INTRODUCTION

This is a contribution to the study of vertical defects in the positions of the eyes. It includes a partial survey of the literature and an analysis of a series of 402 patients with horizontal and vertical defects of the concomitant and paralytic types. Methods of investigation, including two that are described for the first time, are discussed in full, for without accurate observations satisfactory treatment is unlikely. The value of the “division” of diplopia is stressed.

There are many types of ocular vertical deviations. Their recognition is not always easy nor is their interpretation always simple. They must be studied in near and distant vision as well as when the gaze is directed obliquely. Any deviation may depend on the following:

1. The position of ocular rest.
2. The degree of fusion permitting true or alternating hyperphoria.
3. The presence of a paralysed muscle and its stage of recovery.
4. Contraction of antagonist and synergist and inhibitional paresis.
5. The fascial check ligaments restricting ocular rotation.

These will now be considered at length.

Position of Ocular Rest.—A vertical angle gamma is a very rare finding. It occurs when the visual line connecting the fovea passes above or below the geometrical axis. This is revealed by a study of the corneal reflexes.

Many observers have been tempted to seek the so-called position of rest by withdrawing the bond of fusion. The latter may be strong enough to correct a faulty anatomical setting or muscular imbalance. Marlow made a thorough study after prolonged occlusion. He claimed that the Maddox-rod test revealed only a portion of the heterophoria present. Marlow (1924), in a series of 700 cases in which he carried out occlusion. on an average for one week, found that hyperphoria with lateral deviation was present in 79 per cent. and without latent deviation in 5.5 per cent. In his more recent work 1938, he considered that his prolonged-occlusion test was inclined to produce artefacts rather than reveal a true position of rest.

Bielschowsky found that orthophoria did not return for fifteen to thirty minutes after the brief wearing of a prism. Young (1926) and Swab (1930), found that prisms prescribed after occlusion tests were often not tolerated. Abraham (1931), considered that Marlow’s test was “a subjective test for demonstrating Bell’s phenomenon” rather than a test for latent heterophoria. Criland suggested that the hyperphoria found by the test is a revelation of the protected position of the eye as found in sleep.
THE FOUR POSITIONS OF REST.

Cridland (1941), described
1. the anatomical position of rest,
2. the physiological position of rest,
3. the fusion-free position of rest and
4. the "functional primary direction" or binocular fixation at infinity.

The first position depends purely on anatomical and non-nervous physiological factors. It is one of divergence of about 10° associated usually with about 5° of sursumvergence. This occurs only in coma, deep anaesthesia and death. There is no rest during waking hours. "Only when both eyes are cut off from every source of stimulation do they assume a position of rest that is absolute. No reflex action, no reflex muscular tonus then disturbs the anatomical picture." Chavasse (1939). The factors determining this position include the shape and the size of the orbit and its relation to its fellow, the size and disposition of the orbital contents, the muscular insertions, the elasticity of, and the tension in, the various structures contained in the orbit, eyelids and, indirectly, the skin of the face. Cridland (1941). Asymmetry of any of these factors is common and such asymmetry is one factor in explaining the different heterophoria found in the two eyes.

If to this position is added the effect of minimal, balanced tonus of all the extra-ocular muscles acting together the second position is reached. This is a position of rest, which is nearly incapable of measurement and only potentially capable of realisation. Cridland. If to this is added the effects of the postural reflexes, viz., those that direct the eye in relation to the body and not to its fellow, and of the fixation reflexes then the third or fusion-free position is obtained. The eyes tend to reveal this position in the tests for heterophoria. The varying measurements obtained by these tests depend largely on their efficacy in eliminating fusion or on "the effectiveness of dissimilar retinal images in holding the eyes straight as compared with" that of similar images. Adler, (1945).

Many observations of interest have been made on the distance esophoria that follows alcohol excess, Colson (1940), and anoxia, Velhagen and Adler. The results are due not simply to the removal of fusion-control but also to some influence on the convergence centre.

The fourth position is one of visual parallelism and in it the influence of fusion is felt.

"Basic heterophoria is the latent tendency of the eyes in vision at infinity to deviate from the functional primary direction in the
presence of actual or effective emmetropia." Cridland. Most writers agree that a degree of heterophoria is the rule, but few would agree with Marlow's early conclusions.

Posner (1944), considered there was a characteristic "position of rest" for each state of activity of the central nervous system. When the cerebral cortex is inhibited, as in sleep, the eyes assume the position of divergence and upward rotation. Fixation, convergence and fusion influence the tonus of the muscular apparatus, and it is wrong to consider any of the resulting positions to the exclusion of the others as the position of rest. The fusion-free position is the one that we usually meet clinically. Fixation alone exercises its influence then. "Fixation is maintained reflexly as long as visual stimuli are transmitted to the brain, but its reflex arc probably passes out subcortically. It is only when a change of fixation is involved that the cortical fixation comes into play."

The absence of the cortical control of the cranial nerves was shown by Gordon Holmes (1938) in patients with pseudo-bulbar palsy. Such patients cannot shift fixation from one object to another voluntarily, yet their eyes will follow a slowly moving object or will maintain fixation when the head is rotated passively. The eyes are in a state of obligatory reflex inhibition. "An inhibitory stimulus from the cortex is required to suspend the prevailing fixation long enough to allow it to shift in response to a visual stimulus impinging on a peripheral portion of the retina."

THE CONTROL OF OCULAR MUSCLE-TONE.

Unfortunately, little is known of the complex co-ordination that permits the smoothness of the ocular movements as they respond to a reflex or volition. "The great co-ordinating centre for smoothing out muscular activity in time and extent is the cerebellum." The vestibular division of the eighth nerve has much influence in this way on ocular movements. Adler (1945). "The ocular muscles are kept under a constant changing state of tone through the vestibular nuclei by impulses coming in from the neck muscles and from the otolith apparatus in the labyrinths." These may be separated as follows:—

(1) Changes in position of the head in respect of gravity produce static reflexes which lead to appropriate ocular postures and movements.

Tilting of the head produces stretch reflexes in the neck muscles and proprioceptive impulses that lead to compensatory ocular movement. More important in man are the impulses that come from the otolith apparatus to increase tone in the right superior
and decrease tone in the right inferior muscles, for example, when the head is tilted to the right. The resulting torsion maintains the vertical position of the vertical meridians of the eyes.

**ILLUSTRATION 1.**

The Effects of Head Tilting.

