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DEFECTIVE OCULAR MOVEMENTS AND THEIR DIAGNOSIS

E. & M. LANDOLT

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DEFECTIVE OCULAR MOVEMENTS AND THEIR DIAGNOSIS

BY

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PREFACE

THE movements of the eyes and their anomalies constitute certainly one of the most important and without doubt one of the most difficult parts of Ophthalmology. The problems which are placed before us in oculomotor affections are much more complicated than those in optics, which are, by no means, considered as simple.

In optics the eye alone is the factor with which we have to deal, whereas under the heading of oculomotor system, much more than merely the eyes must be taken into consideration. If sometimes affections of the movements of the eyes are due only to local lesions, they more frequently accompany all kinds of general diseases. On the other hand, the analysis of the various forms of oculomotor disturbances is of the highest importance in diagnosing the different diseases of the brain and nervous system.

The more thorough the examiner's knowledge of the origin, course, centres of association of these nerves, the greater is the probability of a complete correct diagnosis being made. In other words, the cerebral portion of the oculomotor system and the whole nervous system also must always be taken into account in oculomotor affections.

The course of the nerves, so easy to follow from their origin to their entrance into the muscles, has been fully established, anatomical and clinical research work has also done much to aid the specialist and the practitioner in forming a firm foundation for diagnosis.

We have endeavoured in this work to group the facts which are absolutely necessary for knowledge of the oculomotor affections, particularly those which are most apt to

be overlooked or forgotten by the general practitioner, as by so doing we hope to define and use the symptoms to their full advantage.

Pathology is preceded by a short summary on anatomy and physiology; we go somewhat more deeply into the symmetrical movements of the eyes, as well as the practical application of prisms.

We classify the oculomotor affections under four headings :—

1. Concomitant strabismus.
2. Paralytic strabismus (paralysis and contractions).
3. Anomalies of the associated movements.
4. Paradoxical motor lesions.

We have endeavoured above all to discuss precisely the symptoms of paralysis, and to classify them in an explicit manner and elucidate them with illustrations.

By referring constantly to anatomy we have attempted to facilitate the diagnosis of the nature and seat of the lesions causing the various diseases. Contrary to the method usually adopted in textbooks, we have avoided enumerating the various diseases and then describing their symptoms; but have done just the reverse. We begin with the symptoms and then lead up to the disease which causes them. It is really thus that the problem presents itself to us in practice. The patient does not come with the name of his disease, asking for the symptoms; he relates to us the symptoms, and we should, from these, diagnose the disease and deduce the nature and seat of the lesion.

The wealth of the subject has been condensed as much as possible. We have not attempted to write a handbook, but merely a handy book, a guide to aid the student and practitioner in finding his way over the oculomotor system, a path on which it is so easy to go astray.

DEFECTIVE OCULAR MOVEMENTS AND THEIR DIAGNOSIS

Anatomy and Physiology.

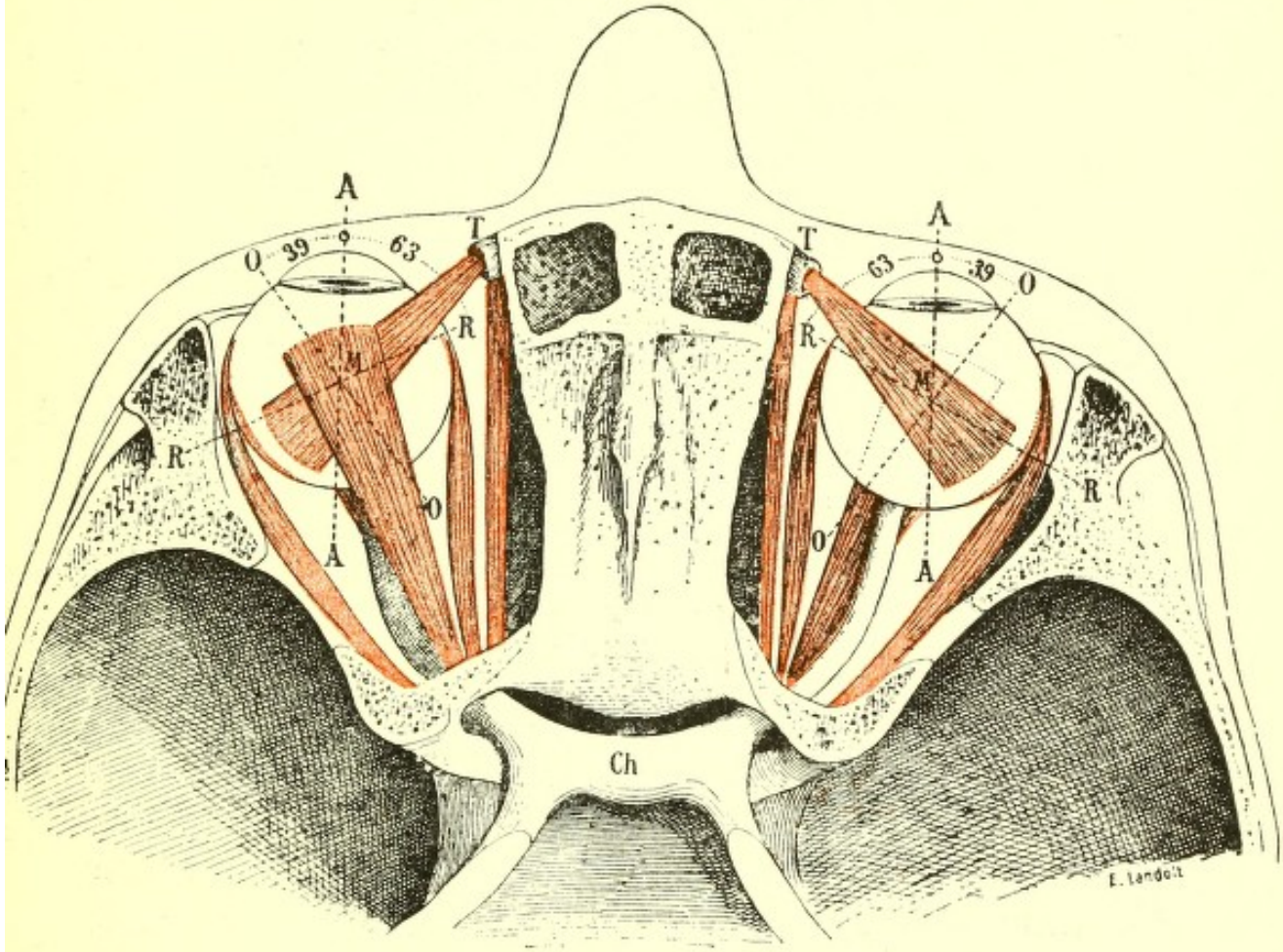


FIG. 1.—Semi-diagrammatic view of the outer musculature of the eye. (The rectus superior of the right eye has been removed to allow of the rectus inferior being seen.) The insertion of the obliquus inferior is only just visible from above. Fig. 2 gives it in its whole extent.

T, Trochlea; M, centre of rotation; AA, antero-posterior or sagittal axis; RR, axis of rotation of rectus superior and inferior. It forms with AA an angle of 63°; OO, axis of rotation of obliquus superior and inferior, forming with AA an angle of 39°; Ch, Chiasma.

