The diagnosis of ocular paralyses / by Archibald S. Percival.

THE

DIAGNOSIS OF OCULAR PARALYSES.

ILLUSTRATED WITH DIAGRAMS.

BY

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THE DIAGNOSIS OF OCULAR PARALYSES.

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The exact diagnosis of a paralysis of one or more of the ocular muscles is of extreme importance to both surgeon and physician; and while it seems at first sight to be surrounded with difficulties, it is hoped that many of these will disappear on a careful consideration of their nature.

A brief reference to the anatomy of the orbital muscles must be first made. The four recti muscles arise from a common tendinous oval ring, which is attached to the inner side of the optic foramen, and then stretching obliquely across the sphenoidal fissure, is attached to its lower border. From this point the common origin again crosses the sphenoidal fissure—this time about its middle, and vertically—to join the upper edge of the optic foramen. The insertions of the recti tendons into the globe are too well known to need a description in detail. I would only call attention to the four intra-capsular ligaments, which are strong fibrous loops through which the recti pass to gain the interior of the capsule of Tenon. These intra-capsular ligaments, being attached through the capsule of Tenon to the bony walls of the orbit, act as pulleys, and protect the globe of the eye from pressure during contraction of the recti muscles. The external rectus abducts, the internal rectus adducts the eye; in fact, their action is one of simple rotation of the eyeball round a perpendicular axis through its centre of rotation. This centre of rotation is in emmetropic eyes about 13·5 mm. behind the apex of the cornea—that is, rather more than 1·7 mm. behind the centre of the visual axis.

The line of action of the superior and inferior recti forms an angle of 27 degrees, with the sagittal (or antero-posterior) axis of the eyeball, consequently it is only when the eye is abducted 27 degrees that the action of the superior and the inferior recti is to

elevate or depress the cornea, without imparting a torsional movement on the globe. In adduction a torsion occurs under the action of the superior rectus, such that the upper extremity of the vertical meridian is inclined inwards; when the inferior rectus acts, the torsion occurs in the reverse direction (*vide* Fig. 2). Both muscles also tend to adduct the eye and, like the other recti, to retract the globe.

To learn the action of the superior oblique, we have merely to consider that section of it which lies between the trochlea and the eyeball. The trochlea lies a little behind the upper and inner margin of the orbit. After traversing the trochlea the tendon of the superior oblique passes beneath the superior rectus, where it spreads out in the form of a fan, and is inserted near its outer edge into the posterior part of the eyeball. When the superior oblique contracts, the posterior half of the eyeball is drawn forwards, inwards, and upwards. The globe is therefore protruded while the cornea deviates outwards and downwards. At the same time a torsional rotation occurs, which is most marked in abduction, by which the upper extremity of the vertical meridian is inclined inwards.

The inferior oblique arises from the orbital plate of the superior maxilla, just on the outer side of the orifice of the nasal duct. It is inserted into the posterior part of the eyeball, under the cover of the external rectus. The inferior oblique causes a torsional rotation of the globe, by which the upper extremity is inclined outwards; furthermore, it protrudes the globe, while the cornea deviates outwards and upwards.

The line of action of the two obliques forms an angle of 51 degrees with the sagittal axis of the eyeball, consequently when the eye is abducted 39 degrees the action of these muscles is to impart a torsional rotation to the globe without any movement of elevation or depression (*vide* Fig. 1).

When the eye is adducted, the torsional rotation due to the obliques is minimised, while their power of elevating and depressing the eye reaches a maximum. Consequently when the eyes are converged and depressed as in ordinary reading, together with the