The head is tilted to the right and the oblique muscles are producing torsion in response to impulses from the oto-lith apparatus. Only the impulses leading to contraction are shown, +.
(2) When the head is moved through space the movement of the fluid in the semicircular canals that ensues leads to a lateral or vertical conjugate deviation. The slow component—the essential part—of nystagmus is due to tonic impulses from the labyrinths. The direction of the quick component gives the name to the nystagmus.

The following additional influences on tonus of ocular muscles must be considered.

(3) Sensory impulses. The posterior longitudinal bundle carries impulses to the motor nuclei when sudden noise or pain stimulates the cochlear cells or the trigeminal nuclei.

(4) Visual impulses. There is no doubt concerning the influence of visual impulses on ocular reflex activity. The involuntary fixation reflex provides for continuation of macular fixation in case the object moves. A fusion component of this reflex ensures that single and even stereoscopic vision is obtained if images are not bi-macular. On to this involuntary fusion reflex is built voluntary control which is called into action when diplopia occurs. This control can be developed by practice.

(5) The influence of proprioceptive sensation. This, and even its very existence, is uncertain. Adler (1945). The subject is aware of the position of the eyes after a willed but not after a reflex or an enforced deviation. In nystagmus, except in a minority of congenital cases, the patient is conscious of the apparent movement of the room, but not, as a rule, of the movement of his eyes. It is a sense akin to stereognosis rather than to proprioception that explains the sensation of ocular movement that occasionally follows stimulation in an area anterior to the Rolandi fissure. Penfield and Erickson (1941).

Posner regarded divergence-excess as an anomaly of the postural tonus of the extrinsic muscular apparatus, in an atavistic sense. Irvine (1936) showed that the proprioceptive impulses from the ocular muscles are inadequate to account for the delicate balance of tone found in these muscles. "They are supplemented by visual impulses originating in an area of the retina immediately surrounding the fovea. In addition, the presence of grape-like nerve endings such as those found in the muscles of cold-blooded reptiles and amphibians, in the extra-ocular muscles and in no other mammalian skeletal muscles, suggests the retention of a characteristic of involuntary muscle. These muscles respond to choline and nicotine as do the voluntary sub-mammalian or mammalian muscle before it receives, or after it is deprived of, its nerve supply." Duke-Elder (1932).

Duction may be carried out by the fibres resembling smooth muscle. The relative slowness, the limited range and persistence
of duction are characteristic of such muscle. Verhoeff. It is an involuntary process which can be greatly developed in power yet the exercise of which, strangely enough, causes obvious strain.

THE CONTROL OF OCULAR MOVEMENTS.

The cortex concerned with oculomotor activities is widespread. Yet probably no one area is completely in control or solely devoted to voluntary activity. Gordon Holmes (1938) described the "encephalization" of oculomotor control in man. This is the transfer of primary visual reactions to the cortex until they can be evoked through it only and not from lower levels as they can lower in the scale. These reactions include the reflex direction of the eyes to light, the maintenance of fixation, fusion and accommodation.

After this evolutionary transfer and that of visual perception, associational areas appeared around the occipital visual area, which permitted the elaboration of the faculties of spatial perception, discrimination and recognition. At a later stage, as the influence of higher functions, such as attention and discrimination was felt, the frontal cortex assumed control. This control included not simply the response to volition but also a power to veto the occipital reflexes if they were considered inimical. The forebrain is not concerned with "labyrinthine and other proprioceptive influences." They are effected by a subcortical mechanism.

The afferent paths for and probably the cortex associated with the visual reflexes just referred to are shared by them and by visual perception. The efferent reflex path, however, is distinct and takes a dorsal course through the pulvinar. A lesion of this path may cause a failure of fusion as well as an inability to maintain fixation, particularly if the patient or the object is in motion. Such a state must be contrasted with another dissociation in which voluntary movement is deficient, but fixation is excessive and persistent. This has resulted from bilateral injuries to the frontal oculomotor centres, or to their projection fibres within or caudal to the internal capsule.

Though stimulation of the posterior part of the middle frontal gyrus produces conjugate deviation to the opposite side, yet destruction of this area does not produce a persistent palsy except perhaps in response to volition (Tilling and Lutz, quoted by G. Holmes, 1938). Bilateral representation of oculomotor activity is supported but not proved by this finding. It is said that conjugate deviation upwards follows stimulation below and deviation downwards follows stimulation above this frontal centre (Wolff).
VARIETIES OF BINOCULAR VERTICAL MOVEMENTS.

At the disposal of each eye there are two pairs of vertically acting muscles. Each pair consists of a rectus and an oblique muscle which, working together, permit supra- and infra-version without a simultaneous torsion. There are four ways in which these muscles combine.

1. Parallel vertical movements.

2. Opposite vertical movements to produce a positive or a negative vertical divergence. Bielschowsky, August, 1938. Pascal (1943) suggested "positive vertiduction" for right supra- and left infra-duction and "negative vertiduction" for left supra- and right infra-duction.

3. The corrective movement when head-tilting towards one shoulder causes a parallel rotary movement towards the opposite side. If the head is tilted towards the left the inferior muscles of the right eye and the superior muscles of the left eye contract. If these antagonists are normal, torsion to the side will occur but no vertical deviation.

4. A rotary movement in opposite directions, that is, intorsion and extorsion of the vertical meridians.

The following are significant features of vertical movements:

1. Co-ordinated upward movement has been claimed as the only conjugate ocular movement present at birth.

2. An upward rotation of fifteen degrees occurs during blinking. This appears soon after the sixth month of life.

3. An upward rotation occurs under normal conditions when the eyes are firmly closed. Of this the individual is unaware. Bramwell, (1928). If the eyes are forcibly closed a similar movement occurs. Bell's phenomenon. This is revealed when the lids fail to close in peripheral facial paralysis.

4. In response to certain emotions a characteristic ocular movement may occur. For example, (a) upturned eyes are part of the expression of devotion and sometimes of those who attempt to concentrate or recall some memory.

(b) Disdain or disapproval may be expressed by "casting" the eyes downwards. It is interesting to recall the origin of the title "patheticus" for the superior oblique. It was considered to be the muscle that rotated the eye up and in and so added pathos to the expression. In the light of its true action, however, it is rather an expresser of disgust or contempt. Wolff, (1940).
Downward rotation of the right eye

Inability of the right eye to rotate above the midline.

Except on attempting to close lids against resistance.

Such a finding proves the integrity of the nuclei for elevation of the right eye but interference with cortical impulses to them.