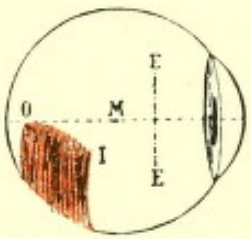


FIG. 2.—Right eye seen from the temporal region. M, Centre of rotation; EE, Insertion of rectus lateralis; OI, Insertion of obliquus inferior.

The muscles governing the movements of the eye, with the exception of the inferior oblique, all take their origin from the apex of the orbit (Fig. 3). Their course is illustrated in Fig. 1.

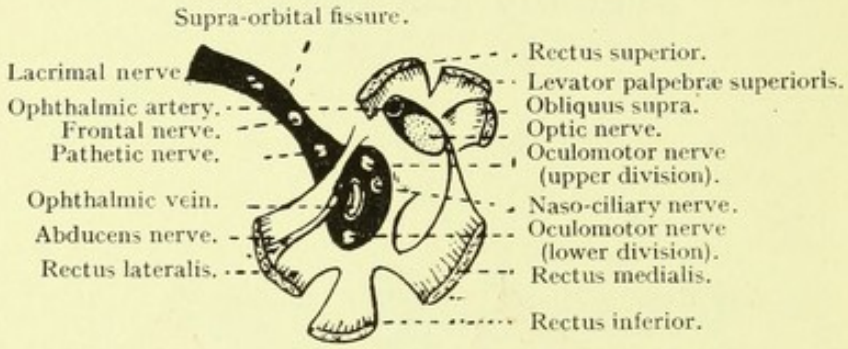


FIG. 3 (after Poirier).

Their insertions on the eyeball are found at the following distances from the corneal limbus :—

Rectus mediales	..	5·5 mm.
,, inferior	..	6·5 mm.
,, laterales	..	6·9 mm.
,, superior	..	7·7 mm. (Fuchs)
Obliquus superior	..	16 mm. (17·9 mm.)
,, inferior	..	17·3 mm. (Merkel), 19 mm. (Krause)

As is seen from this the line of insertion of the ocular muscles forms a spiral, beginning with the medial rectus, passing by the inferior and widening more and more from the limbus.

The width of the insertions :—

Rectus mediales	..	10·3 mm. (10·17 mm.)
,, inferior	..	9·8 mm. (10·35 mm.)
,, laterales	..	9 mm. (9·67 mm.)
,, superior	..	10·6 mm. (Fuchs), 10·75 mm. (L. Weiss)
Obliquus superior	..	10·15 mm.
,, inferior	..	9·55 mm. (L. Weiss)

It should be noted that the muscles, already very close

together by their broad insertions, are still more bound closely by the fascia bulbi.

The *inferior oblique* takes origin from the orbital plate near the posterior border of the lacrimal duct, vertically beneath the *supra-orbital notch* and to the medial side of the infra-orbital canal. It is directed laterally, upwards and backwards, and passes under the *inferior rectus*, from which it is separated by a small serous bursa, and is then inserted to the outer portion of the posterior hemisphere of the eyeball, a little below the horizontal meridian. If we group the muscles according to their action we have—

I. Lateral Rotators.

(*a*) Muscles directing the eyes towards the right :

Rectus lateralis of the right eye, and rectus medialis of the left eye. To a certain extent the *oblique* muscles of the *right* eye and the *superior and inferior rectus* of the *left* eye also participate in lateral rotation.

(*b*) Muscles rotating the eyes to the left :

Rectus lateralis of the left eye. Rectus medialis of the right eye. To a certain extent the *oblique* muscles of the *left eye* and the *rectus superior* and *inferior* of the *right eye.*

II. Muscles of Upward Rotation.

Superior rectus and *inferior oblique* of both eyes.

III. Muscles of Downward Rotation.

Inferior rectus and *superior oblique* of both eyes.

IV. Muscles governing Movements of Rotation round the antero-posterior Axis.

(*a*) Muscles rotating the superior extremity of the vertical meridian of the eyes to the right:

Rectus superior and *superior oblique* of the left.

Rectus inferior and *inferior oblique* of the right.

(*b*) Muscles directing the superior extremity of the vertical meridian towards the left:

Superior rectus and *superior oblique* of right, and *inferior rectus* and *inferior oblique* of the left.

According to the side to which they incline the vertical meridian, *i. e.* towards the opposite side or towards the same side, we may divide the vertical motors into—

Inward rotators: oblique and rectus superior.
Outward ,, : ,, ,, inferior.

The *red* lines of the column VIII of the synoptic diagram show exactly the position and inclination which would give the left vertical meridian (L) and right meridian (R) a rotation of 40 degrees around the axis of the muscle indicated in column I (see pp. 44–5).

The **muscular plane** passes through the origin and insertions of the muscle and the centre of rotation of the eye; it corresponds to the direction of the pull of the muscle. In the case of the superior oblique it is the trochlea which must be considered as the fixed insertion.

The muscular plane is horizontal for the *medial and lateral recti*. For the *superior and inferior recti* and for the *oblique muscles* it is vertical, but oblique in relation to the meridian plane.

The axis of rotation common to one pair of muscles (RR of the superior and inferior recti, OO of the obliques—Fig. 1) is perpendicular to the muscular plane.

The centre of rotation (M, Fig. 1) is situated in the normal eye at about 13·5 mm. behind the anterior pole, or 10 mm. in front of the posterior pole (Donders).

In *hypermetropic* eyes, which are short, the centre of rotation is situated relatively further back; in *myopic* eyes, which are long, further forward. In looking upwards the centre of rotation recedes slightly, in lowering the eyes it advances slightly (Helmholtz).