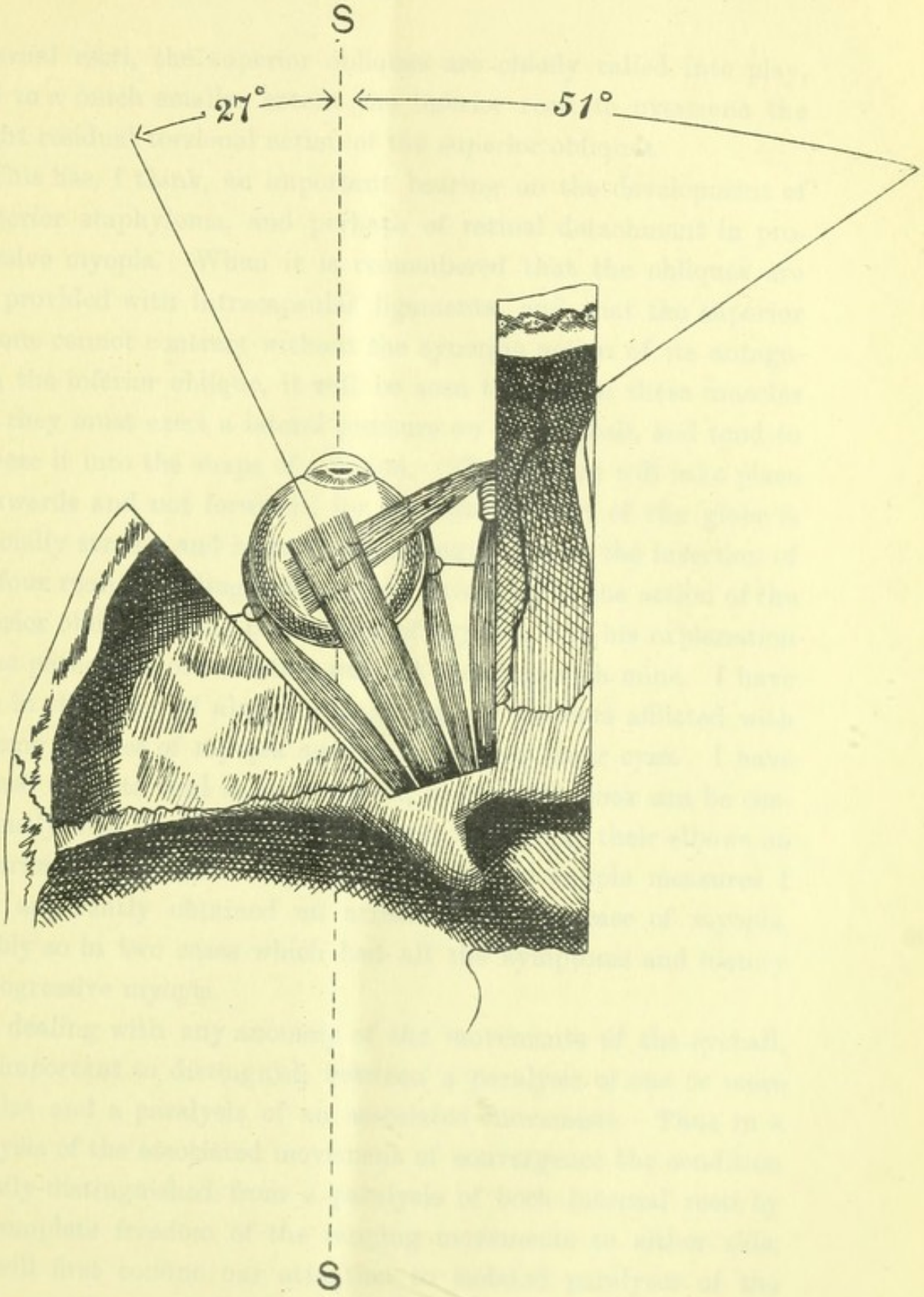

FIG. 1.

internal recti, the superior obliques are chiefly called into play, and to a much smaller extent the inferior recti to overcome the slight residual torsional action of the superior obliques.

This has, I think, an important bearing on the development of posterior staphyloma, and perhaps of retinal detachment in progressive myopia. When it is remembered that the obliques are not provided with intracapsular ligaments, and that the superior oblique cannot contract without the synergic action of its antagonist, the inferior oblique, it will be seen that when these muscles act, they must exert a lateral pressure on the eyeball, and tend to squeeze it into the shape of a lemon. The bulging will take place backwards and not forwards, for the anterior part of the globe is especially strong, and is moreover strengthened by the insertion of the four recti. Stilling paid especial attention to the action of the superior oblique in the production of myopia, but his explanation of the pathogeny of its action was not identical with mine. I have been in the habit of always cautioning my patients afflicted with extreme degrees of myopia against depressing their eyes. I have advised them to read in an armchair, so that the book can be conveniently held at the level of the head by resting their elbows on the arms of the chair. By these and similar simple measures I have apparently obtained an arrest in the increase of myopia, notably so in two cases which had all the symptoms and history of progressive myopia.