**Illustration 2.**

Paralysis of the right superior rectus with retention of its response to resisted closure of the lids. Bell's phenomenon.
In sleep the eyes rotate up and outwards. This occurs in general anaesthesia and may be a complication in ophthalmic surgery. Often, too, under a local anaesthetic, an inability on the part of an old cataract patient to look down has caused the surgeon considerable worry. This inability appears to increase with effort and nervous tension.

DISTURBANCES OR DEVIATIONS OF THESE MOVEMENTS.

A. THE INFLUENCE OF FUSION. The Concomitant Group.

The vertical range of fusion is relatively small and so a slight error does not remain latent but becomes a hypertropia. Vertical fusion, unlike the normally strong fusion-control in convergence, is neither well-controlled nor in constant use. It is known from experiments with prisms that limited unilateral vertical movements do occur to maintain fusion. Verhoeff wrote that the duction mechanism is the only known function that could prevent vertical deviation. By a conditioning process, he thought that voluntary control over sursumduction could possibly be acquired.

Normally we observe unilateral vertical deviation when the strength of a rotating prism, held base up or down before one eye, is increased. Single vision is maintained by continued stimulation of corresponding retinal points as the eye behind the prism rotates up or down. Such movements are not due to unilateral muscular innervation. Opposite innervation goes to each eye and is neutralised in one by determination to fix and is effective in rotating the other eye with double force.

In this concomitant group there are the manifest squints—hypertropia, and the latest type, hyperphoria. In both there is practically the same degrees of deviation in all directions of gaze. As no muscle is chiefly affected the double images show no obliquity. Neither does head tilting affect the deviation. Fatigue and illness have been claimed to permit the latent to become the obvious defect.

Cridland (1941) wrote recently that the heterophoria test should be repeated until two successive measurements were equal. Bielschowsky (Nov., 1938) held that the same test should be repeated about six times at short intervals until a constant vertical deviation was found, or that at least fifteen minutes should elapse before the contrary kind of fusion movement be tested. It is essential to cover the eye behind the Maddox rod and then ascertain the apparent position of the streak on uncovering. Verhoeff (1939). Many of the tests employed are apt to measure tendencies for the eyes to deviate with respect to each other in the absence
instead of in the presence of binocular fixation. Verhoeff described a test for the former—"presumptive heterophoria"—using stereopsis as an indicator.

Hypertropia may be associated with a convergent or a divergent squint. Use of the synoptophore or other methods of investigating the latter may reveal an anomalous retinal correspondence. They sometimes reveal an apparent revulsion to vertical fusion. With the arms of the instrument locked, on horizontal rotation the height difference may appear to vary constantly. This peculiarity may be confused with suppression.

The incidence of heterophoria is not greater amongst patients with so-called functional neuroses or epilepsy than amongst perfectly healthy persons. The symptoms arising from heterophoria are much commoner in the former group for the effort to maintain binocular single vision is a greater strain if the patient has a depleted nervous system.

In my series of over three hundred patients with defects of the vertical muscles and movements not one instance of true hypertropia or true hyperphoria was found.

Patients with errors of less than one degree were excluded. A condition resembling true hyperphoria was presented by a woman

Illustration 3.

Projection Chart of R.S.W. Almost concomitant hyperphoria but probably right trochlear palsy.
(Mrs. T.) aged 45 years, with two dioptres of left hyperphoria at reading distance, but none in the distance. As the error was greatest during adduction in elevation a lack of balance in the oblique muscles may have been the cause. Two other patients were almost truly hyperphoric, (1) R.S.W., aged 53 years, on the synoptophore with the left eye at $-10^\circ$ and at $0^\circ$ he had eight dioptres and at $+10^\circ$ nine dioptres of right hyperphoria. The results of his projection test suggest a weakness of the right superior oblique and of the left inferior rectus. Though his head was carried slightly towards the left yet tilting in the opposite direction made little difference to his diplopia. The torticollis suggested that a partial right trochlear palsy was the underlying cause of the apparent "true" hyperphoria. (2) G.D.P., aged 52 years, had right hyperphoria of 4 5 dioptres near and of 1 dioptre in the distance. His projection test at two metres revealed so little separation of the images that no regular variation in the different directions of gaze was found. No diplopia was elicited with red and green glasses at a closer range. His condition is therefore one of almost true hyperphoria.

A certain variation in heterophoria may be considered physiological. Friedenwald (1936). This may be called anisophoria.

When a patient looks at an object situated in a diagonal direction of gaze the adducted eye will make a smaller rotation than the abducted. If they fail to do so a slight heterophoria exists. Friedenwald described two types of pathological anisophoria. In one the anomaly was due to a weakness or an overaction of one of the vertical ocular muscles. The other type followed unequal peripheral prismatic action from lenses prescribed for the correction of anisometropia.

Friedenwald discussed the relative merits and limitations of three methods of correcting these defects.

Note.—On the completion of this manuscript I found White’s paper "Hyperphoria," 1932. In it he wrote: "In my experience, at least 98 per cent. of cases showing a difference in level of the eyes are due to a weakness of one or more of the elevators or depressors, with usually some secondary contraction of the direct antagonist and secondary deviation of the associate antagonist.” This certainly is my experience.

The existence of a difference in hyperphoria for distance and near was explained by White as follows:—In the distance, in frontal gaze, the vertical recti are the main elevators or depressors. At reading distance the obliques have the greater power. So a paresis of a rectus muscle leads to a hyperphoria that is greater for distance and less for near. The reverse state accompanies a paresis of an oblique muscle. This may not be so if a marked secondary deviation is present.
B.—CONFLICT OF FIXATION, FUSION AND ABNORMAL STIMULI. DISSOCIATED DEVIATION.

1. Alternating hyperphoria.
2. Vertical dissociation with unilateral amblyopia.
3. Vertical dissociation combined with other deviations.
4. Periodic vertical squint.

In the last condition one eye turns up or out when the patient is fatigued or diverted and returns to normal fixation when an object attracts the attention of the other eye. "The unilateral movement is produced by bilateral and equal motor innervations, one driving both eyes in the same parallel direction, while the other drives the eyes in opposite direction." Bielschowsky (1945).

Lack of fusion power is the principal cause of this condition. If the power of fusion is weakened the patient loses the power of rotating the eye to the normal position again. Fusion, Bielschowsky (1945), plays little or no part in causing the upward rotation in unilateral vertical dissociated movements. It evidently does, however, in pure alternating hyperphoria.