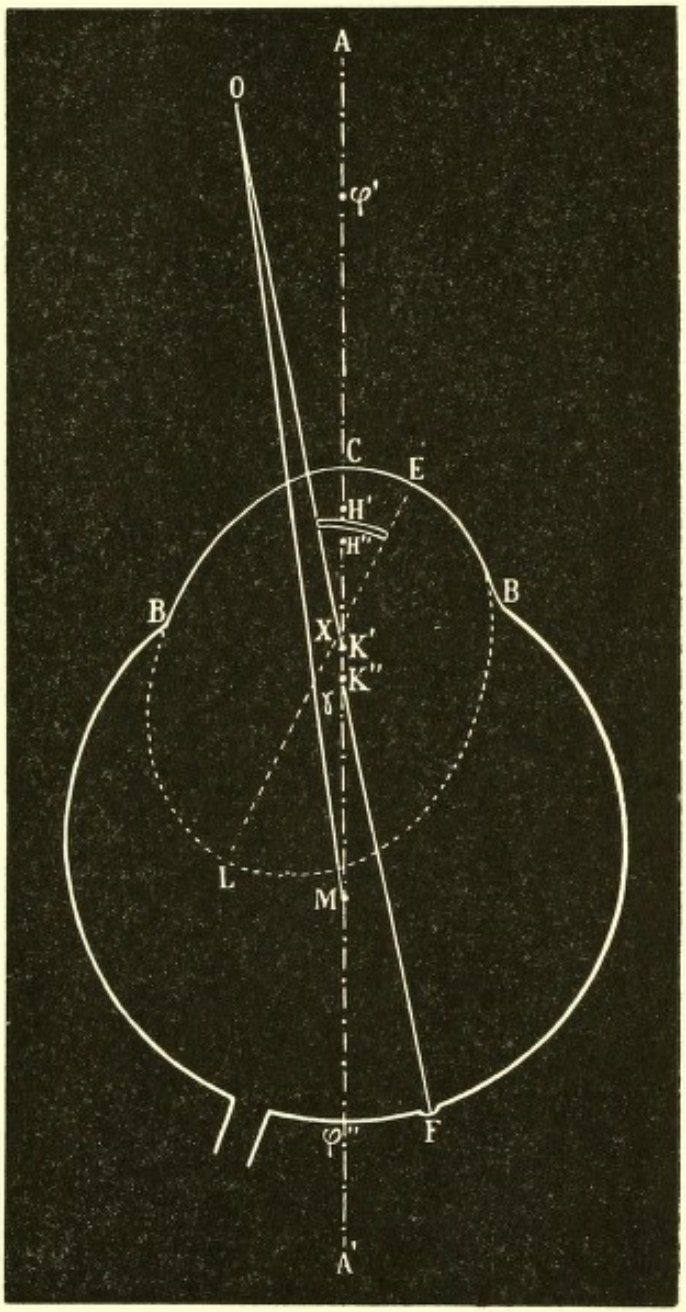


FIG. 4.

By **"optical axis"** (AA, Fig. 4) is meant the axis of the dioptric system of the eye. It comprises the cardinal points, and passes not far from the centre of rotation and practically through that of the cornea (C) (Helmholtz).

The **line of vision** is the straight ray which falls on the fovea from the fixed point. The anterior part of this line goes from the fixed point O to the first nodal point K; the posterior part from the second nodal point K″ to the fovea (F) (Helmholtz).

If the two nodal points are considered as coinciding, the straight line that passes through this point from the point of fixation to the centre of the fovea is called the **line of direction**.

The **line of fixation** (Blicklinie) is that which joins the fixed point O to the centre of rotation (M) (Helmholtz).

The **line of sight** is the line that passes from the point of fixation through the centre of the pupil (exactly the centre of the image that the cornea forms of the pupil) (Helmholtz).

The angle OMA formed by the line of fixation and the ocular axis is called angle *gamma* (γ). It is positive when the ocular axis is to the outer or temporal side; it is negative when the axis is on the nasal side.

The angle OXE formed by the line of vision (OF) and the greatest axis of the corneal ellipse (LE) is called the angle *alpha* (α). It is positive when to the temporal side, negative when to the *nasal side* of the visual line.

The angle *kappa* (κ) is the angle formed by the visual line and the corneal axis which passes through the centre of the pupil, in short the pupillary axis (E. Landolt). The angle kappa is positive when the pupillary axis is to the temporal side of the visual line, negative when to the nasal side.

The angles alpha and kappa do not interest the clinician, but the angle kappa has great practical importance, as apparent strabismus depends on this angle. If the line of vision and the pupillary axis do not coincide, appearance of strabismus may exist even though the visual lines are normally directed, that is to say, directed simultaneously, on the same point. There is apparent divergent strabismus when the pupillary axes are temporal, and apparent convergent strabismus when these axes are placed towards the nasal side. Thus a *positive* angle kappa may give the appearance of a divergent squint, or a divergent squint may be made to appear greater than it really is, or kappa *positive* may diminish the appearance of a convergent squint. Conversely, if the angle kappa be *negative*, there is an apparent convergent squint, and if there actually be a

convergent squint it appears increased, whereas a divergent squint appears to be of less degree than it really is.

Primary position of the eyes is the direction the eyes have when their visual lines are parallel and situated in the same horizontal plane (Helmholtz).

Listing's Law.—The movement of the eye from the primary position to any other secondary position may be considered as a rotation round a fixed axis passing through the centre of rotation of the eye, and perpendicular at the same time to the primary and secondary directions of the optical axis.

The *vertical meridians* of the two eyes or longitudinal meridian sections (Hering) which really correspond with each other, may diverge sometimes more than one degree in their upper extremities.

During convergence as well as in downward rotation these meridians diverge more and more (Volkmann, Helmholtz, Donders, Hering, Le Conte, E. Landolt).

Corresponding points are the points of the two retinæ which are simultaneously stimulated, and correspond to one and the same point in space. If the two retinæ were superimposed, the lines of direction corresponding to two such points would coincide (Hering).

The two eyes are innervated at the same time and to the same degree. They cannot move independently one from the other; they are controlled as if they were a single organ, the "double-eye" situated usually in an imaginary fashion, in the middle and between the two eyes. It happens, however, that one eye predominates in binocular vision, so that objects situated simultaneously on the lines of direction of both eyes, appear to lie on the line of direction of the predominating eye. The "double eye" is thus situated to one side, and coincides with the eye which we may call the **governing eye.**

All which in reality lies on two corresponding lines of direction appears as if situated on a straight course of this imaginary eye, **the binocular line of vision.**

The amount of space taken in by all the directions of the visual line, the head being immobile, would represent a cone whose apex is at the centre of rotation. A section of this cone on a plane perpendicular to the visual line in the primary position is called the field of sight, or **field of fixation.** It comprises consequently all the points in a plane towards which the visual line may be directed, without any movement of the head taking place. The extent of the monocular field of fixation in the normal state is 47 to 50 degrees. To the inner side and downwards it is more or less limited by the nose.

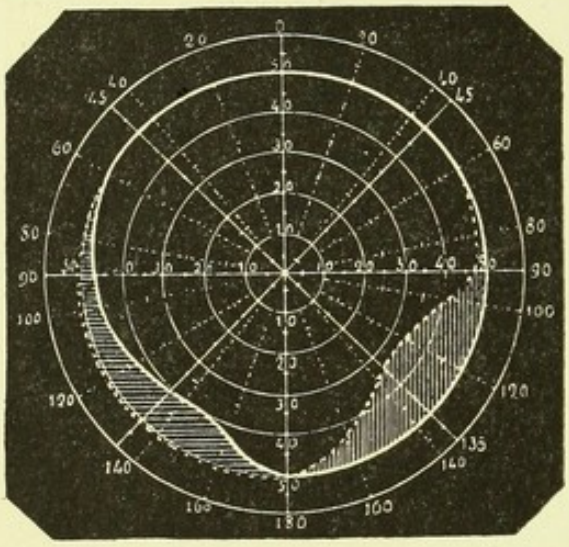


FIG. 5.—Field of fixation of the left eye (dotted line) and of the right eye (white line) in the same person (E. Landolt) both conceding in the centre the field of binocular vision of the same observer corresponds exactly with the plain black part of the figure.

Even in the normal condition the *binocular* field of fixation appears to be of less extent than the space controlled at one time by the two eyes.

This difference is considerably accentuated in the case of insufficiency of the symmetrical movements as well as in paralysis (cf. Figs. 13 and 20).

Symmetrical Movements.

By symmetrical movements we mean convergence and divergence, or, in other words, positive and negative convergence of the eyes. *Convergence* is produced by the

simultaneous contraction of the adductors with relaxation of the abductors, *divergence* by relaxation of the adductors with contraction of the abductors.

The degree of convergence is expressed by the angle formed by the visual line and the line of parallelism when the eyes both fix a point on the median line. Thus the angles IOC and I'O'C' (Fig. 6) represent the angle of convergence corresponding to the point C. This angle increases inversely to the distance (D) of the fixed point. It can thus be considered as inversely proportionate to this distance and expressed as $\frac{I}{D}$.

