ALTERNATING HYPERPHORIA*

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THE original aim of this investigation was the classification of cases of hypertropia and hyperphoria. Much more frequently than we had expected, we found alternating hyperphoria, and almost all the cases of hypertropia which were not clearly of a paretic nature proved to be complicated by this phenomenon.

When a large number of cases of hypertropia and hyperphoria had been examined, it became evident that a definite syndrome was present in the majority, alternating hyperphoria being the dominating feature.

The material was provided by the patients of the Amsterdam Motility Clinic. Alternating hyperphoria proved to be so frequent that more than one hundred cases were noted in 18 months.

Methods of Examination

Most of the patients were examined several times. Special attention was paid to hereditary factors, to the time when squinting was first observed, and to nystagmus.

The horizontal angle of squint and the degree of hypertropia were measured by the method of reflex images and a tangent-screen. Quantitative data concerning the vertical angle of squint seldom came within the scope of this study.

The following directions of gaze were examined:

- (1) Primary direction of gaze, also with head tilted to the right and to the left.
- (2) Looking to the right and to the left.
- (3) Looking upwards and downwards.

When possible we always examined with both the right and the left eye fixing. Examinations in diagonal directions of gaze were frequently carried out but produced no useful data. Examination of the position of the eyes by means of subjective methods was often unsuccessful because of the absence of diplopia either through suppression or through abnormal retinal correspondence. Nevertheless examination by means of the Maddox rod yielded important results to which we shall return later on in this paper. Cyclophoria was determined by two Maddox rods (the vertical lines observed had to be placed by the patient perpendicularly upon each other), or by the synoptophore.

Occlusion of the Eye.—As a rule one of the eyes was occluded by the examiner's hand, while the non-occluded eye was directed to a spot-light. All the movements of both eyes were carefully examined (the occluded eye by studying it from the side behind the hand.) At first we examined the reactions to occlusion in the various directions of gaze, but they are not to be influenced to any appreciable degree, and

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this examination was later omitted. Bielschowsky's phenomenon, in which the non-fixing eye deviates downwards when a dark glass is placed before the fixing eye, was looked for in every patient.

The Syndrome

All the patients showed the following anomalies to a greater or less degree:

(1) *Squint*.—This squint dated from birth in 50 per cent. of the cases, and from the earliest years of childhood in the rest. A squint starting after the second year was rare. In 55 per cent. squinting was hereditary, according to the statement of the parents. Convergent squint was much more frequent than divergent squint.

(2) *Hypertropia in Lateral Directions of Gaze*.—In the primary position there was hypertropia of the non-fixing eye or none at all. In the lateral directions of gaze there was almost always hypertropia. In most cases the non-fixing eye turned upwards in the position of adduction, in a few it turned up in the position of abduction. This hypertropia did not change when the gaze was raised or lowered.

(3) *Nystagmus*.—This was pendular and rotatory. In unilateral severe amblyopia the amblyopic eye often showed monocular vertical nystagmus. If one of the eyes was covered the rotatory pendular nystagmus changed into jerk (latent) nystagmus, the rapid phase of which was directed to the side of the fixing eye.

(4) *Alternating Hyperphoria*.—When one eye was covered it rotated upward with a slow movement. If a dark glass was placed in front of the fixing eye the other eye might deviate downwards to below the horizontal (Bielschowsky's phenomenon).

(5) *Torsions*.—There was excycloduction of the covered eye, and incycloduction of the fixing eye.

(6) *Torticollis towards the fixing eye*.—In some cases the head was tilted on the sagittal axis towards the shoulder on the side of the fixing eye.

Classification of Cases

The 113 patients were divided into five groups (Figure):

- (1) Esotropia surso-adductoria (with hypertropia of the squinting eye in adduction).
- (2) Esotropia surso-abductoria (with hypertropia of the squinting eye in abduction).
- (3) Alternating hyperphoria without horizontal squint.*
- (4) Exotropia surso-abductoria.
- (5) Exotropia surso-adductoria.

The patients fell into the following age groups:

Age Group (yrs)	No. of Patients
0-5	19
6-10	34
11-20	34
21-30	17
31-60	9

*In all these cases horizontal squint had existed earlier on.