In dealing with any anomaly of the movements of the eyeball, it is important to distinguish between a paralysis of one or more muscles and a paralysis of an associated movement. Thus in a paralysis of the associated movement of convergence the condition is easily distinguished from a paralysis of both internal recti by the complete freedom of the ranging movements to either side. We will first confine our attention to isolated paralyses of the ocular muscles. Of these, paralysis of the external rectus or of the superior oblique is the most frequent, as they are each supplied by a separate nerve, viz., the sixth and the fourth, whereas the other ocular muscles, being all supplied by the third nerve, are more usually paralysed in combination.

Paralysis of the External Rectus.—Suppose the left eye is affected; the eye cannot be abducted, and therefore there is diplopia on the attempt at ranging movements to the left. To avoid this diplopia the patient usually turns his head to the left. In paralysis of any ocular muscle, the patient tends to place his head in that position in which the diplopia can be best avoided. As I observed in a previous paper, the general law may be stated in the following form:—The head assumes a position similar to that which the eye would take were the paralysed muscle to act upon it. The sound eye assumes a direction similar to that of its paralysed fellow. This peculiar carriage of the head is so characteristic in different paralyses that an attentive observer is able from it alone to suspect the exact nature of the paralysis.

In incomplete paralysis of an ocular muscle, valuable aid to diagnosis may be afforded by the false orientation induced by the paresis. This depends on the fact that our knowledge of the position of things in space is largely due to the innervation we give to the ocular muscles. A patient, for instance, with paresis of the left external rectus will have to make a considerable effort in order to turn his eye at all to the left. If the sound eye be covered, he will imagine that an object to the left occupies a more lateral position than it does in reality, and, if asked to touch it, he will stretch his hand to the left of it. False orientation is always in the direction of the action of the paralysed muscle. It is always a prominent symptom in recent paralyses, but in old-standing cases it is often absent, as the patient learns to compensate for his error.

Closely associated with the increased innervation, which gives rise to false orientation, is the so-called *secondary deviation* of the sound eye. In the above case of paresis of the left external rectus on covering the sound eye, the patient will have to send out an exceedingly strong innervation from the cortical centre for ranging movements to the left, in order that the left eye may fix an object on the left. The right eye behind the screen will therefore undergo a much stronger deviation to the left. Suppose, for instance, that the conductivity of the left sixth nerve is diminished two-thirds;

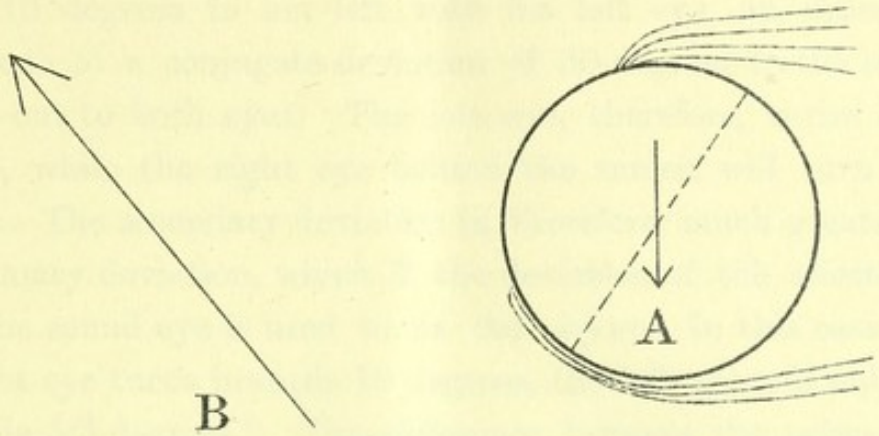

FIG. 2.