1. ALTERNATING HYPERPHORIA.

The characteristics of this condition are:

(1) The rotation of either eye upwards when occluded. Then as the fixing eye is occluded it makes a slight downward movement and then rotates upwards as soon as its fellow resumes fixation. Fatigue or abstraction may be sufficient to produce the upward rotation.

(2) A variation in the rotation that is not found in "true hyperphoria." Peculiar slow irregular vertical movements of the upward eye are often seen. The rotation may vary from time to time and may occasionally be replaced by a movement downwards. These oscillations are involuntary and any voluntary motor impulse will restore binocular fixation and movement.

(3) Failure, as in "true hyperphoria," of change in direction of gaze to alter upward deviation as occurs in overaction of the inferior oblique and paretic cases.

Change of fixation from one eye to the other replaces an upward rotation of one eye by a downward rotation of the other in paretic or true concomitant vertical deviation. This is not so in overaction of the inferior oblique or alternating hyperphoria.

Change of gaze in a vertical direction has no influence on the vertical deviation in overaction of the inferior oblique.

Change of gaze in a horizontal direction alters the vertical deviation in overaction of the inferior oblique but not that in alternating hyperphoria.
Alternating hyperphoria.
(1) Visual axes parallel, (2) left eye and (3) right eye elevated after occlusion.

**ILLUSTRATION 4.**

Unilateral dissociation. The right eye rotates upwards on fatigue or on right occlusion. (1) shows normal forward gaze, (2) shows rotation upwards after occlusion, (3) shows slight weakness of right inferior rectus, and (4) normal action of left inferior rectus.

**ILLUSTRATION 6.**
ILLUSTRATION 5.

Bielschowsky's phenomenon. (1) Eyes normally directed. (2) Upward rotation of left on occlusion. (3) & (4) Dense portion of glass wedge before fixing eye leads to downward rotation of left which lessens if wedge is slightly raised (4)
(4) The field of fixation is normal in extent.

(5) Bielschowsky's phenomenon. Cords (1930). If a dark glass wedge is moved in front of the fixing eye so that the light is gradually dimmed, the previously occluded upward-rotated eye will move downward below the horizontal plane, sometimes in almost exact proportion to the darkening. It will rotate up again as the darkening is lessened. The fixing eye maintains its position unaltered during this movement.

(6) These movements are independent of the will as long as the fixation position and the elimination of the compulsion to fuse remain unchanged. Every voluntary impulse or a return of fusion, if such is well developed, will restore the former state of binocular central fixation.

(7) The presence of ability to overcome vertical prisms of 10 dioptres and more in either direction by corresponding vertical divergence innervations. This is approximately three times the strength of prisms that normally can be overcome. In concomitant hyperphoria the amplitude of the power to overcome prisms held one way will be increased, and may be absent when they are held the opposite way.

(8) Alternating hyperphoria may be found in any type of strabismus, whether it be alternating or uniocular, convergent or divergent, or associated with normal or anomalous correspondence, or with normal or defective sight.

Sometimes the upward rotation is not equal for each eye. Neither is it always constant for one eye—the degree varying as a "result of the intermittent nature of the stimuli from the vertical divergence centres." Bielschowsky.

Variations from the pure type of alternating hyperphoria are sometimes found. Bielschowsky (1930-31) described patients whose upward turned eye would make little or no downward movement when a red glass was placed before the fixing eye. In these, however, a change in fixation from one eye to the other would reverse the eye that was rotated upwards.

Occasionally only one eye when covered turns upwards, the other rotating in- or outwards when it is covered. Sometimes when the upward eye is covered its fellow rotates slightly downwards and then laterally. In isolated cases of constant squint the covered eye rotates upwards and displays some torsion.

2. Vertical Dissociation with Unilateral Amblyopia.

Not uncommonly while a good eye maintains fixation of a light its visually defective fellow will show a series of irregular vertical movements. These can be arrested and replaced by a downward rotation if a dark glass is put before the fixing eye.
This again is Bielschowsky's phenomenon. This movement on an average is ten degrees. If occlusion is continued the amblyopic eye rotates upwards in a jerky fashion. The amblyopic eye generally moves upwards if it alone is covered.

3. Vertical Dissociation with Squint, Concomitant and Paralytic.

The extent and direction of the vertical deviation may be influenced to a certain degree by occlusion of the squinting eye or by darkening the fixing eye. Bielschowsky showed that the downward movement of the upward-turned squinting eye took place even when the fixing eye was darkened by a coloured glass and though the squinting eye remained uncovered.

Various combinations of horizontal defects with variable vertical deviations may occur. They may be difficult to recognise unless each eye is studied separately in the oblique position of gaze. Bielschowsky described a type of paralysis of the right trochlear nerve in which the right eye was higher when behind a red glass because the positive vertical divergence increased as the dissociated component was added to the paretic component. The left eye behind the red glass failed to rotate up or even displayed overcompensation and rotated slightly downwards.

The element of dissociation may pass unobserved unless, for example, it is noticed that a dark glass before the fixing eye produces a downward rotation of its fellow. On removing this the squinting eye will regain its horizontal deviation. Covering the squinting eye may cause it to rotate upward, and it may show the irregular vertical oscillations characteristic of alternating hyperphoria. To follow the movements it is essential to watch the movements of the eye behind the cover or the dark glass.

An alternating upward deviation in association with an exophoria or divergent squint occasionally occurs. It must not be forgotten that any exophoria tends to increase during upward rotation.

The explanation of the atypical conditions is more complicated than that of pure alternating hyperphoria. Fusion can have nothing to do with many of these unilateral movements, for many are associated with amblyopia. The affected eye may move up and out or down, but it never fixes the object fixed by the other eye.

The dissociation may not be easy to demonstrate, but it may be sufficient to hinder diagnosis in the red and green test. Various forms, such as the following, may exist:

1. A dark glass before the fixing eye may send the squinting eye downwards. It will return to its former squinting position
on removal of the dark glass. If one occludes the squinting eye it rotates up and, after a time, shows vertical oscillations.

(2) Place a dark glass before an eye that has an upward deviation. It may rotate up further, but if its fellow is treated similarly the deviation lessens. Or one may cover the fixing eye and the other may rotate downwards. Now cover the second eye and it moves slowly upwards, but no movement of the former may occur. Uncover the second and cover the former and an extra movement of the second occurs. If the formerly fixing eye is watched behind the cover it will be seen to rotate downwards with its fellow and quickly recover.