The Figure also shows the following facts:

- (1) Esotropia is generally surso-adductory but the preponderance of esotropia surso-adductoria diminishes with age.
- (2) Exotropia is almost always surso-abductory.
- (3) When the horizontal squint is entirely, or almost entirely, lacking there is as a rule no hypertropia in the lateral directions of gaze.

FIGURE.—Distribution of cases by age group and ocular condition.

Occlusion of the Right Eye.—The right eye turned up with a slow movement and underwent a slight excycloduction. The left eye showed a slight incycloduction.

*This showed that we are not dealing with an ordinary case of paresis of a superior oblique muscle.

Characteristic Cases of Alternating Hyperphoria

Case 1, a girl aged 16, had had a squint since birth. In 1941 a tenotomy was performed of both internal recti with advancement of both external recti. Her visual acuity was 5/5 (+2D sph., +3/4D cyl.) in the right eye, and 5/10 (+2D sph., +1D cyl.) in the left eye. The media and fundi were normal; there was no torticollis; rotatory nystagmus was present. The rotatory oscillations were slow and of small amplitude.

There was still an esotropia of 5°. The right eye fixed habitually but the patient could quite easily fix with the left eye. On fixing with the right eye there was a slight hypertropia of the left eye. This hypertropia increased on looking to the right and disappeared on looking to the left. Looking up and down had no influence on the vertical deviation. When the head was tilted towards the right shoulder, the left eye assumed a slightly higher position, whereas on the contrary the vertical deviation decreased when the head was tilted towards the left shoulder.*

With the left eye fixing, the position of the eyes in various directions of gaze behaved in a precisely symmetrical manner. In the primary position the right eye was too high and this increased on looking to the left and decreased on looking to the right. The vertical deviation was now greater when the head was tilted towards the left shoulder and decreased when the head was tilted towards the right shoulder. The examination with the Maddox rod did not succeed owing to suppression.

Occlusion of the Left Eye.—The left eye rotated upwards in the same way with excyclo-duction. The right eye showed incyclo-duction. A dark glass placed in front of the fixing eye partly caused the covering reactions to disappear.

If, during fixation with the left eye, the covered right eye was left free, it sometimes remained elevated; it assumed its ordinary position again if a dark glass was for a moment placed in front of the left eye. If one eye was first covered and the hand was transferred to the other eye, the originally occluded eye was forced to turn down in order to take over the fixation; the now occluded eye also made this downward movement, and then, as described, it rotated upward.

Synoptophore.—Suppression, no fusion; normal retinal correspondence.

Not all the patients exhibited as many anomalies as Case 1, who belongs to the first group.

In many patients with esotropia surso-adductoria the occlusion hypertropia was hardly visible. It is remarkable, that the alternating hyperphoria after an operation for the esotropia often became manifest.

Case 2, a girl aged 9, had squinted from the 6th month. There was no squint in the family. In 1949 the angle of squint was 55° , with correcting glasses it was 45° . On the left eye an advancement of the external rectus and a tenotomy of the internal rectus were performed.

Her visual acuity was 5/8. (+5D sph., +1D cyl.), in the right eye and 5/300 (+6D sph., +1D cyl.) in the left eye. The media and fundi were normal. There was torticollis towards the right shoulder. The left eye showed a vertical monocular pendular nystagmus. There was still an esotropia of 12° . In the primary position there was no hypertropia. Only on looking to the left and on tilting the head to the right shoulder did the left eye assume a higher position. At the same time it was noticeable that the horizontal angle of squint increased when the patient looked upwards and decreased when she looked downward (Urist, 1951).

Occlusion.—When the right eye was covered it became slightly excyclo-ducted; the left eye deviated downwards below the horizontal with incyclo-duction.

When the left eye was covered it rotated upwards whilst the right eye showed incyclo-duction.

This patient belongs to the second group with esotropia surso-abductoria. The left eye lacked the power of fixation, and did not take over the fixation when the right eye was covered. Instead of the right eye rotating upward, the left eye deviated downward. It is remarkable that nevertheless excyclo-duction of the right eye took place.