If the distance from the fixed binocular point to the centre of rotation is measured by metric scale, the degree of convergence for each eye can be obtained in **metre angles** (m.a.) (Nagel).

The metre angle is the angle of excursion through which each eye must be rotated to fix a point situated at one metre on the median line (MM', Fig. 6).

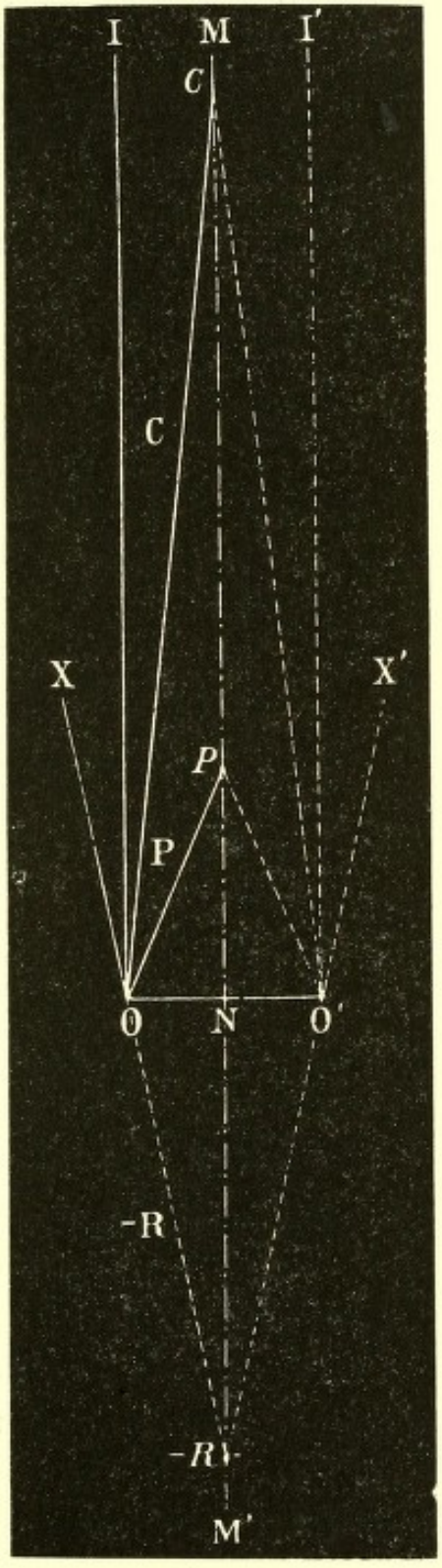


FIG. 6.

The absolute value of the metre angle depends on the distance between the eyes, or more exactly the distance

between the two centres of rotation (OO′, Fig. 6), which is called *the base line.*

In children with a base line (B′) of 58 mm. in length the metre angle is 1° 40′ = 100′.

In adults with base line (B″) of 64 mm. in length the metre angle is 1·50° = 110′.

To convert metre angles (m.a.) into ordinary angles (x), all that is necessary in the first case (B′) is to multiply the metre angles by 100, in the second case (B″) by 110, and divide by 60.

$$\text{For B}' : x = \frac{10 \text{ m.a.}}{6} = \frac{5 \text{ m.a.}}{3}$$

$$\text{For B}'' : x = \frac{11 \text{ m.a.}}{6}$$

9 metre angles, for example, would correspond in children to $\frac{10 \times 9}{6} = \frac{9}{6} = 15°$. In adults $\frac{11 \times 9}{6} = 16{\cdot}5°$.

Conversely, to find the number of metre angles which correspond to a given angle (x), we write—

For B′ (base line of 58 mm.) m.a. $= \frac{6x}{10} = \frac{3x}{5}$.

For B″ (base line of 64 mm.) m.a. $= \frac{6x}{11}$.

Example : 3° of convergence is equivalent in children to

$$\frac{6 \times 3}{10} = 1{\cdot}8 \text{ metre angles.}$$

In adults to $\frac{6 \times 3}{11} = 1{\cdot}63$ metre angles.

These figures always refer to the excursion of one eye only; to obtain the excursions of both eyes, that is to say the total angle of convergence (or divergence), we must multiply our result by 2.

If, for example, a convergence of 9 metre angles was manifest wholly in one eye, the other looking straight

forward, the converging eye would be deviated towards the nose by

$$\frac{2 \times 9 \times 10^{\circ}}{6} = \frac{9 \times 10^{\circ}}{3} = 30^{\circ}.$$

Conversely, a convergent squint of 30° would represent (*for each eye*) a convergence of

$$\frac{3 \times 30}{10} = 9 \text{ m.a. for each eye.}$$

To convert the degree of convergence (c) for each eye into angles of strabismus (s) the calculation is as follows—$s = \frac{10c}{3}$. To convert a degree of strabismus in terms of convergence the formula is $c = \frac{3s}{10}$. To simplify matters let us take a base line of 58 mm. when 1 m.a. = 100.[1] To enable the practitioner to dispense with long calculations we insert here a table to which he can easily refer.

TABLE OF REDUCTIONS

BETWEEN DEGREES AND METRE ANGLES, AND BETWEEN METRE ANGLES AND DEGREES

Degrees to Metre Angles.				Metre Angles to Degrees.			
BASE LINE 58 mm.		BASE LINE 64 mm.		BASE LINE 58 mm.		BASE LINE 64 mm.	
Degrees.	Metre Angles.	Degrees.	Metre Angles.	Metre Angles.	Degrees.	Metre Angles.	Degrees.
0° 50′ =	·30	0° 50′ =	·27	0·50 =	0° 50′	0·50 =	0° 55′
1° =	·60	1° =	·55	1 =	1° 40′	1 =	1° 50′
1° 50′ =	·90	1° 50′ =	·82	2 =	3° 20′	2 =	3° 40′
2° =	1·2	2° =	1·09	3 =	5°	3 =	5° 30′
2° 50′ =	1·5	2° 50′ =	1·36	4 =	6° 40′	4 =	7° 20′
3° =	1·8	3° =	1·64	5 =	8° 20′	5 =	9° 10′
4° =	2·4	4° =	2·18	6 =	10°	6 =	11°
5° =	3·0	5° =	2·75	7 =	11° 40′	7 =	12° 50′
6° =	3·6	6° =	3·27	8 =	13° 20′	8 =	14° 40′
7° =	4·2	7° =	3·82	9 =	15°	9 =	16° 30′
8° =	4·8	8° =	4·36	10 =	16° 40′	10 =	18° 20′
9° =	5·4	9° =	4·91	11 =	18° 20′	11 =	20° 10′
10° =	6·0	10° =	5·45	12 =	20°	12 =	22°

By base line is meant the distance between the centres of rotation of the two eyes.

[1] Cf. p. 17. The calculation of the influence of a prism placed before one eye only on the convergence.

By **amplitude of convergence** (*a*) we understand the total range of the *symmetrical movements* of the eyes.

It corresponds, therefore, to the difference between the maximum (*p*) and the minimum (*r*) of convergence (or maximum of divergence) $a = p - r$.

The maximum of convergence is inversely proportional to the distance (P) of the punctum proximum of convergence (P, Fig. 6), $p = \frac{I}{P}$.

The minimum of convergence (*r*) is inversely proportional to the distance (R) of the punctum remotum of convergence (R, Fig. 6), $r = \frac{I}{R}$.