(3) One eye when occluded may rotate upwards and soon show irregular oscillations while the other eye, when occluded, remains stationary.

These are varied forms of dissociation, but in all the characteristic of concomitant and paretic deviations is absent, viz., that in changing fixation from one eye to the other an upward is replaced by a downward rotation.

Comparison with Overaction of an Inferior Oblique Muscle.

Change of fixation from one eye to the other replaces an upward rotation of one eye by a downward rotation of the other in paretic and in true concomitant vertical deviations. This is usually so in overaction of the inferior oblique muscle but not in alternating hyperphoria. Change of direction of gaze leads to alteration of a paretic deviation. If the change is horizontal the deviation in overaction will alter, but not that in true alternating hyperphoria. If the change is vertical there is no alteration in overaction, Bielschowsky (1931), though frequently the upward rotation appears to be more obvious when looking up.

It must be remembered that both overaction of an inferior oblique muscle and alternating hyperphoria may be found in the same patient.

The Frequency of Dissociated Defects.

Bielschowsky (October, 1938) found heterophoria in over 80 per cent. of normal people and in a similar proportion of those with eye strain or nervous troubles. In addition over 41 per cent. of these showed apparently dissociated vertical deviations.

The pure type of alternating hyperphoria is not frequently detected. This is partly because it is not self-revealing. When binocular vision is normal this defect is not always easy to detect. In 1930 Bielschowsky had found well over a dozen cases of this type. He frequently found examples of dissociation in patients
with disturbed binocular vision. In a series of 289 patients without binocular vision 41 per cent. showed dissociation in the form of unilateral vertical movements that varied from time to time in degree and even in direction.

EXPLANATIONS OF ALTERNATING HYPERPHORIA.

It must be asked, is the upward turn associated with the tendency for eyes to rotate upwards when closed? Is there any difference in the anatomy of the upward rotators and of the downward rotators that gives the former a mechanical advantage? The inconstancy of the error is incompatible with the conception that it is based on an imbalance between these two groups. The deviation of the upward eye may be transferred into a downward rotation. This further undermines this conception.

Bielschowsky’s explanations, (1930-1945). Bielschowsky at first thought that unilateral stimulation to upward rotation occurred and that the condition was similar to unilateral vertical nystagmus. But this theory would not explain the variants or the fact that not only covering either eye leads to its upward rotation, but also that illumination of the other leads to its downward rotation.

At one time (1931) he considered that an inequality in the stimulation of each retina produced the disjunctive vertical deviation. He (1945) concluded by assuming an abnormal excitability of the subcortical vertical divergence centres. The cause was obscure and the condition was usually congenital though sometimes acquired. Unfortunately there is neither anatomical nor physiological evidence that centres for opposite vertical movements exist. Bielschowsky used his clinical observations of dissociation to support the conception of such centres.

The alternating upward rotation and the one-sided variable vertical movements "must be attributed to intermittent and alternating innervations for positive and negative vertical divergence, which are independent of the will and fluctuate in intensity." These go to both eyes, but only one rotates because the inhibiting influence of the intention to fix neutralises the involuntary stimulus to vertical rotation. Normally the fusion mechanism is stronger than the anomalous innervations arising from the vertical divergence centres. Fatigue or a visual defect may permit the underlying abnormal vertical divergence to overcome the control of fusion. Then alternating hyperphoria or, if unilateral amblyopia be present, irregular upward and downward movements of the amblyopic eye occur. See "Periodic vertical squint."

Posner’s Explanation.—Posner (1944) preferred to explain this hyperphoria "as a synthesis of the primitive monocular tonus-regulators and the higher binocular innervation."
In the tendency of blind eyes to diverge we may have a return to the lateral position of more primitive eyes. This position was once a survival factor for panoramic vision made for safety. Have we in alternating upward rotation a persistence of a primitive protective rotation such as is found on closing of the eyes in sleep? If so, this upward tendency is potential in us all and may become marked if the general tonus of the neuro-muscular system is increased. Posner considered that fixation might increase tonus and that one of the following conditions might reveal the upward rotation—unilateral occlusion, amblyopia or blindness, inadequate fusion from either a weakness of fusion itself or a relative inadequacy due to anisometropia and heterophoria. He wrote that the tonus of eye muscles depended on

(a) the voluntary nerve supply which is invariably bilateral and symmetrical, and on

(b) the reflex postural mechanism that varies with fixation, illumination and attention.

These factors may increase the tonus of muscles and this may produce an upward turn, which after all is a primitive tendency, especially in those people with a relatively weak binocular linkage. When higher visual attainments such as fusion, convergence and accommodation intrude the more primitive upward divergence is inhibited.

Chavasse's explanation.—Chavasse described the manner in which the reflexes can become so interwoven and the reciprocal and synergic relationships of the two eyes so exact that stimulation of one retina alone can achieve the precise binocular posture which, during the conditioning of the reflexes, was achieved only by stimulation of both.

In alternating hyperphoria, however, if one retina is made less dominant by artificial means the eye will move up and out. If the embarrassment is transferred to the other eye it deviates and its fellow resumes fixation. When the dominance has not asserted itself by fixation of the object this eye may slowly rotate down to or even below the object.

"In cases in which there are no complications the direction of gaze has no influence on the anomaly under discussion."Bielschowsky, August, 1938. Chavasse, however, wrote: "In alternating hyperphoria the elevation is greatest when the gaze is led so as to abduct the non-fixing eye."(Chavasse, p. 218.) Cords stated that any variation in elevation in lateral rotation was rare.

Verhoeff’s Explanation.—Verhoeff (1941) preferred the term "occlusion hypertropia." He objected to Bielschowsky's explanations because the centres for vertical divergence were unproven and the intermittent excitations unexplained. Mainly
from a study of nystagmus which may exist only when one, and not the other eye, alone is covered, did he conclude that two monocular conjugate mechanisms exist. He believed also that a binocular mechanism was present because in certain cases of strabismus when either eye alone is covered, there is horizontal conjugate nystagmus, whereas, when neither is covered, there is either a different type of or no nystagmus. Further proof of these three mechanisms is forthcoming from a study of occlusion hypertropia with strabismus. In many of these "the deviation of the eyes with respect to each other markedly differs according to whether either alone or neither eye is covered. Insufficiency of either one or both mechanisms may explain occlusion hypertropia." This conjugate insufficiency usually concerns the superior oblique, but sometimes the inferior rectus or both. It is usually associated with other motor defects, especially "esophoria." Often overaction of one or both inferior oblique muscles due to nuclear hypoplasia is present. The late phylogenetic acquirement of the anatomical peculiarities of the trochlear nerve and its muscle make the development of defects in its conjugate pathways likely. In most of his patients Verhoeff found extorsion with the upward deviation. This increased when the eye was conjugately turned towards the nose and decreased when the fixing eye was elevated.