Case 3, a girl aged 11, started to squint in her early childhood. An old case report stated: "convergent squint, insufficiency of the right superior oblique muscle, alternating hyperphoria". Her visual acuity was 5/10 (−2D sph., −1D cyl., 7°) in the right eye, and 5/15 (−3D sph., −1D cyl., 7°) in the left.

The media and fundi were normal. There was no torticollis. There was a strong rotatory pendular nystagmus which when the eye was covered changed into horizontal jerky nystagmus. This was especially marked when the right eye was covered. The eyes remained straight in all directions of gaze except when the adducted eye was covered by the bridge of the nose, and the typical covering reactions made their appearance. On testing with the Maddox rod there was an exophoria of 3° . The hyperphoria fluctuated: at first the red line was indicated on both eyes under the lamp (the Maddox rod then caused an occlusion hypertropia). After a few seconds the lamp and the red line

were seen at the same level in all directions of gaze. If the patient was asked to fix the red line with special attention, the red line then came above the lamp (Maddox rod before the right or left eye).

Occlusion.—Covering the right eye caused upward rotation and excycloduction of this eye and, incycloduction of the left eye. Covering the left eye caused elevation and excycloduction of this eye and incycloduction of the right eye. The reactions on covering the eye were distinct and were found to be equal in all directions of gaze as well as on tilting the head towards the shoulders. The occlusion hypertropia did not noticeably decrease when the gaze was directed upwards, and the ratio between the vertical and rotatory component did not change according as the occluded eye was in the position of adduction or abduction.†

The covering reactions took place promptly, even if the patient was placed in a lighted room in front of the spot-light and the line between one of the eyes and the light was interrupted by a finger at a distance of 10 cm. in front of the eye. A strong convex lens (+20D sph.) placed in front of one of the eyes caused it to rotate upwards. If a prism of 10D with base placed nasally was put in front of one eye, this eye deviated upwards. If at the same time the other eye was covered it remained elevated even after it had again been freed.

Bielschowsky's darkening phenomenon was however negative and the covering hyperphoria appeared behind a screen brightly illuminated on the eye-side just as clearly as it did behind a dark screen.

Synoptophore.—A horizontal fusional amplitude of $+6^\circ$ to -5° was found. There was an enormous vertical fusional amplitude from $+14^\circ$ to -15° ‡. The fusion was however unstable, a moment later it could not be demonstrated owing to alternating suppression.

This patient, who did not squint, undoubtedly had binocular vision, and showed no hypertropia in lateral directions of the gaze, conformed the most nearly to Bielschowsky's classical description. Nevertheless in this case too there is no question of an "essential" alternating hyperphoria: there was in her previous medical history a convergent squint, with hypertropia of the adducted eye, which meant that she belonged to the first group. At the present time there is still a distinct defect of binocular vision, as appears from the examination with the synoptophore.

Finally, the nystagmus, and the rotatory deviations on occlusion, are much too prominent to be omitted in the description of the complex of symptoms.

Covering Reactions

As a general rule the following reactions took place when one eye was covered:

- (1) Elevation and excycloduction of the covered eye.
- (2) Incycloduction of the fixing eye.
- (3) Nystagmus with rapid phase in the direction of the fixing eye.

If the uncovered eye lacked the power of fixation, then the covered eye only carried out an excycloduction while the former eye rotated downwards with incycloduction (Case 2).

The conditions producing covering reactions may be summarized thus:

- (1) Changing the illumination of the retina is a frequent cause; the phenome-

†It follows from this that the movements of the eye on covering are not, as Verhoeff (1941) thought, the consequence of an innervational disturbance of the oblique muscles.

‡This large amplitude of vertical fusion has been described by Bielschowsky (1930).

non of Bielschowsky, in which the non-fixing eye can be made to rotate down by putting a dark glass in front of the fixing eye, is a striking example of this.

(2) It has long been known that one of the eyes can be made to turn up by placing in front of it a strong convex lens. This shows that the *contours of the optical image* play a part in the alternating hyperphoria, and that the reflex path therefore runs very probably along the visual cortex of the brain.

(3) In cases with binocular vision, alternating hyperphoria can sometimes be produced by placing in front of the eye a strong prism, whereby diplopia or suppression is caused. The non-fixing eye turns up, whichever eye the prism is placed in front of. This shows that, when the retinal images are of equal quality, the *attention* decides which eye turns up.