In the normal state the maximum degree of convergence is about 9 to 10 m.a., the minimum about — 1 m.a. (A, Fig. 8). We can thus in round figures count a normal amplitude of convergence as, $a = 10 - (-1) = 11$ m.a.

The positive convergence (adduction) is best measured with Landolt's dynamometer [1] (Fig. 7); the abduction or *negative* convergence, with the abductor prisms, or more simply with a double prism of Herschel which we have graduated in angle metres and in angles of deviation. We have recently

[1] *Arch. d'opht.*, vol. v, p. 97. 1885.
This apparatus consists of a short metal cylinder blackened on the outside, and inside of which is a lighted candle. It is perforated on three different sides : on one by a vertical line of ½ mm. width, in another by a circular point, and in a third by a series of points placed vertically. The interior of the tube is lined with polished porcelain, and thus makes the holes appear bright against a black background. The dynamometer is brought nearer and nearer to the patient's face in the median line with the lighted line towards the eyes. Fixation calls into play an increasing effort of convergence. When the limit—the punctum proximum—is reached the image becomes doubled in crossed diplopia. With zero point fixed to the cylinder a metre tape graduated in cms. on one side and metre angles on the other enables one to read off directly the punctum proximum, and at the same time the figure in metre angles of convergence for each eye to which it corresponds. The series of points can also be used for measuring the punctum proximum of accommodation. The other side of the tape thus gives the number of dioptres.

had an improved model of the double prism made by Zeiss (Jena), on which the divisions are all engraved on the circular frame.[1] Though the prism is usually placed before one of the eyes, still its effect is produced on both eyes.

We have already noted (p. 11) that the two eyes are always equally innervated. Thus when a fixed object is

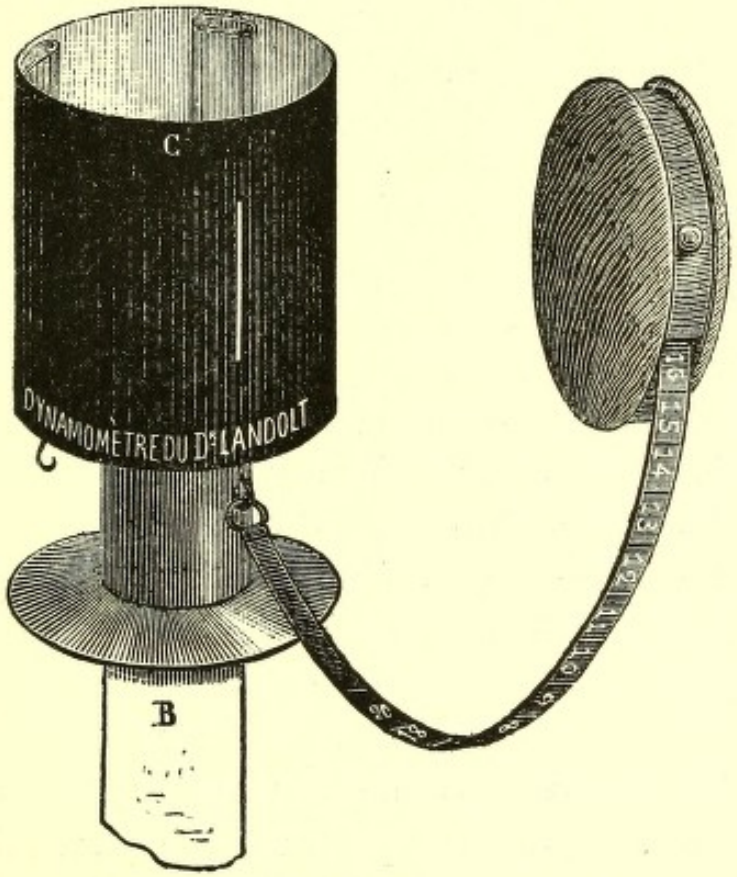


FIG. 7.

carried nearer to or further from the patient on the visual line of one eye in such a way that the other eye makes an apparent movement of convergence or divergence, the eye, which apparently remains immobile, receives the same nerve impulse as the converging eye.

For B′, an angle of deviation of $x°$ corresponds, as we have seen, to $\frac{6x}{10}$ metre angles. If both eyes are open and if

[1] *System of Diseases of the Eye*, Norris & Oliver, iv, p. 145.

a prism of this strength is placed before one of them, the binocular vision would require half of the divergence from each eye $= \frac{3x}{10}$ m.a., the prism being placed base inwards. The same amount of convergence would be required if the prism is placed base outwards.

For B″, the effect of such a prism on *each eye* $= \frac{3x}{11}$.

For example, a child can still overcome an abductor prism of 5°. He possesses, therefore, a minimum of convergence or a maximum of divergence of $\frac{3 \times 5}{10} = -1{\cdot}5$ m.a. For an adult the same prism would represent a value of $\frac{3 \times 5}{11} = -1{\cdot}36$ m.a.

The numbers corresponding to metre angles in our double prism indicate the amount for each eye; they can thus be introduced directly into the formula of the amplitude of convergence. The scale on the handle of the instrument gives in figures the angle of deviation of the prism. In diplopia, this angle corresponds to the angle of deviation and, at the same time, to that of the correcting prism. The amplitude of convergence can be illustrated by Fig. 8, p. 19.

The horizontal line OO portrays the parallelism of the visual lines, that is to say, the point zero in value of convergence represented on the diagram; the lines *below* correspond to *positive* amplitude of convergence, those *above* to *negative* convergence (divergence). The dotted lines give the metre angles.

Insufficiency of Convergence.

To allow continued binocular near work to be carried out without undue fatigue, two-thirds of the total amplitude of positive convergence should be kept in reserve (E. Landolt).

Anyone wishing to work without fatigue at one-third of a metre, that is, with an expenditure of convergence of three metre angles, should have at his disposal a maximum of positive convergence p of 9 metre angles. If he has in reserve the two-thirds = 6 m.a., he will still have exactly the 3 m.a. necessary for his work.

A third of a metre, 33 cm., is the usual distance for work. As is seen, it corresponds to the normal convergence, where $p = 9$ to 10 m.a.

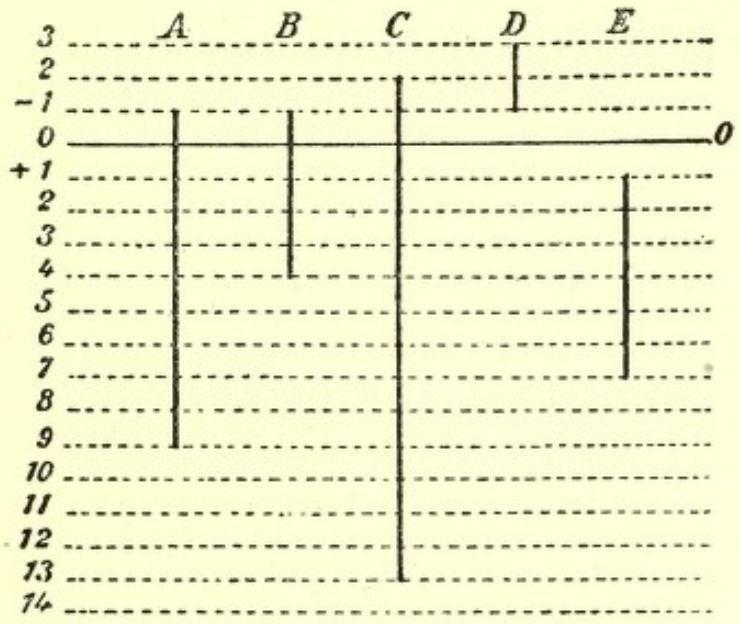


FIG. 8.

