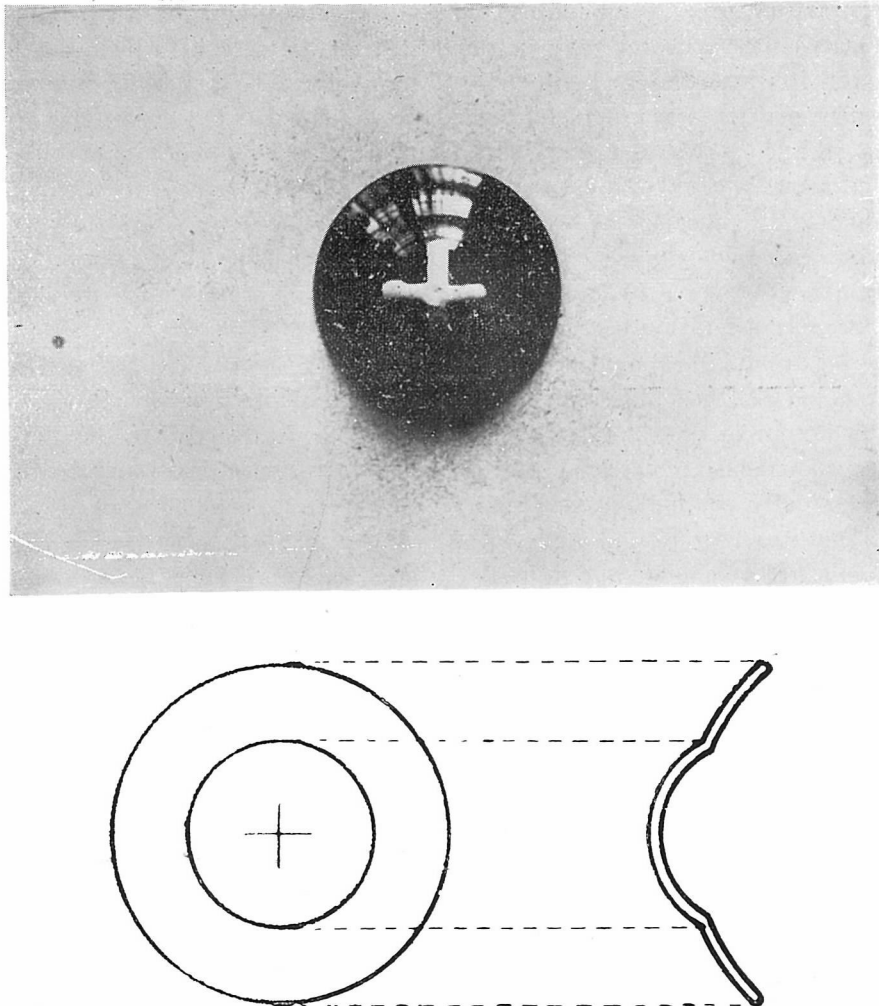


Fig. 2. Contact lens.

Photograph and diagram showing contact lens employed to observe vestibular nystagmus. This black lens is fixed on the cornea in order to except ocular nystagmus.

Nylén¹⁸⁾ stated that direction changing irregular positional nystagmus speak first for a central affection, while direction fixed positional nystagmus may appear in labyrinthine and retrolabyrinthine morbid conditions as well as in centrally localized disease. These statements should not, however, be accepted as a rule without exception.

1) Blomqvist³⁰⁾ reported that positional nystagmus was present in 26

of 571 cases of acute middle ear inflammation in hospitalized and out-patients. In the great majority of these cases positional nystagmus was direction-fixed towards the affected side, while three cases were direction-changing and only one case was direction-fixed to the normal side from beginning of the affected side. In five cases the direction of the nystagmus was changed during the course of the disease from the affected to the normal side. The presence of positional nystagmus in cases of labyrinthitis must be taken as a sign that the affected vestibular organ or at least part of it, still functions. Smidt³¹⁾ Lund³²⁾ and Gering³³⁾ described the relatively common appearance of positional nystagmus in labyrinthitis.

2) In Ménière's syndrome, positional nystagmus has frequently been observed during attacks (Thornval,³⁴⁾ Mygind,¹⁵⁾ V. Eicken,³⁵⁾ Nylén,¹⁰⁾ Lund,³²⁾ Meyer,²⁷⁾ Lindsay,¹¹⁾ Lewy,³⁶⁾ Arnvig,³⁷⁾ Björk³⁸⁾). Many writers, however, speak of spontaneous nystagmus, but the influence of the position of the head was certainly not tested then or at least not examined at the right moment. If this was done, positional nystagmus would be found as a very common occurrence.