(4) The degree of inhibition of the non-fixing eye influences the course of the covering reactions. If there is already total inhibition of the image of one of the eyes, covering can have no further influence on its motility.

Case 3 may help to elucidate the problem, showing the influence of the contours when a strong convex lens is placed before the eye, and the influence of the attention when using a prism. That the Maddox rod caused a hypertropia of very short duration must be explained by the fact that the patient in this examination paid equal attention to the images of both eyes. The absence of the phenomenon of Bielschowsky must be interpreted as follows: as long as there is binocular vision, the eyes are straight; as soon as the binocular vision is interrupted, a total alternating suppression makes its appearance. Because in this special case inhibition is either absent or total, the hypertropia was not affected when a dark glass was placed in front of the fixing eye.

Hypertropia

(1) STRABISMUS SURSO-ADDUCTORIUS.—It is extremely difficult to analyse the bilateral "upshoot" of the eyes in adduction. Nevertheless a few of our patients facilitated our approach to the problem.

Case 4, a boy aged 9, had a squint dating from birth. There was no squinting in the family. In 1948 an advancement of the external rectus was performed on the left eye and a tenotomy of the internal rectus. His visual acuity was 5/5 (+4D sph.) in the right eye, and 5/8 (+5D sph.) in the left eye. The media and fundi were normal. There was torticollis towards the right shoulder on fixing with the right eye; on fixing with the left eye the torticollis was much less pronounced or even absent. There was no rotatory nystagmus, on covering the right eye there was latent nystagmus. The rapid phase of the optokinetic nystagmus in the direction of the occluded eye was practically absent.

There was esotropia with a preference for fixation with the right eye. The angle of squint was 10° with glasses and 15° without glasses.

Position of the eyes with the right eye fixing;

Primary direction of gaze	10° left hyperphoria
Looking to the right	13° left hyperphoria
Looking to the left	3° left hyperphoria
Looking upwards	8° left hyperphoria
Looking downwards	9° left hyperphoria
Tilting head towards right shoulder	15° left hyperphoria
Tilting head towards left shoulder	5° left hyperphoria

On fixing with the left eye there was no vertical deviation in any direction of gaze but, in a second examination, a constant right hypertropia of only 2 to 3°

Occlusion.—Covering of each eye resulted in sursumduction with slight excycloduction.

In this case the only possible diagnosis was hyperfunction of the left inferior oblique. The marked change of the hyperphoria in lateral directions of gaze and the small influence of vertical directions of gaze on the hypertropia is characteristic of a disturbance in the innervation of the oblique muscles. The increase of the hypertropia on tilting the head towards the right shoulder proves the presence of hyperfunction of the inferior oblique, and rules out a paresis of the left superior oblique muscle.

It is an interesting fact that this hyperfunction only appears if the right eye is fixing. This is an important symptom as it shows that the hyperfunction is not based upon a relatively too powerful muscle, but upon a too powerful innervation.

Patients like Case 4 are extremely rare, but in a less evident degree, one may often find that the “upshoot” is strongest in the habitually squinting eye. The disappearance of the strabismus surso-adductorius when the habitually squinting eye takes over the function of fixating, has been found by me in several patients. The influence of head tilting on hyperphoria, so important for the diagnosis of “hyperfunction of the inferior oblique muscle”, is by no means rare.

Case 5, a girl aged 6, had a squint dating from the third month.

Her visual acuity was 5/10 (+2D sph.) in the right eye, and 5/60 (+1D cyl., axis 0°) in the left eye.

The media and fundi were normal. The head was tilted towards the right shoulder and made little nodding movements. There was strong rotatory pendular nystagmus and latent nystagmus.

The position of the eyes revealed a left esotropia of 25°. There was on both sides strong hypertropia of the adducted eye. Tilting the head towards the shoulders and looking upwards and downwards (also in oblique directions of gaze) did not influence the hypertropia. The hypertropia in adduction was however somewhat less pronounced when the adducted eye was fixing, than (*ceteris paribus*) when the abducted eye was fixing.