Line *A* gives the minimum normal condition, where $p = +9$ and $r = -1$; normal extent of convergence = 9 + 1 = 10 metre angles.

B represents a limitation of positive convergence to 4 metre angles, that is to say, an insufficiency of convergence with normal divergence (−1); $a = 4 + 1 = 5$ metre angles.

In *C* divergence and convergence exceed the normal amount; $p = 13$; $r = -2$; $a = 15$ metre angles.

In *D* the whole extent of convergence is negative. The patient can only diverge more or less, but cannot converge; $p = -1$; $r = -3$; $a = 2$ metre angles.

In *E*, on the contrary, the whole extent of convergence is positive; $p = 7$, $r = +1$; $a = 6$ metre angles. The patient cannot bring his eyes to parallelism; he is constantly converging at least 1 metre angle. Therefore convergent strabismus is present.. In spite of this the patient, with the greatest effort, only brings his convergence up to 7 metre angles. He has simultaneously insufficiency both of convergence and of divergence. This occurs, for example, after tenotomy of one or both externi, performed for the correction of so-called insufficiency of the interni.

When p is less than 9 m.a. the patient is obliged to put the work a little further from his eyes or to rest his convergence more frequently by stopping near work. If p is below 8 m.a. asthenopic troubles appear due to **insufficiency of convergence.** Naturally enough, one would think of aiding the convergence by using *abductor prisms*, that is to say, prisms with the base medially, or by *decentring* the spherical glasses which the patient may already wear.

The strongest prisms which can be used in glasses have an angle of deviation of 1° 40′ (a little stronger than the old number 3), that is to say, about 1 m.a. If such glasses are placed before each eye the effort of convergence is only diminished by 1 m.a. Usually patients with asthenopic symptoms have much more than 1 m.a. of deficiency of convergence. It is because of this that prisms are of so little practical use in such cases.

The Use of Prisms in Oculomotor Disturbances.

When prisms are numbered in a logical manner, that is, according to their angle of deviation, it will be very simple to use them in practice, or at least to gauge the limits within which they can be employed.[1]

[1] Prisms are still numbered according to their open angle. By this enumeration the prismatic effect obtained by the prism cannot be gauged properly. This can only be calculated if the refraction index of the substance of the prism is known. If this refraction index were 1·57, the angle of deviation would be equal to half the open angle. Glasses used in practice certainly have not all this refraction index, and thus their effect, in spite of their identity by their number, may vary considerably, and it is only in a very approximate way that the deviating effect can be considered as equal to half the open angle. Spherical glasses are not numbered by their radius of curvature, but by their refractive property. Therefore prisms should be numbered according to the properties for which they are used, that is, their angle of deviation. (*Vide* amongst others E. Landolt, *Congrès internat. d'oph. d'Utrecht*, p. 384, 1899, and *Congrès internat. des sciences med. of Berlin*, 1900.)

In correcting an affection of the *associated* movements (paralysis) the number of the prism will correspond exactly to the degree of deviation of the eye. The paralytic strabismus of 10° will require a prism No. 10, that is, one which will produce a deviation of 10°. Conversely a diplopia corrected by No. 10 prism has a deviation of 10°.

In using prisms to correct affections of the *symmetrical* movements, their effect in metre angles can be calculated from the angle of deviation according to the simple method which we have explained above.

If a patient requires spherical or cylindrical glasses in addition to his prismatic glasses, the spherical or cylindrical effect is obtained by giving the necessary curve to the side of the prism.

The Prismatic Effect of Spherical Glasses.

When, instead of passing through the optical axis of a spherical or cylindrical glass, the line of vision passes to one side of this axis, objects appear to be displaced in the same way as if one were looking through a prism : towards the margin of the glass in the case of a convex glass, towards the axis if it is a concave glass.

This prismatic effect *increases* the further one goes from the axis—*i. e.* the greater the *decentration* and the *stronger* the glass (the shorter its focal axis), the greater is the prismatic effect.

Thus it is clear that glasses must be *centred* very carefully, and especially so when they are strong.

Oculists consider glasses properly centred when the visual lines coincide with their optical axis or pass through their optical centre.

Opticians only consider the frames of the spectacles, and

say that the glasses are centred when the optical axis of the glass is in the middle of the frame.

For distant vision (visual lines parallel) the distance between the centres of the glasses should be the same as the distance between the centres of rotation of the eyes (base line).

In near vision, that is in convergence, the visual lines pass through the medial half of the glasses: thus distance glasses are not properly centred for near vision, but must have the centres brought nearer together, whether they be concave or convex. For an average base line and with glasses at the usual distance from the eye for ordinary near work, each glass should be displaced 2·5 mm. to 2·8 mm. medially towards the median plane. In actual practice the optical centres should be 53 to 57 mm. apart for near vision according to the distance between the patient's eyes (58 to 62 mm.) and to the distance at which the work is held from the eyes.

Opticians call this decentring, but it is merely centring correctly, and the lateral displacement is the same in amount whatever be the strength of the glasses, and only varying according to the distance between the eyes, the distance the glass is from the eyes, and also depending upon the distance of the object looked at. This so-called "decentring," which is really centring, is absolutely necessary to prevent prismatic effect.

Attempts have been made to profit by the deviation produced by the decentration of glasses to correct insufficiency of convergence by decentring convex glasses inwards or decentring concave glasses outwards.

In the first place, of course, it is absolutely necessary that the patient really requires concave or convex glasses. To produce useful prismatic effect these glasses must also be strong enough or sufficiently decentred, and this increases

not only their thickness but also their weight. For example, to facilitate convergence by a single metre angle at a distance of 33 cm., it would be necessary to displace a convex glass of 1 D. 34 mm. inwards, a concave of 1 D. 35·7 mm. outwards.

Now as ordinary spectacle glasses have a diameter of about 38 mm., the optical centre would be 16 mm. outside of the glass, and the bispherical lens which would require to be cut would have a diameter of 11 cm. From these figures we can see what the weight of such a pair of glasses would be.

The relationship between the angle of deviation and the decentring can be calculated by the formula given by Archibald Percival.[1]

For *convex* glasses—

$$tgx = \frac{dp - fm}{-fp + k(p - f)},$$

from which we obtain—

$$d = \frac{tgx(kp - kf - pf) + fm)}{p}.$$

For concave glasses—

$$tgx = \frac{dp + fm}{fp + k(p + f)},$$

which gives

$$d = \frac{tgx(fp + kp + kf) - fm}{p}.$$

The letters denote—

x = Angle of deviation.
d = Decentration in millimetres.
p = Distance of object from the glass.
f = Focal distance of the glass.
k = Distance of the glass from centre of rotation of eye.
m = Half of the base-line.

[1] "The Action of Prismo-spheres and Decentred Lenses," *Arch. d'opht.*, xx, No. 2, 1891; and *B. M. A.*, 1891; and *Optics*, p. 376, 1899.

The object is supposed to be on the median line.

The deviation of rays passing eccentrically through a lens cannot be calculated so simply as was made out. It depends on too many factors which cannot be left out of account.