The amount of upward deviation differs greatly in different cases. This suggested to Verhoeff a variable degree of fourth nuclear hypoplasia with overaction of the inferior oblique muscles.

White's Explanation.—He (1932) wrote that all his cases of alternating hyperphoria were due to paresis, more or less marked, of the same muscle in each eye. They were affected in the following order:—in a large majority both superior recti; next, both inferior recti and much less frequently, both inferior oblique muscles. He had not observed paralysis of both superior oblique muscles. He considered that if both muscles were equally paretic the right and left hyperphoria would be equal. If the right superior rectus were more affected the left hyperphoria would be the greater. Occasionally "the left hyperphoria predominates and there is not right hyperphoria until the eyes are directed up and left, when the left superior rectus is found to be paretic, also with the accompanying increase in right hyperphoria in this field." White (1933) held that the hyperphoria was due to a paralysis of the opposite superior rectus.

Burian (1944) described in detail a patient with concomitant convergent strabismus, and overaction of both inferior oblique muscles and dissociated vertical divergence. There was no sign of weakness of any extra-ocular muscle. Under cover each eye turned inwards and upwards, the left turning up farther than the right. This patient had a moderate degree of stereopsis.
SUMMARY.

The differences expressed here mean either that there is as yet much we do not know about this condition, or that a similar idea is expressed in the various views. Probably there are "centres" or controlling spheres, for uni- and binocular vertical movements, not necessarily well defined structurally but adequate in function, which can, under certain conditions, permit unilateral vertical movements after occlusion or in nystagmus.

The more one studies ocular deviations the more one realises the need to watch for spastic components, overfunction or other results of excitation of the controlling centres, in combination with defects in the position of rest.

C.—INFLUENCE OF ORGANIC DISEASE.

1. Paralysis of vertical rotation and supranuclear lesions.
2. Oculomotor syndromes.
4. Misdirection of regenerating fibres.
5. Cyclic oculomotor paralysis.
6. Anomalies of synergic lid and eye movements.
   Bell's phenomenon.
   Retraction syndrome.
7. Palsies of ocular vertical muscles.
   Bielschowsky, (May, 1935), wrote "I have never seen as a true functional disturbance paralysis of individual eye muscles or unilateral ophthalmoplegia."

1. PARALYSIS OF VERTICAL ROTATION.

As a result of the experimental work of Sherrington, Russell and others, it is reasonable to consider that stimulation of the upper or of the lower part of a region in the second frontal gyrus will produce conjugate downward or upward rotation of the eyes. Paralyses and spasms may result from destructive or irritative lesions of the opposite frontal lobe. Holmes (1921). Stimulation of the frontal centres in the lower mammals may produce ocular torsion. Collins and Spiegel (1938).

Movements to the opposite side follow stimulation of the posterior third of the second frontal gyrus and of the psychic sensory area of the occipital lobe. Responses from the latter are weaker and more transient than those from the former. They may be accompanied by contraction of the pupils, possibly by disordered accommodation and by visual hallucinations. The vertical movements in animal experiments do not appear on stimulation until the area controlling lateral movements has been destroyed.
The fibres from the voluntary eye motor area leave the cortex and with the pyramidal fibres pass in the corona radiata to the knee of the internal capsule. They decussate on their way to the various nuclei, all of which, except the lower portion of the seventh, receive fibres from both hemispheres. According to Collier, (1927), the fibres for the vertical movements cross in the posterior commissure and those for lateral deviation lie laterally and decussate lower down. He found that a lesion affecting one half of this commissure abolished upward and downward movements of both eyes. The fibres from the occipital lobe arise around the area striata and lie medial to the optic radiations as they pass through the pulvinar and the anterior brachium to reach the tectum of the midbrain. Holmes (1938).

Paralysis of upward rotation of the eyes is almost a sure indication of a brain-stem lesion. Cortical lesions do not produce it. Cords (1930).

Where then is the controlling mechanism for conjugate vertical movements? Because of the association of lesions near one abducens nucleus with inability to rotate both eyes to the side of the lesion Holmes (1921) assumed that the controlling mechanism for horizontal movements lies oral and slightly ventral to this nucleus. Occasionally one internal rectus is found to fail in lateral deviation but to act on convergence.

Gordon Holmes (1921) inclined to the view that the mechanism controlling vertical movements was in the anterior corpora quadrigemina. These movements are commonly lost with pineal tumours and those growing from the splenium of the corpus callosum. Such tumours tend to affect upward movement first, then downward movement and then convergence, as their pressure extends from before backwards. Owing to the proximity of the oculomotor nuclei the loss of upward movement is usually associated with paralysis of the individual muscles, particularly the levator of the upper lid and the sphincter pupillae. Both upward and downward movements are usually lost, though loss of the former alone is not rare, whilst loss of the latter alone is very rare. Loss of the upward gaze is often associated with loss of reaction to light with relative mydriasis, but retention of the reaction to convergence. Paralysis of downward gaze is often combined with paralysis of convergence and accommodation.

Collier (1927) considered that supranuclear phenomena can be explained without postulating the existence of supranuclear centres. Impulses reach the various nuclei in terms of conjugate movements and a lesion of the downward path to the nuclei from the cerebrum could produce one or more of the supranuclear defects.

It is probable that the association of fibres necessary for these
conjugate movements occurs by way of the dorsal longitudinal bundle. Palsies of these movements are complete in that there is no response to cortical, subcortical, vestibular or other stimulation. There is, however, another type of conjugate paralysis in which dissociation does occur.

**Supranuclear Ocular Palsies.**

In this type of palsy some only of the afferent paths to the conjugate mechanisms are involved. Not infrequently eyes may fail to respond to an order to look in a certain direction but they will follow a slowly moving object, maintain fixation while the head is moved away or rotate on labyrinthine stimulation.

The form of the associated movement that is lost may aid the localisation of the lesion.

(1) A lesion of the frontal oculogyric centres or of their connections with other cortical areas affects only volitional movements without a fixation object.

(2) A lesion affecting the pathways from these centres and also partly from the occipital lobe, upon which the attraction movements depend, may abolish these volitional movements and the attraction movements that are due to a peri-macular occipito-oculomotor reflex.