Occlusion.—Covering had little effect on the position of the eyes; only when a very dark glass was placed in front of the right eye did a slight downward rotation take place in the left eye. In March, 1951, a recession of both internal recti was performed. In October, 1951, the following data were noted.

There was no longer any horizontal squint. The head was still strongly tilted towards the shoulder. The hypertropia in adduction was very slight. The covering reactions were especially distinct, for on being covered the right eye turned upward with excycloduction, and there was incycloduction of the left eye, and horizontal jerky nystagmus to the left, with rotatory component to the left. When the left eye was covered the opposite reactions occurred symmetrically.

We see in this patient the remarkable fact that the strabismus surso-adductorius disappeared after operation of the horizontal squint, and was replaced by alternating hyperphoria. It can hardly be more clearly demonstrated how close is the connection between alternating hyperphoria and hypertropia in lateral directions of gaze.

The survey of the 67 cases in Group I, alternating hyperphoria with strabismus surso-adductorius, leads to the following conclusions:

(1) Compared with the change of the hyperphoria in horizontal direction, the change in vertical direction can be ignored. A slight increase in the hypertropia was noted in five cases when the gaze was turned upwards, and in six cases when it was turned downwards.

(2) In 23 cases a *hyperfunction of the inferior oblique muscle* of the non-fixing eye was considered probable. In making this diagnosis special attention was paid to the influence of head tilting on this hypertropia.

(3) In four cases a *hypofunction of the superior oblique muscle* appeared to be present, either in the fixing eye, or in the non-fixing eye.

(4) In forty cases it was not possible to differentiate further the strabismus surso-adductorius. Here and there, however, an enlargement of the excursion of the eye upwards and nasally was found, so that a *hyperfunction of the inferior oblique muscle* is probable in these cases too, combined possibly with hypofunction of the superior oblique muscles.

(II) STRABISMUS SURSO-ABDUCTORIUS.—In fifteen of the twenty-five cases in this group a more exact differentiation was not possible. In Case 2 (see p.000) and six other patients it was possible from the behaviour of the hypertropia when the head was tilted to diagnose a hyperfunction of the superior oblique muscle of the fixing eye. In three patients hypofunction of one of the two inferior oblique muscles appeared to be more probable.

Nystagmus

Rotatory pendular nystagmus is the most frequent symptom in alternating hyperphoria, followed by unilateral vertical nystagmus which is especially found in severely amblyopia eyes. Horizontal and bilateral vertical nystagmus do not belong to the syndrome. Vertical see-saw nystagmus did not occur in the material here reviewed and is undoubtedly very rare. It is described by Matteucci (1947) in a case of alternating hyperphoria.

When one of the eyes is covered latent nystagmus occurs. This is a horizontal jerk nystagmus of which the rapid phase is directed towards the side of the uncovered eye. Frequently there is a rotatory component of which the rapid phase is also directed to the side of the uncovered eye. The rapid phase is a refixation movement which therefore does not occur if the non-covered eye has no power of fixation. One then sees at times (*i.e.* in cases with eccentric fixation) a conjugated deviation towards the side of the covered eye as an expression of the slow phase of the nystagmus ("latent deviation").

In cases of alternating hyperphoria with exotropia, latent nystagmus occurs less frequently than in cases with esotropia. In such patients what happens to the rotatory nystagmus after covering one of the eyes can be seen more clearly; it changes into a tonic rotatory deviation in the direction of the covered eye, or into a rotatory jerky nystagmus in the direction of the uncovered eye. The latent nystagmus in our patients forms part of the occlusion reactions, and a separate discussion of the conditions under which this nystagmus can

become manifest, may be omitted. Whether all cases of latent nystagmus are based on the same mechanism as in our patients, must be left undecided, but the following particulars are worthy of note:

(1) In 113 cases of alternating hyperphoria, rotatory nystagmus was found 69 times and latent nystagmus 81 times.

(2) In seventy cases of rotatory nystagmus described by Ohm (1938), latent nystagmus was seen eighteen times, and alternating hyperphoria nineteen times.

(3) In thirty cases of latent nystagmus reported by Verhage (1942), there were nine cases of alternating hyperphoria.