The following table, according to Percival, gives the decentring necessary for glasses of 1, 2 and 3 D respectively, with a base line of 64 mm. to produce a deviation of 1 m.a.

The decentration is according to the visual lines, that is to say, in distant vision the centre of the spectacles and for vision at 33 cm. to a point situated 2·6 mm. to the medial side of the centre.

	CONVEX.			CONCAVE.		
	1 D mm.	2 D mm.	3 D mm.	1 D mm.	2 D mm.	3 D mm.
For distant vision	31·1	15·1	9·78	32·9	16·9	11·55
For vision at 33 cm. . . .	34·0	16·6	10·7	35·7	18·3	12·5

If the object is considered as placed at infinity, which is only rarely the case, as the use of prisms comes into play especially in near vision, *i. e.* to aid convergence, p becomes $= \infty$ and m and k disappear from the formula. It is thus reduced to the following simple formula— $\tan x = \frac{d}{f}$, from which $d = f \tan x$, which is just a repetition in algebra of what we have previously stated, that the prismatic effect, the angle of deviation ($\tan x$) of a glass, increases with the decentration (d) and diminishes inversely to the focal distance of the glass.

If for the focal distance of the glass (f) we substitute the refracting power in dioptres (D), and if the tangent and the angle are considered as equal, which can be done in considering such small angles as these, the first of

the two expressions above becomes the following formula—

$$x = -Dd\ 3{\cdot}4376.^{1}$$

As previously stated, this calculation, relatively simple, is only applicable in vision at infinity; for near vision the above formulæ or a reducing table must be used if exactitude is desired.

According to Imbert,[2] the degree of the prismatic deviation (d) produced by a spherical lens can be expressed by the following formula—

$d = 006° \times lD$ where
D = the refraction of the lens expressed in dioptres.
l = the distance between the incident and refracted ray from the axis of the lens expressed in millimetres.

It really seems to be more practical to combine the prism required with the necessary lens properly centred.

Position of Equilibrium or of Rest of the Eyes.

By "position of rest or of equilibrium of the eyes" we designate the relative positions assumed by the eyes when they are free from all effort of fixation and accommodation.

To obtain this position we must overcome accommodative efforts by mydriatics, or by means of suitable convex glasses which render accommodation unnecessary in fixing an object.

To prevent any effort of fusion we place a sufficiently strong vertical prism before one eye. Besides the vertical diplopia thus produced, we find *horizontal diplopia*, due to the divergence or convergence which the eyes assume when controlled solely by the equilibrium of their muscles.

A prism placed *horizontally* with base directed medially and sufficiently strong to produce a horizontal diplopia may demonstrate the existence of a *vertical divergence* of the eyes.

[1] A. G. Percival, *Optics*, p. 377.
[2] Imbert, "Calcul de l'effet prismatique des verres décintrés." *Ann. de l'ocul.*, 95, p. 146, 1886.

To estimate the degree of the horizontal diplopia in the first instance, of vertical diplopia in the second case, we place a red glass before one eye, and the reading is made on our mural scale (see p. 33).

A good method for diminishing the tendency to fusion consists in deforming the image of one eye. For this Maddox uses a set of red glass rods placed side by side; these placed before the eye transform a luminous point into a long red line perpendicular to their axis.

To measure the degree of deviation Maddox uses our tangent scale placed on a horizontal board with a vertical one extending above and below, forming a cross at whose centre is the luminous point of fixation. In reality the scale has a double figuring, one for a distance of one metre, the other for 5 metres.

The patient fixes the zero point and indicates which figure on the scale the red streak passes through.

To measure a horizontal deviation the rods are placed horizontally, for vertical diplopia vertically.

By this method we find that the eyes which in the normal condition are directed towards the point of fixation may diverge, converge, or be deviated in the vertical when the impulse of fusion is eliminated. These conditions we call **latent** divergence, convergence, or vertical deviation.

Such conditions are far from always being pathological, they do not even necessarily constitute a tendency to deviation. They show the position in which the eyes would be deviated if they were no longer able to fix binocularly.

Thus a muscular paralysis may be complicated by one of these deviations, which becomes manifest, because the patient is incapable of directing his eyes simultaneously on the same fixed object (*vide* diplopia in the paralysis of the vertical rotators).

DISTURBANCES OF THE MOVEMENTS OF THE EYE.

If we use the word "strabismus" to designate all the anomalies of movements of the eye we must separate these anomalies into two classes.

(*a*) **Strabismus of dissociation.**—This is the most frequent type. One eye is directed towards the fixed object, whereas the visual axis of the other passes either to the medial or lateral side, above or below it, or in an intermediate direction.

(*b*) **Associated strabismus.** — Certain associated movements of the eyes remain normal while others are deficient. For example, the eyes in such a case may be parallel and can converge, but cannot be directed upwards, downwards or laterally.

Strabismus of Dissociation.

This form of strabismus is characterised by the fact that the visual lines are not directed simultaneously towards the point on which the patient fixes his attention.

As we cannot see directly the visual lines of a patient, nor judge the direction of the eyes by the pupils, which have not a fixed relative position to the visual lines, we may diagnose a strabismus which is only apparent, or overlook one when it really exists. *Divergence* (positive angle κ) and *convergence* (negative angle κ) of the pupillary axes in relation to the visual lines may be taken for divergent or convergent strabismus, or *difference in level of the pupils* for a vertical strabismus.

To differentiate between a real and an apparent strabismus, the patient is made to look at a fixed point while the examiner rapidly covers each eye alternately.

If at the moment when one eye is *covered*, the *other* makes a correcting movement towards the fixed point, it cannot have been directed towards it while both eyes were uncovered; therefore this eye squints (E. Landolt, *Encyclop. Gräfe und Sämisch*, vol. iv, p. 708).

The correcting rotation of the eye is just opposite to the direction of the strabismus.

This test should be made in fixing a distant object as well as in fixing a near object.

Thus one distinguishes: convergent, divergent, upward and downward strabismus. The latter forms may also be called vertical divergence (Hering). Frequently the deviations occur in an intermediate position, and this oblique strabismus can be considered as consisting of a horizontal and vertical component part.

Degree of Strabismus.

The degree of strabismus is measured by the angle of deviation of the eye. **The angle of strabismus** is formed by the direction that the visual line should have, and the direction which it really has.

In a case where a real diplopia exists the degree is measured by the distance between the two images; in other words, it can be measured subjectively.

Fig. 9 and the accompanying explanation show that the *distance between the two images is equal to the tangent of the angle of strabismus*. The degree of strabismus is also estimated by the angle of deviation of the *correcting prism*, *i. e.* the one which gives rise to fusion of the two images.

When binocular vision is absent the degree of strabismus

can only be estimated *objectively*. The simplest method is by making use of the perimeter. The method is as follows:—

The centre of the arc of the perimeter, the zero point on the arc and a point at a distance are placed so as to be in one straight line. Under these conditions the visual axis of an eye, placed at the *central* point of the perimeter and fixing the distant point, passes through O on the arc of the perimeter.

The patient is seated in front of the perimeter so that the eye which is constantly deviated is in the centre, the other eye fixing the distant point. The visual axis of the squinting eye will then meet the arc at a point which will give the amount of the angle of deviation.