(3) A lesion more subcortical than this may remove volitional and attraction movements and "following" movements due to a macular-occipito-oculomotor reflex. Compensatory movements due to a vestibular reflex on quick rotation of the head remain intact.

(4) A lesion in the dorsal longitudinal bundle may remove the four types of movements but if the nuclei of the internal recti are intact convergence persists.

When the loss is to volition alone—"Parinaud's syndrome"—the eyes may reflexly rotate upwards if a light is flashed above or if the patient is asked to follow a slowly moving object or to bend his head forwards while he watches a fixed object. This is the only undoubted onesided paralysis due to a supranuclear lesion apart from the loss of adduction in conjugate deviation while it is intact in convergence. In the supranuclear loss of upward movement the lesion must be below the bifurcation of the pathway from the cortex into a branch to each oculomotor nucleus. The integrity of the nuclei and the infranuclear pathway is shown by the upward rotation when opening the lids against resistance, that is Bell's phenomenon. For an excellent description of the associated "catastrophic reaction" see G. Holmes (1938).

Sometimes conjugate deviations and paralyses clear up. This has suggested the presence of bilateral innervation.
A peculiar dissociation known as "skew deviation" may occur in acute cerebellar lesions if extensive. Holmes, (1921). The homolateral eye is turned downwards and inwards and its fellow upwards and outwards.

In the progressive exophthalmos and external ophthalmoplegia described by Brain and Turnbull (1938), the elevators tend to be affected and the depressors to escape.

One of the interesting manifestations that followed the wave of epidemic encephalitis after the last war was the tendency some patients showed to oculogyric crises. The most common deviation then was upwards. Not infrequently there was an inability to rotate the eyes upwards and more rarely downwards. The lesion affected the superior colliculus or its brachium to the lateral geniculate body or the adjacent tegmentum.

In myasthenia gravis a variable degree of hyperphoria has been found. Abraham (1932). This and other findings, including the close proximity of the images when diplopia is produced with a rotating prism indicated that the muscle or muscles when stimulated "contracted, but failed to relax promptly or completely, remaining in a prolonged contraction state." Some authors state that loss of upward movement is most common and loss of downward movement least common. Walsh's findings (1945), did not confirm or refute this.

It is usual with mesencephalic lesions near the posterior commissure to find partial bilateral ptosis, often with small pupils and sometimes with complete inability to rotate the eyes upwards. Collier (1927). Lesions in the same area may produce the "corresponding opposite," that is retracted lids and enlarged pupils. Collier considered that the latter condition was due to lesions further forwards than those producing the former. Indeed, he held that "tucked" or retracted lids with unvarying ocular parallelism, without diplopia, suggested a supranuclear lesion and ptosis with squint, diplopia and internal ophthalmoplegia a nuclear lesion. He added that in most cases both the supranuclear mechanism and the nuclei were affected.

Frequently the fibres of the roots of the nerves are affected by disease as they pass through the brain-stem. This is a site of attack characteristic of disseminated sclerosis. Such lesions usually interfere with adjacent structures and the signs that follow are common causes of one or other of the following syndromes.

2. Oculomotor Syndromes. Illustrations 7A and B.

Various syndromes involving the ocular nuclei have been described. They include those of

a. Parinaud. A lesion of or near the superior colliculus causes
conjugate deviation or a loss of upward or downward movement and sometimes dissociated ocular movements.

b. the central grey matter (Lyle, 1945). The oculomotor and trochlear nuclei and/or their nerve fibres and some of the adjacent nuclei may be affected.

c. Benedikt. A complete or incomplete ipsilateral oculomotor paralysis occurs with contralateral ataxia, hemitremor, hemianaesthesia and hemihyperkinesia.

d. Weber. Complete or incomplete ipsilateral oculomotor paralysis is associated with contralateral hemiplegia.

e. Foville. The "centre" for lateral gaze is affected in combination with contralateral hemiplegia or hemianaesthesia. If the lesion is destructive eyes and usually head will rotate to the opposite side. The seventh nucleus or nerve may be involved.

f. Millard-Gubler. Ipsilateral external rectus palsy is combined with contralateral hemiplegia and hemianaesthesia. A lower neuron ipsilateral facial paralysis is present and possibly involvement of the fourth and eighth nuclei.

g. Raymond. This is similar, but the seventh nerve is not involved.

h. Wallenburg. Involvement of the ipsilateral fifth, sixth and seventh nuclei or roots may accompany nystagmus, and ipsilateral ataxia and hypertonia if the posterior inferior cerebellar artery is involved.

The oculomotor nerve may be involved in the interpeduncular region and so produce a second type of Weber's syndrome. Its paralysis may be the only sign of a basal lesion whether it be meningitis, tumour or aneurysm of the posterior cerebral or the middle cerebral arteries. In the posterior part of the cavernous sinus it may be affected with the trochlear nerve and the first two branches of the fifth and the sixth nerves. Anaesthesia of only the area supplied by the first branch of the fifth nerve may be the sole sign accompanying an oculomotor paralysis if the lesion be in the anterior portion of the sinus. There may be no concomitant signs if there be a cavernous sinus thrombosis and an arteriovenous aneurysm of the carotid artery without venous communication. Kestenbaum (1946). The third, fourth, fifth and sixth nerves may be involved in a lesion of the orbital fissure or of the apex of the orbit. The optic nerve may be involved in the latter. In the orbit the contiguous muscles tend to be affected rather than a nerve group. When a muscular disease is present all the muscles may be affected.

It is significant that the two depressors receive only crossed fibres and the two elevators receive entirely or in part uncrossed fibres (Kidd) from the nuclei. The cells representing the depressor supplied by the oculomotor nucleus, viz., the inferior rectus, are
Preparation of brain, viewed from below, to show the areas principally associated with vision and ocular motor function. The lower portions of the left temporal and occipital lobes have been removed. The left half of the mesencephalon has been sectioned transversely at the level of the superior colliculus and the site of emergence of the oculomotor nerve. The pons has been sectioned (1) dorsally in its lower third and (2) ventrally at the junction with the mid-diencephal portion of the mesencephalon and the pons and the cerebellum have been sectioned in the mid-line. Sites for the lesions that may produce several well-known syndromes are shown in 7b. Oculomotor path shown in red dots.
ILLUSTRATION 7B.