Nylén and Krey³⁹⁾ examined a series of 118 cases of Ménière's syndrome. Of these the symptoms were typical for the syndrome, still frequently referred to as Morbus Ménière, in 47 cases. In the remaining 71 cases the symptoms were such as would make the same diagnosis probable. Their findings may be summarized as follows: (1) Nystagmus was found in 83 cases, 55 of which being affected by position, (2) of the 55 cases 13 had direction-changing and 42 direction-fixed nystagmus, (3) of the 47 typical Ménière cases, 29 had nystagmus, of these 6 direction-changing, 15 direction-fixed positional and 8 spontaneous nystagmus. (4) in the 17 cases in which only one ear could be pointed out as being diseased, nystagmus was in the great majority of cases directed towards the normal ear when the hearing was obviously impaired, and towards the affected ear when the hearing power was only slightly impaired.

Lindsay²⁶⁾ observed positional nystagmus in only 4 out of 66 cases of typical Ménières disease, and that in 29 of 30 cases of atypical syndroma Ménière, direction-changing positional nystagmus was present.

One can presume that the direction-fixed positional nystagmus towards the affected or healthy side in cases with functioning labyrinth is above all an expression of peripheral etiology, such as disturbances in circulation or secretion, in the metabolism of salts, vitamins, hormones, fluids, proteins etc. As if in support of this presumption, the statements of Brühl,⁴⁰⁾ F. Alexander and Manasse,⁴¹⁾ Mayer,²⁷⁾ Portmann,⁴²⁾ Mygind and Dederling,⁴³⁾ Watkyn-Thomas and Lowndes Yates,⁴⁴⁾ Furstenderg,⁴⁵⁾ Granström and Nylén,

Dohlman,⁴⁶⁾ Kjaer,⁴⁷⁾ Adam,⁴⁸⁾ Atkinson,⁴⁹⁾ Asherson,⁵⁰⁾ Godtfredsen,⁵¹⁾ are of greatest significance. The histological findings from the human ear by Wittmack,⁵²⁾ Hellman,⁵³⁾ Fowler,⁵⁴⁾ Lindsay¹⁹⁾ are of the greatest interest in this aspect.

The direction-changing from of positional nystagmus is more indicative of a cause localized in the vestibular system. Thornval,¹⁴⁾ Nager,⁵⁵⁾ Skoog,⁵⁶⁾ Nylén,¹⁸⁾ Lindsay,¹⁹⁾ Atkinson⁴⁹⁾ and Herberts⁵⁷⁾ are of the opinion that Ménière syndrome may possibly also have a central mechanism.

Vernet and Ferrer maintain that, the labyrinthine hydrops, for example, which has lately been widely discussed as a cause of Ménière's syndrome, especially through the work of Hallpike-Cairns-Wright,⁵⁸⁾ should not be considered as a necessary qualification for, but as a result of an attack of syndrome. Nager pointed out in 1949 that there is a possibility that a small number of cases of aural vertigo without labyrinthine ectasias may constitute a special group of the disease.

3) It is usual to find positional nystagmus in person after taking drugs of bartiturate group such as veronal or luminal or after exposure to carbon monoxide Nyman,⁵⁶⁾ Almgren,⁵⁷⁾ Noro⁵⁸⁾. In experiments on animals, other substances such as quinine, salicylic acid or alcohol have also produced spontaneous nystagmus or positional nystagmus (Seiferth,¹⁴⁾ Falbe-Hausen,⁵⁹⁾ Rothfeld,⁶⁰⁾ de Kleyn-Versteegh⁶¹⁾). In humans also, ethylalcohol intoxication is prominent in this connection according to Frenzel,¹⁷⁾ who described a regular from of direction-changing positional nystagmus which corresponds with that produced by luminal. According to Meyer, a nystagmus in lateral position is observed after sufficiently high dosage. Generally this nystagmus is horizontal directed. Importance of the findings in the various positions cited above to forensic medicine was pointed out by Pleakers. He relates that positional nystagmus, during a certain number of hours—up to about 6 hours after commencing intoxication—was directed to the side of position, but that later on the direction was opposite. In other words, positional nystagmus is not only a sign of toxic influence on the centers, but the direction of the nystagmus will, within certain bounds, be able to reveal something about the commencement of the intoxication. It is of importance for the physician always to hear in mind that certain drugs can him differential diagnostic difficulties when he is recommending especially large doses.

4) According to Seiferth,¹⁴⁾ Beyer,⁶²⁾ Nylén,²⁴⁾ Lindsay¹⁹⁾ and Boenninghaus⁶³⁾ positional nystagmus may occur following skull trauma, and in the opinion of the first named author, this is probable the most constant symptom of all after an injury to the cranium. Direction-changing or

irregular types of positional nystagmus indicating central trauma, are very common. It should be pointed out that under all circumstances a posture-test should be included in neurological examination in connection with injury, more especially as the symptom is now usually considered to be an expression of more or less serious damage in the vicinity of the vestibular nuclei in the brain-stem (Tonvies, Barbey) and is seldom thought to depend on a so-called commotio labyrinthitis.