Torticollis

Torticollis was present in 26 of our 113 cases of alternating hyperphoria. All these patients had esotropia. In the great majority the head was tilted towards the shoulder on the side of the fixing eye. In view of the great frequency of the alternating hyperphoria, the form of torticollis which is described in connection with these patients is perhaps the most usual type. The usual explanation of ocular torticollis, *i.e.* that it aims at restoring binocular vision by relaxing a paretic muscle with the help of the vestibular reflex of compensatory torsion, is inadequate.

In eleven patients squinting dated from birth and in the remainder squinting appeared at a very youthful age, frequently in the first months of life. The quest for binocular vision is quite excluded as a cause of torticollis in all these cases. Also, in cases of paresis of eye muscles, whereby diplopia is avoided by means of torticollis, hypertropia appears when the head is tilted towards the opposite shoulder. In our cases hypertropia rather decreased when the head was tilted towards the opposite shoulder (Meesmann, 1949). It seems that the torticollis in our cases was caused by a different mechanism from that due to paresis of the eye muscles.

If we do not assume that the torticollis is a survival from a time when the patient had binocular vision, the only possibility remaining is that the torticollis is the consequence of a monocular deviation in the position of the eye, in fact of a deviation in the position of the fixing eye. As the torticollis, in by far the majority of cases, is directed towards the side of the fixing eye, we shall have to assume that it has the function of compensating incyclo-duction of the fixing eye. A more detailed research will have to be undertaken to verify this hypothesis. Especially will it be necessary to pay attention to cyclophoria and to the position of the subjective vertical. Our data, although scanty on this point, already give some indications; in 21 patients the cyclophoria was determined, and we found the cyclophoria negative in eleven, positive in two, and absent in the rest. The subjective vertical of the fixing eye was ascertained sixteen times with no deviation, nineteen times with an inward inclination of the vertical meridian, and only three times with an outward inclination.

These data support our view of the incyclo-duction of the fixing eye.

Discussion

It thus appears that all the changes which can be brought about in the eye position by covering one of the eyes can be reduced to one formula: "sursumduction and excycloduction of the covered eye, deorsumduction and incycloduction of the non-covered eye". If we also take into account the slow phase of the latent nystagmus, the formula reads as follows:

$$\begin{array}{cc} \text{Covered Eye} \left\{ \begin{array}{l} \text{sursumduction} \\ \text{excycloduction} \\ \text{abduction} \end{array} \right. & \text{Uncovered Eye} \left\{ \begin{array}{l} \text{deorsumduction} \\ \text{incycloduction} \\ \text{adduction} \end{array} \right. \end{array}$$

These movements have a coordinated character and have nothing in common with the isolated relaxation or contraction of one or several eye muscles. The direction of gaze has no influence on the trend of the movements and the rotatory component does not increase at the expense of the vertical component when the covered eye is abducted. A disturbance in the innervation of the oblique muscles, such as Verhoeff (1941) assumed in his ingenious explanation of the alternating hyperphoria, is not involved. The movements must have a supranuclear origin.

It is as if the eyes behaved in the case of alternating hyperphoria like the pans of a balance. Ohm (1938, 1949) has already spoken in this connection of an *Augenwaage* (eye-balance). The balance dips always towards the side where the weight of the optical stimuli is greater, either owing to their own peculiar quality or to the attention paid to them.

Great importance is attached to the question whether the innervational disturbance of the alternating hyperphoria is a disturbance in the conjugated binocular innervation or in a—still mainly hypothetical—monocular innervation. There is much that at first sight argues for a binocular conjugated innervational disturbance: the bilateral homonymous torsions, the bilateral nystagmus. If one assumes a disturbance in the conjugated innervation, then, on covering the right eye, the following reactions take place:

Positive vertical divergence, dextrocycloverion, dextroversion.

If, however, we keep in mind that covering reactions are the consequence of a disturbance of the *Augenwaage* (eye-balance), it is just as possible that when the right eye is covered there is simultaneously sursumduction, excycloduction, and abduction in the right eye, and deorsumduction, incycloduction, and adduction in the left eye.

It is our task to find in the physiology and pathology of eye movements points of contact for both possibilities, those of a conjugated and those of a monocular innervational disturbance.