Enlarged diagrammatic drawing from previous preparation of brain. The usual sites for lesions that may produce the well known syndromes are shown. The upper section is through the left superior colliculus and the lower is through the dorsal portion of the right half of the lower pons and ventrally through the junction of pons and medulla.

The following abbreviations are used:
- L.G.B. Lateral geniculate body.
- M.G.B. Medial geniculate body.
- P.B. Pineal body.
- M.F. Medial fillet.
- R.N. Red Nucleus.
- M.L.B. Medial longitudinal bundle.
- R.B. Restiform body.
those that are closest to the trochlear cells which supply the other depressor muscle. The cells of the elevators lie at the other end of the oculomotor nucleus. Therefore in a pure nuclear oculomotor lesion one expects to find the homolateral elevators and the contralateral depressor affected. In other words bilateral partial oculomotor palsies are the characteristic of a nuclear lesion and a complete unilateral paralysis of all the muscles supplied by the oculomotor nerve characterises a subnuclear lesion.

3. CONGENITAL ABSENCE OF AN ELEVATOR OR DEPRESSOR.

Such an occurrence is much rarer than are congenital pareses. Harles (1880), reported congenital absence of the oblique muscles. Seiler appears to be the only other author reporting absence of the oblique muscles. Posey (1923), discussed this and his own and the previously reported instances of absence of the vertical recti. Casten (1940), reported two cases of absence of the inferior rectus and referred to cases reported recently by Hardy, Coover and Natale of congenital absence of the superior and inferior recti. These findings were established at operation.

4. MISDIRECTION OF REGENERATING OCULOMOTOR FIBRES OR PSEUDO-GRAEFE SYNDROME.

Walsh and King (1942) reported instances of regenerating fibres finding their way to the wrong muscles as a frequent sign of intracranial saccular aneurysms. The most common was for inferior rectus fibres to reach the levator; in one case they reached the sphincter pupillae. In others "when an effort is made to move the eye in any direction, impulses flow at the same time into all the muscles innervated by the third nerve." No movement up or down occurs as the vertical recti contract together. But the eye may be adducted and the lid elevated. Bielschowsky, (1945), agreed with the above explanation and disagreed with that of Fuchs who claimed that the condition was due to a spreading of a localised stimulation to a degenerate or injured third nucleus. This presupposed a complete retrograde nuclear degeneration. In nearly all patients the third nerve must be affected by trauma or tumour in the base of the skull. Bielschowsky. These cases are similar to the rare examples of Marcus Gunn's jaw-winking phenomenon that appear to be acquired and not congenital.

One type of the retraction syndrome is that in which an impulse to adduction leads to a rotation either up and in or down and in. The underlying cause is probably a congenital fibrosis of the muscle which may need a tenotomy of the external rectus and of the inferior oblique. (See below.)
5. Cyclic Oculomotor Paralysis.

In this condition automatic alternation of spastic and paralytic conditions of a paretic eye is seen. Bielschowsky (1945), collected 32 patients of whom he had examined 10. In half the phenomenon was not congenital but had appeared in early childhood. In most cases the affected eyes were highly amblyopic or ametropic. He suggested that a part of the oculomotor nucleus retained part only of its function and acted only when it received abundant blood supply. This might occur when an impulse reached the nucleus, but after a short interval the spastic phase recurred. If an antagonistic impulse went to the abducens nucleus contraction of the blood vessels to the oculomotor nucleus occurred and the paralytic phase ensued. The elevator muscles never, the inferior recti rarely and the internal recti more frequently, are affected. In one-third of the cases the upper lid and in all, the pupil of the paralysed eye was affected.

One such patient was a boy who, when staring into the distance, would develop twitching of a paralysed upper lid. The twitching would increase until the lid was raised and then the formerly dilated pupil would contract. After ten to twenty seconds the lid would fall and the pupil dilate. Provided that no voluntary effort was made this cycle would recur day and night. If the patient looked to the left during a spastic phase it would be prolonged and on looking to the left during a paralytic phase it would be replaced by the spastic one. On looking to the right the affected right upper lid would droop and the pupil would dilate. Bielschowsky (1945).

6. Disorders of Synergic Lid and Eye Movements.

Charles Bell, in 1832, described the rotation upwards and outwards that accompanies tight closing of the lids. The upward rotation varies with the vigour of the closing. It increases sometimes when in facial palsy an extra effort is made to close the lids. The upward rotation is lessened in coma and deep sleep, when the lids rotate downwards, remaining only 10-15° above the horizontal axis and usually divergent.

Bell’s phenomenon is intact in supranuclear lesions. Its preservation in oculomotor pareses, when elevation fails to respond to volitional and vestibular stimuli, shows the integrity of the nuclei and the nerves of the elevator muscles though severed from all supranuclear pathways but that needed for Bell’s phenomenon. Bell’s phenomenon may fail also in paralysis of upward rotation. Kraupa, (1913), however, reported its presence in spite of total ophthalmoplegia in three brothers and sisters. This phenomenon
tends to fail in central facial palsy and it may be absent in peripheral cases also. Cords. In its continued absence, the effects of exposure during sleep are shown in the cornea and the conjunctiva.

Graefe and others reported downward rotation in some cases of facial palsy. This paradoxical Bell’s phenomenon was described by Coppez in two of 200 patients.

In addition it is known that lid-closing is easier when the gaze is upwards, and harder when it is downwards.

Further relationship between palpebral and ocular movement is seen in Graefe’s sign in goitre. This is probably due, as Dalrymple wrote in describing it, and the sign known by his own name—to increased tone in the levator.

Retraction Syndrome.

The following are the characteristics of this rare syndrome:—

1. Partial or complete absence of horizontal movements of the affected eye.
2. Attempted adduction leads to ocular retraction and narrowing of the palpebral aperture.
3. Attempted abduction rarely leads to retraction but causes retraction of the lids.
4. Attempted adduction may cause rotation up and in or down and in.
5. Remote near point of convergence. As a rule fibrosis of the affected muscle is present. This may be due to a birth injury or imperfect development. Spaeth, (1944). Kirby (1946) and White (1946) described the results of treatment.


Summary.

1. A syndrome.
2. Palsy obscured.
3. Three stages.
4. Summary of movements.

I. Types of Palsy and their characteristics.
   Incidence and causes of ocular palsies.
   Analysis of 402 cases of muscular defects.
   Analysis of defects associated with “concomitant” strabismus.

II. Concomitance.
   Overaction and contracture of antagonist and synergist.
   Inhibitional palsy of contralateral antagonist.