Left Superior Oblique (or left superior rectus) rotating the upper extremity of vertical meridian inwards.

A.—The inverted image on the retina of a vertically directed arrow.

B.—The oblique image as projected by an eye in this position of torsion.

on covering the right eye, and directing the patient to fix an object 10 degrees to his left with his left eye, an innervation equivalent to a conjugate deviation of 30 degrees to the left will be sent out to both eyes. The left eye, therefore, turns out 10 degrees, while the right eye behind the screen will turn in 30 degrees. The secondary deviation is, therefore, much greater than the primary deviation, which is the deviation of the affected eye when the sound eye is used to fix the object. In this case, when the right eye turns inwards 10 degrees, the left eye will only turn outwards $3{\cdot}\dot{3}$ degrees.* This difference between the primary and secondary deviations constitutes the essential distinction between a paralytic and a concomitant squint. Under the title of concomitant squints are included not only the convergent strabismus of hypermetropia and the divergent strabismus so commonly seen in myopia, but paralyses of the associated movements. The distinguishing feature of these squints is their concomitancy, and always implies a lesion or anomaly of action at or above the nuclear origin of the nerves.

Often what is called the movement of readjustment is more readily seen and recognised than the secondary deviation, which takes place behind the screen. Suppose, as above, a secondary deviation of the right eye behind the screen of 30 degrees inwards. On removing the screen and placing it before the left eye, and on directing the patient to now fix the object with his right eye, a distinct movement outwards of readjustment of the right eye may be seen. This movement enables us to distinguish very slight paretic conditions, and a want of balance between the ocular muscles from a normal state, or one associated with apparent squint (due to an anomaly in the value of the angle α).

Another common symptom in ocular paralysis is vertigo. This giddiness depends upon the diplopia, and on the false orientation; it is often most marked on going up or down stairs. In severe cases it may even induce a tendency to vomit. This vertigo always disappears at once on closing the paralysed eye.

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* The measurement of the extent of the secondary deviation is the most accurate and reliable mode of estimating the progress of a case of ocular paralysis under treatment.

Diplopia.—This is due to the image formed on the retina of the paralysed eye being not formed on the area corresponding to that which the image of the sound eye occupies. With left external rectus paralysis, when the object viewed is in the horizontal plane to the left of the patient, his left external rectus is innervated, but the muscle does not contract. The patient, therefore, imagines that his eyes are in a position of conjugate deviation to the left, while they are really in a convergent position. The image of an object formed at the macula of the sound (right) eye is formed to the right of the macula in the left eye. The patient, from his false orientation, assumes that there is another object in that position which it would have were his eyes in the position of conjugate deviation to the left. The position in space corresponding to the image on his retina is to the left. The diplopia is consequently homonymous—*i.e.*, the false image, that seen by the left eye, appears in space to the left of the true image. Homonymous diplopia implies convergent strabismus, whereas heteronymous diplopia implies divergent strabismus.

It will be noticed in the chart that the lateral separation of the images increases on an attempt to abduct the paralysed eye, and to a less extent on depression. The reason that every pathological convergence is most strongly marked on depressing the eyes is, that physiologically some amount of convergence is usually associated with depression, and this quotum has to be added to the pathological amount.

In diagonal positions of the object there is some tilting of the false image. Why does this tilting take place?

It will be remembered that when the eye is abducted 27 degrees the action of the superior and inferior rectus is simply one of elevation and depression. Therefore, when the object is held 27 degrees towards the paralysed side, and above the horizontal plane, the appropriate innervation is sent out to the external rectus and to the superior rectus of the paralysed eye. The superior rectus, however, alone contracts, and causes a rotation of the globe, turning the upper extremity of the vertical meridian of the retina inwards. The projection (B), due to false orientation,

corresponds to the image of a vertically-directed arrow seen by the eye in this position of torsion (Fig. 2). When the object is held below the horizontal plane, the inferior rectus acts and rotates the eye in the opposite direction; the false image, therefore, is tilted in the opposite direction. Careful examination will shew that the false image is less raised and less depressed than the true image in these positions, as part of the vertical displacement yields to the torsional displacement induced.