5) Positional nystagmus is a common appearance in encephalitis of different kinds, acute and chronic. Nylén reported some cases in which this symptom was the only demonstrable neurological sign of the disease indicating a pathological change near or in the vestibular centers at the bottom of the fourth ventricle. In sclérose en plaques nystagmus appears, according to von Leden-Horton in 43 per cent (in 1948). When the patients are tested at different head positions the cases with nystagmus are numerous, 90 per cent, or more. From has found that nystagmus is affected by position in almost two-thirds of these cases, direction-changing in about 33 per cent, of these and in the remainder direction-fixed.

6) Positional nystagmus is a very common symptom in tumor of the brain. Nylén²⁴ in a series of 673 verified cases of brain tumor demonstrated a positional nystagmus in 279 of the 360 cases with nystagmus (80.6 per cent). In 50 per cent the positional nystagmus was direction-fixed, in 30 per cent direction-changing and in 20 per cent the nystagmus was spontaneous.

In subtentorial tumor positional nystagmus of all types was found in 69 per cent of 240 cases, while in supratentorial tumors the incidence was 26 per cent of 433 cases. Nylén also observed that in tumors of the posterior fossa the positional nystagmus most frequently appeared in the side positions or in many other positions while in supratentorial lesions it was most frequent in the hanging head position. Regarding the occurrence of positional nystagmus in brain tumors of various structures and the mode of growth in their typical localizations, such as acoustic neurinoma and different types of glioma in the cerebellum, the following discussion in pathogenesis will clarify the matter.

Pathogenesis:—The appearance of positional nystagmus may be interpreted uniformly by the assumption of pathologically altered tonus in all or part of the vestibular system. In spontaneous nystagmus the entire vestibular system on one side is engaged alike in all positions of the head. In direction-changing or irregular positional nystagmus, on the contrary, the tonus preponderance in the vestibular system of both sides asserts itself by turns, though only in certain head positions. Direction-fixed positional nystagmus

appears as a result of tonus-prepondrance in the vestibular system of one side when the patient adopts certain head positions.

Behind these conditions of altered tonus there are as primary causes different types of processes, arising in the first place as a result of disturbances in circulation and capillary permeability, vasomotor disorder, compression, thrombosis, hyper- and hypotension, arteriosclerosis, etc. The contents of blood-vessels, labyrinth- and cerebrospinal fluids, such as toxins, bacteria, hormones, vitamins, salts, proteins, drugs, and other substances also play an important part in the development of pathological tonus conditions in the vestibular sense organs and in their connections with different parts of the brain. Finally the tonus status may become pathological in various types of intracranial disease, some of which bring about, among other things, altered pressure conditions.

Peripheral affections. The positional nystagmus observed in acute inflammatory states customarily occurs in the early stages of inner ear involvement according to experimental and clinical observations. If the inflammatory reaction increases in severity a spontaneous nystagmus appears. The positional nystagmus may thus represent an initial stage in the development of diffuse labyrinthitis.

In cases of secretory otitis media the inner ear reaction may remain in the initial stage probably because the inflammatory process in the middle ear is sterile and the effect on the inner ear remains limited in degree.

The positional nystagmus which occurs and persists for months or years following a single labyrinthine disturbance requires a somewhat different explanation. A disturbance in a vestibular receptor of a more permanent nature seen to be the plausible explanation.

Central affections. Experimentally it was shown that a positional nystagmus might be produced by a lesion of various parts of the central vestibular system. It may be produced without participation of the peripheral sense organs, may arise from the vestibular nuclei, and from the cerebellar connections of the vestibular system. Clinical observations indicate that it may be produced by a number of diseased states, including an impairment of vascular supply or a local vasomotor disturbance or intoxications involving certain nerve elements and the pressure effect of tumors.

The vestibular centers, primarily the nuclei may be considered as normally in a state of equilibrium. It has been suggested by Nylén¹⁸⁾ that the presence of a diseased state affecting on one side or both sides unequally might result in an abnormal response to a normal peripheral stimulus. Such an abnormal response may occur also without participation of the peripheral sense organs.

Treatment:—Positional nystagmus is a symptom of variety of conditions affecting the vestibular system. Therapy is directed primarily at the etiologic process. In the event that the presence of a definite systemic or local disease cannot be established the treatment may be limited to an attempt at relieving the symptoms.

When vertigo is postural in character a specific history regarding the effect of various positions and a routine set of postural tests not only provide factual objective evidence but give the patient assurance that his problem is appreciated. While the treatment is usually directed to the etiologic disease, many cases are encountered in which an etiologic diagnosis cannot be made with certainty.

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