(1) Ohm (1949), who after Bielschowsky (1930) has contributed most to the clinical study of alternating hyperphoria, assumed a connection between the alternating hyperphoria and the vertically disjunctive eye movements which, in animals with lateral position of the eyes, occur when the head is turned round the sagittal axis.

In man, however, disjunctive vertical eye movements are not known to proceed from vestibular reflexes, neither has an influence of optical stimuli on the compensatory positions of the eye ever been described.

(2) The only neurological syndrome that displays any similarity to the alternating hyperphoria is the syndrome of Magendie-Hertwig, the "skew deviation". In patients with a disease in the posterior cranial fossa, sometimes a strong hyperopia occurs combined with horizontal deviation of the eyes towards the side of the eye that has turned upwards. The mechanism of this syndrome is not known. There is no question of any known connection between skew deviation and alternating hyperphoria; the similarity is in all probability quite fortuitous.

(3) Bielschowsky (1930) pointed out the relationship between the vertical fusion movements and the eye movements in the case of alternating hyperphoria. Both eye movements are dependant on optical stimuli; both have the slow gliding character which distinguishes them from movements of gaze. The correspondence does not however go very much farther: the fusion on movement is made in the service of binocular vision and the alternating hyperphoria appears to exist where binocular vision is not present. Bielschowsky thought that the optical stimuli of the right eye in the case of alternating hyperphoria caused a stimulation in the centre for positive vertical divergence and stimuli of the left eye in the centre for negative divergence. Nothing is known of either the existence of these centres or of the manner in which optical stimuli could influence them.

(4) Some investigators have attempted to explain alternating hyperphoria by an innervational disturbance in the monocular eye movements. Generally in such a case a connection is sought with the Bell's phenomenon (Posner, 1944; Piper, 1948), but it seems that Bell's phenomenon is invariably bilateral.

The existence of monocular eye movements is not generally accepted. For most writers Hering's law of innervation has such absolute validity that they pay too little attention to such contradictory facts as the following:

(a) The observation of monocular eye movements in newborn babies by Raehlmann and Witkowsky (1877) was later confirmed by many investigators. Bing Chung Ling (1942) analysed the eye movements of newborn infants by a cinematographical method, and showed that the first step in fixation is frequently monocular.

(b) Keiner (1951) has described monocular optomotor reactions in young patients with esotropia. He often saw an adduction movement of the squinting eye when he let light fall on the temporal half of the retina. When the light fell on the nasal half a corresponding monocular abduction movement was practically never seen. He concluded that convergent squint was caused by a disturbance in monocular optomotor reflexes, whereby the temporal half of the retina dominates the nasal half.

(c) There exist forms of monocular nystagmus which are very probably based on a unilaterally diminished optomotor innervation. This is evident in the case of monocular nystagmus amblyopicus.

I observed a case of monocular horizontal nystagmus with spasmus nutans in which when the non-affected eye was occluded for some weeks the nystagmus was transferred from the eye originally affected to the covered eye. It is scarcely possible to find a clearer proof of the relation between the monocular sensory and motor mechanism.

(d) In the lower vertebrates monocular eye movements are widespread. The

monocular searching movements of the chameleon are the best known, but to a smaller degree monocular optomotor movements are to be found in other reptiles and in birds. Generally monocular and conjugated motility occur alongside each other in the same animal species (Bartels, 1939; Rochon-Duvigneaud, 1933).

It appears that exceptions to Hering's law are not rare and that monocular optomotor eye movements are seen when binocular vision is little developed. We are inclined to look upon the monocular eye movement as a primitive reflex which, through the development of binocular vision, has gradually retired to the background. This point of view is formulated in detail by Zeeman (1943) in his theory of the role of conditioned reflexes in eye movement. This theory differs from that of Chavasse, owing to the place which is given to the monocular optomotor reflexes.