Paralysis of the Internal Rectus.—Adduction of the paralysed eye is prevented, so that as the object is moved towards the sound eye the divergence becomes more excessive and the diplopia more markedly heteronymous. When the object is raised above the horizontal plane, and displaced laterally towards the sound side, the false image is tilted. This occurs because normally in this direction of fixation the internal rectus and the inferior oblique are chiefly innervated, and the superior rectus to a much smaller extent. Consequently, when the internal rectus is paralysed, the preponderant contraction of the inferior oblique makes itself felt by a torsion of the globe, with a corresponding torsion of the false image in the reverse direction. When the object is displaced downwards and inwards, a similar torsion of the false image occurs in the opposite direction, due to the over-action of the superior oblique.

This paralysis is one of the most rare isolated paralyses, but when it occurs the patient will usually keep his head turned towards the sound side, so as to minimise the inconvenience occasioned by the diplopia.

Paralysis of the Superior Rectus.—Movements of the eye upwards are restricted, especially when the eye is turned outwards. There is heteronymous vertical diplopia in the upper part of the field of fixation, but a difference is seen in the position of the false image on abduction and on adduction. On abduction elevation of the eye is caused by the superior rectus almost entirely, hence on paralysis of that muscle, the false image is a good deal higher than the true image, and nearly or quite parallel to it. On adduction a different state of things occurs. If the eye could be adducted 51 degrees,

the inferior oblique alone would raise the eye and no diplopia would be observed; but the eye cannot be adducted to such an extent. Therefore in intermediate positions of adduction both the inferior oblique and the superior rectus act in raising the eye, the latter chiefly to counteract the torsion induced by the inferior oblique in such positions. Hence on paralysis of the superior rectus this tersion is not counterbalanced, and the false image appears inclined to the true image and more nearly on a level with it, due to the unopposed action of the inferior oblique (*vide* chart). The position of the head is that in accordance with the general rule above stated, and it only remains to add that when the levator palpebræ is not at the same time affected (they are both innervated by the superior branch of the third nerve) there is often, on the attempt to raise the eye, a retraction of the upper lid which gives a peculiar staring appearance.

Paralysis of the Inferior Rectus.—In this case almost the opposite of the preceding symptoms present themselves. Movements of the eye downwards are restricted, especially when the eye is turned outwards. There is heteronymous vertical diplopia in the lower part of the field of fixation; on abduction the false image is a good deal lower than the true image; on adduction the false image rises and becomes more inclined owing to the unopposed action of the superior oblique. On the attempt to depress the eye the upper lid does not descend in consort with its fellow, but remains raised in correspondence with the position of the eye.

Paralysis of the Superior Oblique.—There is defect of movement downwards in the eye affected, especially marked when it is turned inwards. There is homonymous vertical diplopia in the lower part of the field of fixation. On abduction there is some torsion of the globe and consequently of the false image, due to the unopposed action of the inferior rectus. The position of the head follows the general rule so that it is held downwards and towards the sound side, while the chin is directed towards the paralysed side. The patient may also remark that the false image seems to lie closer to him than the real image. This curious symptom is also quite frequently met with in cases of paralysis of the inferior

rectus, and no satisfactory explanation of it has been offered. It may, I think, be due to the fact that most near objects are below the horizontal plane, and consequently when a depressor of the eye is strongly innervated, even though it does not contract, the sensation of nearness attends the innervation.

Paralysis of the Inferior Oblique.—Movements of the eye upwards are restricted, especially when it is turned inwards. There is a homonymous vertical diplopia in the upper portion of the field of vision, and a torsion of the false image due to the unopposed action of the superior rectus. This muscle is rarely paralysed alone, and when it is remembered that the lenticular ganglion derives its motor root from the branch of the third nerve which supplies this muscle, its frequent association with dilation of the pupil and loss of accommodation is explained. Occasionally an isolated paralysis of this muscle has been met with, owing to the accidental detachment of its origin caused by wounds about the inner angle of the orbit (Berry).

In isolated paralyses of the ocular muscles a reference to the chart will, it is hoped, make the diagnosis of the particular muscle affected sufficiently clear under normal circumstances. It will be noted that in these conditions when there is any inclination of the false image, its direction is always such that the two images appear to converge towards the horizontal plane. For instance, suppose a patient complains of homonymous vertical diplopia in the lower part of his fixation field, this may be due to an isolated paralysis of the superior oblique, or to a combined paralysis of the external rectus and of the inferior rectus. In the latter case the images will appear to diverge from the horizontal plane; besides, when the eye is adducted, the characteristic displacement of the false image due to inferior rectus paralysis will make itself manifest. That is to say, the diplopia will be heteronymous, and the false image will converge with the true image towards the horizontal plane.

Under exceptional circumstances the diplopia may be heteronymous when one would expect it to be homonymous or *vice versa* This arises from one of two causes. (1) The previous position of

equilibrium in the eyes. (2) The so-called secondary contracture of the antagonistic muscle.—(Berry.)

(1.) This is closely related to the subject of faulty tendencies of the ocular muscles. If the natural position of equilibrium before the onset of paralysis be one of esophoria (or convergence), the heteronymous character of the diplopia, due to paralysis of the superior or inferior recti, will become less marked, or may even become homonymous. In this case the two images will diverge in the direction of the horizontal plane (see chart). If the previous condition of the eyes be the abnormal one of exophoria (or divergence), the homonymous character of the diplopia, due to paralysis of the obliques, may become less marked, or may even become heteronymous.

(2.) Secondary contracture in ocular paralysis is not analogous to contracture of muscles elsewhere in the body. There is, for instance, no fixing of the eye in one position; it is free to move in the direction opposite to that of the supposed contraction. The term is obviously a misnomer (Berry). Suppose, for instance, a case of left superior rectus paralysis. An increase of the cortical innervation to the left superior rectus will be attended by the synergic cortical innervation to its antagonists, the left inferior rectus and the left superior oblique. But the path to the left superior rectus is blocked; in other words, the muscle is paralysed, therefore the synergic contraction of the antagonists alone manifest itself. This seems to me the most rational way of explaining the pathology of this so-called secondary contracture of the ocular muscles. To return to our case: If the contraction of the left inferior rectus preponderates, adduction of the eye will ensue, and consequently homonymous diplopia, extending to the lower half of the field of fixation; if there is secondary contracture chiefly of the left superior oblique, heteronymous diplopia will be the result, and it will extend into the lower half of the field. In this case it is clear that the subjective orientation of the paralysed eye is false. The patient imagines that his eye is raised, owing to the innervation which he has sent out to the left superior rectus. He is not aware that the innervation has never reached the muscle,

and that in consequence the eye deviates downwards; hence the centre of his field of fixation is falsely projected upwards, whereas it is in reality displaced downwards. This explanation appears more complete and satisfactory than the more usual one of the two images being formed on non-corresponding retinal areas of the two eyes.

It may be remarked that spasm, or secondary contracture of a muscle, produces the opposite results to paralysis of that muscle. On contraction of the left superior rectus—for instance, the cornea is raised and adducted, while the globe is retracted and rotated in such a way that the upper extremity of the vertical meridian of the retina is turned inwards. The diplopia is therefore homonymous and occupies the lower part of the field; the torsion of the false image is most marked when the eye is adducted and takes place in such a direction that its upper extremity is deviated outwards, while the vertical separation between the images is most marked on abduction. Since the diplopia is homonymous and occurs in the lower part of the field the two images appear to diverge from the horizontal plane, so that this distinction, as well as the position in which the greatest torsion takes place, serves to distinguish clearly between an isolated contraction of the superior rectus and an isolated paralysis of the superior oblique.