These considerations indicate that there may be a disturbance of the monocular optomotor reflexes in alternating hyperphoria. This is very probable in view of the extreme frequency of disturbances of binocular vision in alternating hyperphoria. We only need then to assume that optical stimulation of one eye in alternating hyperphoria causes an asymmetric increase in tonus of the eye muscles, which results in deorsumduction, cycloduction, and adduction. Under the influence of fixation the deorsumduction will, as a rule, be compensated by a sursumversion. Owing to this the non-fixing covered eye turns up. The adduction (except in cases of eccentric fixation) is compensated by an opposed lateroversion (rapid phase of the latent nystagmus). The rotatory deviation is sometimes compensated by the rapid rotatory phase of latent nystagmus.

The preponderance of the stimuli to deorsumduction and incycloduction leads to hyperfunction of the superior oblique muscles as these take care of both the deorsumduction and the incycloduction. Bilateral hyperfunction of the superior oblique muscle, stronger on the side of the fixing eye, leads to strabismus surso-adductorius, the form of squinting which is seen in a number of the convergent and in practically all of the divergent cases of alternating hyperphoria.

The strabismus surso-adductorius occurring in the majority of convergent cases of alternating hyperphoria is more difficult to explain (Crone, 1952). The inhibition of the non-fixing eye may have some part in this, for the upward rotation in adduction often occurs only when the line of vision of the adducted eye disappears behind the nose bridge and the other eye takes over the fixation. Also strabismus surso-adductorius after operation (whereby owing to the change of position the inhibition mechanism is temporarily interrupted) can change into alternating hyperphoria without hypertropia in adduction*. The smaller number of cases of strabismus surso-adductorius in the older age groups could be explained by diminution of the inhibition which in the course of years makes way for other forms of sensory adaptation.

*The opposite process is described by Piper (1948) as "consolidation" of the alternating hyperphoria to strabismus surso-adductorius.

The asymmetric motor influence of the light stimuli on both eyes causes a bilateral impulse to adduction. We see in this the cause of the esotropia in the majority of our cases. It is possible that in the cases of exotropia another disturbance in the optomotor reflexes is present, whereby optical stimulation results in deorsumduction, incycloduction, and abduction, but it is also possible that the ordinary type of optomotor disturbance is superimposed on exotropia. This last is supported by the fact that the latent nystagmus is never reversed in cases with exotropia (rapid phase in the direction of the covered eye), but is more frequently lacking than in cases of alternating hyperphoria with esotropia.

The theory of alternating hyperphoria sketched out above is actually an extension of the theory of esotropia advanced by Keiner (1951), who considered the motor preponderancy of the temporal half of the retina as one of the causes of esotropia. The motor impulses from the nasal half of the retina thereby suffer a shortage.

Our cases are to be explained by the hypothesis that the motor impulses from the lower nasal quadrant of the retina are deficient. The adduction and deorsumduction under the influence of optical stimuli directly follow from this. Little known, but in fair correspondence with our observations, is the fact that from this lower nasal quadrant stimuli issue also to excycloduction. This is shown by the experiments of Mesker (1953), who studied the optomotor eye torsion which occurs on rotating optical contours in the frontal plane. Also, the observation that cyclovergence occurs as a fusion movement mainly under the influence of vertical contours, proves that on stimulation of the lower nasal quadrant the motor impulses to excycloduction are stronger than those to incycloduction.

That the lower nasal quadrant of the retina is deficient in the field of motility may indicate the relative unimportance of optical stimuli from above and from the side compared with those from the front and from below. It is not to be wondered at then if the asymmetry of the optomotor reflexes, which is most clearly evident in alternating hyperphoria, is widely spread in a latent form. This appears from the hypertropia and divergence shown by many blind eyes. The results of Marlow's well-known "prolonged occlusion test" also support this idea. In this test, which was originally intended to make latent heterophoria manifest, a hypertropia of the covered eye very frequently occurs, no matter whether the right or the left eye is covered (Abraham, 1931). Also without prolonged occlusion one frequently finds a hyperphoria which has a different degree according as one places the Maddox rod in front of the one or the other eye. When no paresis is present this phenomenon must be explained by a dissociated element in the hyperphoria.

The aim of this study is to contribute to the problem of squint. The theories and hypotheses advanced raise questions which cannot yet be answered. It is desirable that more attention should be paid to monocular eye movements

and that assumptions regarding them should be tested by accurate experiments. An extensive field of research lies open in this direction.

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