Exceptional cases may occur in which even the torsion of the false image may fail to manifest itself. I saw a remarkable instance of this last month. The patient was a lady who had received an accidental injury to her right eye $2\frac{1}{2}$ years ago from the point of a gentleman's walking stick. Since that time she complained of supraorbital neuralgia reaching to her occiput, and vertical homonymous diplopia on looking downwards. She made no complaint of any tilting of the false (lower) image. A simple explanation was given of this puzzling circumstance on examining her eye. A small scar over the inner part of the eyelid shewed that the site of the injury was immediately over the trochlea. No doubt the trochlea had been detached and pushed backwards into the orbit, where it had formed a new connection by fibrous adhesions with the inner wall of the orbit at a point below and

deeper in than its old site. On this supposition all the peculiarities of her diplopia were explained, while her neuralgia was doubtless due to a fracture of her supraorbital foramen or to a laceration of the nerve. For her diplopia I ordered her prisms (edge up before the right eye), which relieved her of it, and seem to have been of great benefit.

The characteristic symptoms of a paralysis are more unmixed and pronounced the more recent it is.—(Fuchs.)

(1.) In a paralysis of long standing the so-called contracture of the antagonists sets in, with the result of an increase in the deviation of the eye and of its becoming manifest over a more extensive area than before, even over the entire field of fixation. Consequently paralytic strabismus acquires a constantly greater and greater resemblance to a concomitant squint, which is the characteristic symptom of an anomaly of the associated movements. However, even in old cases, the concomitancy will be found imperfect. In a paralytic case the deviation of the eye, and consequently the displacement of the false image, will be found to increase as the object of fixation is moved towards the side corresponding to the action of the paralysed muscle. For instance, with complete paralysis of the left external rectus, together with secondary contracture of the left internus, there may be homonymous diplopia over the entire field of fixation; but the concomitancy is imperfect. There will be a greater deviation of the eye, and a greater lateral separation between the images, when the object of fixation is moved towards the left. This serves to distinguish it from a convergent concomitant strabismus, or an anomaly of the associated movement of convergence, when the anomaly depends on the associated innervation to the centre for accommodation as in the common squint of hypermetropia, or on some anomaly existing, either at or above the nuclei of the third nerve. It will be remembered that the most anterior parts of the converging third nerve nuclei at the openings of the aqueducts of Sylvius are associated with three acts, viz., accommodation, contraction of the pupil, and convergence, so that excess or loss of one or more of these acts will be probably due to a lesion at or above this point.

(2.) In old paralysis the false orientation is much less marked. The patient learns by experience that the innervations to the paralysed eye correspond to much slighter actions than those for the sound eye.—(Fuchs.)

(3.) The diplopia may disappear owing to the mental suppression of the sensory impressions on the paralysed eye. This suppression is rendered easier by the fact that the image corresponding to the paralysed eye is formed on an eccentric and consequently less sensitive part of the retina. The latent diplopia may frequently be made manifest by lowering the visual acuity of the sound eye, by placing before it a piece of coloured glass. Sometimes, however, owing to this eccentric spot on the retina becoming from practice more sensitive than the macula, no diplopia exists. Thus, I have had a case of divergent concomitant strabismus associated with myopia, in which diplopia could be made manifest by the use of weak abducting prisms (edges outwards). Though these prisms were not strong enough to correct the divergent strabismus, the diplopia complained of was homonymous in character. The fact that eccentric fixation took place when the eye with the best vision was covered, and neither secondary deviation nor a movement of readjustment occurred when the screen was removed, confirmed the diagnosis.

In conclusion, I will only make a few remarks on two further difficulties which may confront the physician in making a diagnosis. The first is due to the occasional presence of monocular diplopia. This usually occurs from an anomalous refraction of the rays of light (a form of irregular astigmatism, subluxation of the lens, or incipient cataract), or from a double pupillary opening (*e.g.*, from iridodialysis) when the refraction of the eye is not adjusted for the distance at which the object of fixation is placed. Monocular diplopia is easily distinguished from binocular diplopia, by the fact that it persists when the sound eye is closed.

The second difficulty occurs when, either from the blindness of one eye or from the mental condition of the patient, diplopia and all other subjective ocular conditions cannot be revealed. In this condition the diagnosis is rendered much more difficult, and one

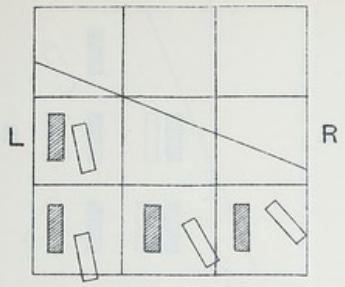
must rely solely upon the observed limitations of movement in the paralysed eye. Observation with the ophthalmoscope of a torsion of the direction of the retinal vessels, as the eye is moved in different directions, may lead to a diagnosis of a paralysis of the obliques, &c., which other means had failed to discover.

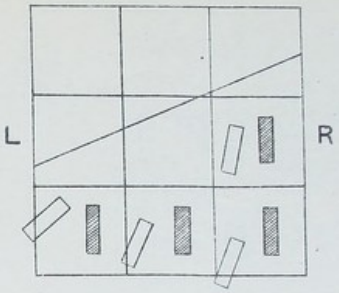
"Daily Journal" Office, Clayton Street, Newcastle-upon-Tyne.

LEFT.

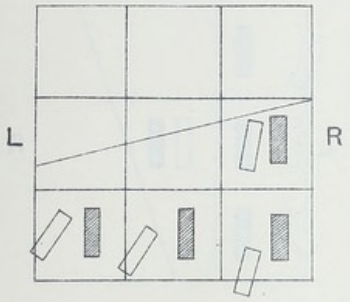
RIGHT.

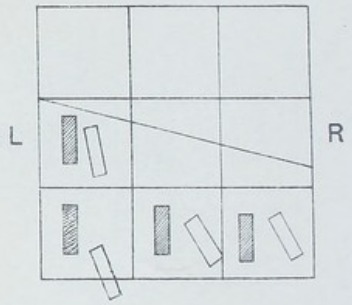
Inf. Rectus.





Sup. Oblique.





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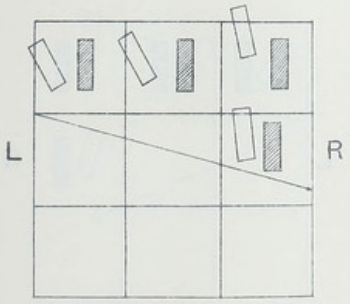
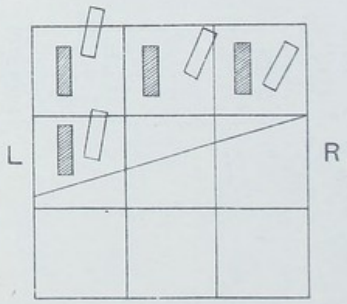
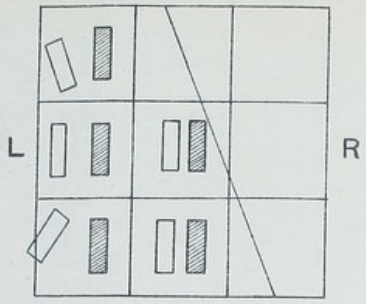


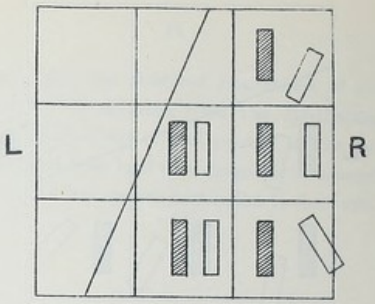


LEFT.

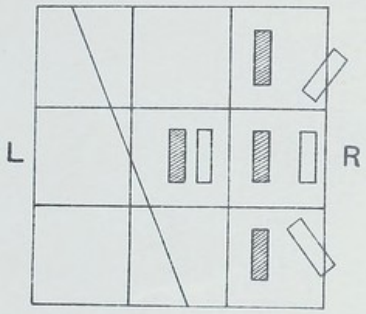
RIGHT.

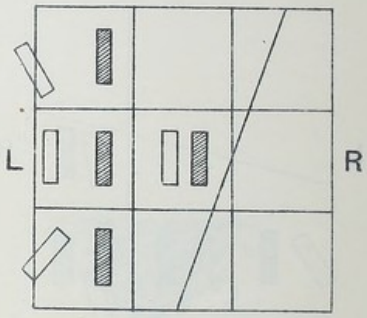
Ext. Rectus.





Int. Rectus.





Sup. Rectus.