As the visual axis cannot be seen, we must look for the pupillary axis instead (Fig. 4). To obtain this, a luminous point is made to travel along the arc of the perimeter, and the reflex on the cornea of the eye under examination is noted. The point on the arc is sought where the image of the light falls on the centre of the patient's pupil; the visual line of the examiner, the light, and the reflex of the light on the patient's pupil should lie on the same radius of the perimeter. This point on the arc of the perimeter gives the degree of the angle of deviation. The visual line and pupillary axis do not necessarily coincide, and to obtain the real degree of strabismus we must subtract from it the value of the angle κ if $+$ and add the value of κ if $-$. The contrary will require to be done in divergent squint obtained by the objective method (see p. 10).

Classification of disturbances of the movements of the eye according to their causes:—

(1) Lesion of the muscles or their nerves (*paralytic or spastic strabismus*).

(2) Defective binocular vision (*concomitant strabismus*).

(3) Affections of the centres governing symmetrical or associated movements : paralysis or spasm of *convergence, divergence,* the *lateral* movements, or *upward* and *downward* rotation of the two eyes (*associated paralysis*).

(4) Affections of centres and organs governing the equilibrium of the body (*paradoxial affections*).

I. PARALYTIC STRABISMUS.

Symptomatology of ocular paralysis. General Laws.

Direction of the paralysed eye.—An eye affected with paralysis of one of its muscles is rotated in the direction and position exactly opposite to that which the normal action of the muscle produces.[1]

The retinal image of the deviated eye (false image)[2] is projected in exactly the opposite direction to the pathological deviation and inclination taken by the eye.

The result is that the red line which corresponds in column VIII of diagram (p. 45) to the position of the false image, shows at the same time the direction and inclination of the eye when it is under the normal influence of the corresponding affected muscle.

The distance between the false and true image is equal to the tangent of the angle of strabismus.[3]

[1] If the eye which has the better sight, and is usually employed for fixation, be affected with muscular paralysis, the paralysed eye remains straight, while the other squints. (See below : secondary deviation.)

[2] For the sake of simplicity we call the image corresponding to the deviated eye " false image." Affected side the side of the affected eye, normal side that of the normal eye. Left and right inclination the inclination of the image, or the head, towards the left and right shoulder.

[3] E. Landolt, *Ann. d'ocul.*, 1875. *Traité compl. d'oph.* (De Wecker et Landolt, i, p. 926, 1878; *Meth.*, p. 730.) Gräfe u. Sämisch, iv, p. 730, 1904. Norris & Oliver, *System of Diseases of the Eyes.*

In Fig. 9, L represents the left eye with a *paralysis of the lateral rectus*, and therefore is turned towards the right, *f* the fovea centralis, O a distant point of fixation, *m* the centre of rotation.

Instead of being directed towards O like the normal eye (R), the paralysed eye (L) is directed towards Ω. *o*O is the direction which it should have, $f\Omega$ that which it has in reality. O$m\Omega$ is then the *angle of strabismus*, OΩ its *tangent*.

The image *o* of the object, which, in the normal eye, falls on ϕ fovea, falls in the affected eye on a point *o* towards the inner side of the fovea corresponding to the value of the angle of strabismus (O$m\Omega$ = *fmo*). This point is nasal to the retinal centre, and is projected laterally on the temporal side to a point where an object should be situated to give a retinal image on the point *o* if the direction of the eye were normal. In the normal position of the eye the fovea is opposite to the point O. To take its normal direction, the eye would require to be turned through an angle equal to the angle of strabismus; *f* comes to where *o* was, and *o* passes into *o'* (*fmo* = *omo'*; *fo* = *oo'*).

To find the projection of the image *o'*, the line *o'm* is prolonged, and the point O' in the plane of the object O gives us the position where the image *o* of the point O is projected. Given that the normal eye sees the object at O, and that the paralysed eye sees it at O', the distance OO' gives the distance between the two images. As the angle *o'mo* = *omf* and *omf* = angle of strabismus O$m\Omega$, the angle O*m*O' = angle of strabismus, and OO' = OΩ the tangent.[1]

[1] The lines towards which the visual impressions are projected do not cross at the centre of rotation as is made out on our figure, but the error is so small that we are justified in making the two points coincide for our requirements.

The same holds good with necessary modifications in paralytic deviation *laterally*. It becomes even clearer in *vertical* deviations, because, in these, the two eyes seen in profile cover each other and coincide. We can even

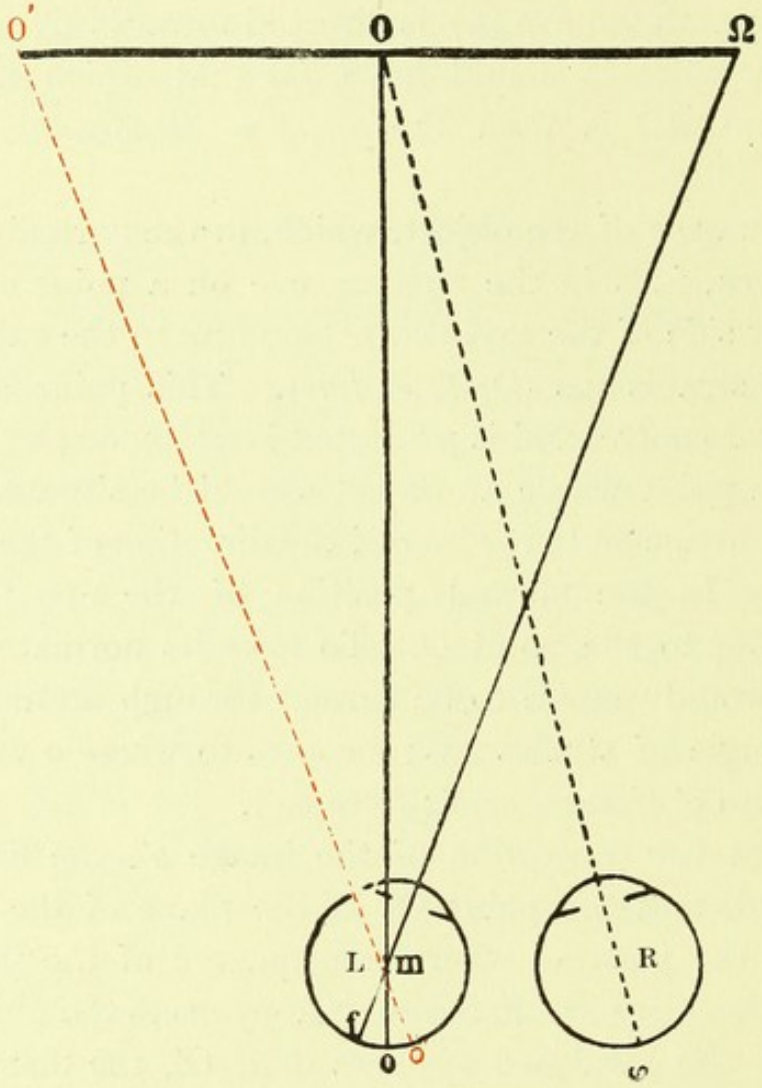


FIG. 9.

admit of this coincidence of the eyes for lateral deviations, because the examination is always made at so great a distance that the distance between the two eyes becomes negligible and they may be considered as the "double eye" of Hering.

For making the examination the simplest and most exact

method should be employed, which is found on our mural scale shown in Fig. 10.

The patient is seated facing the wall and at a known

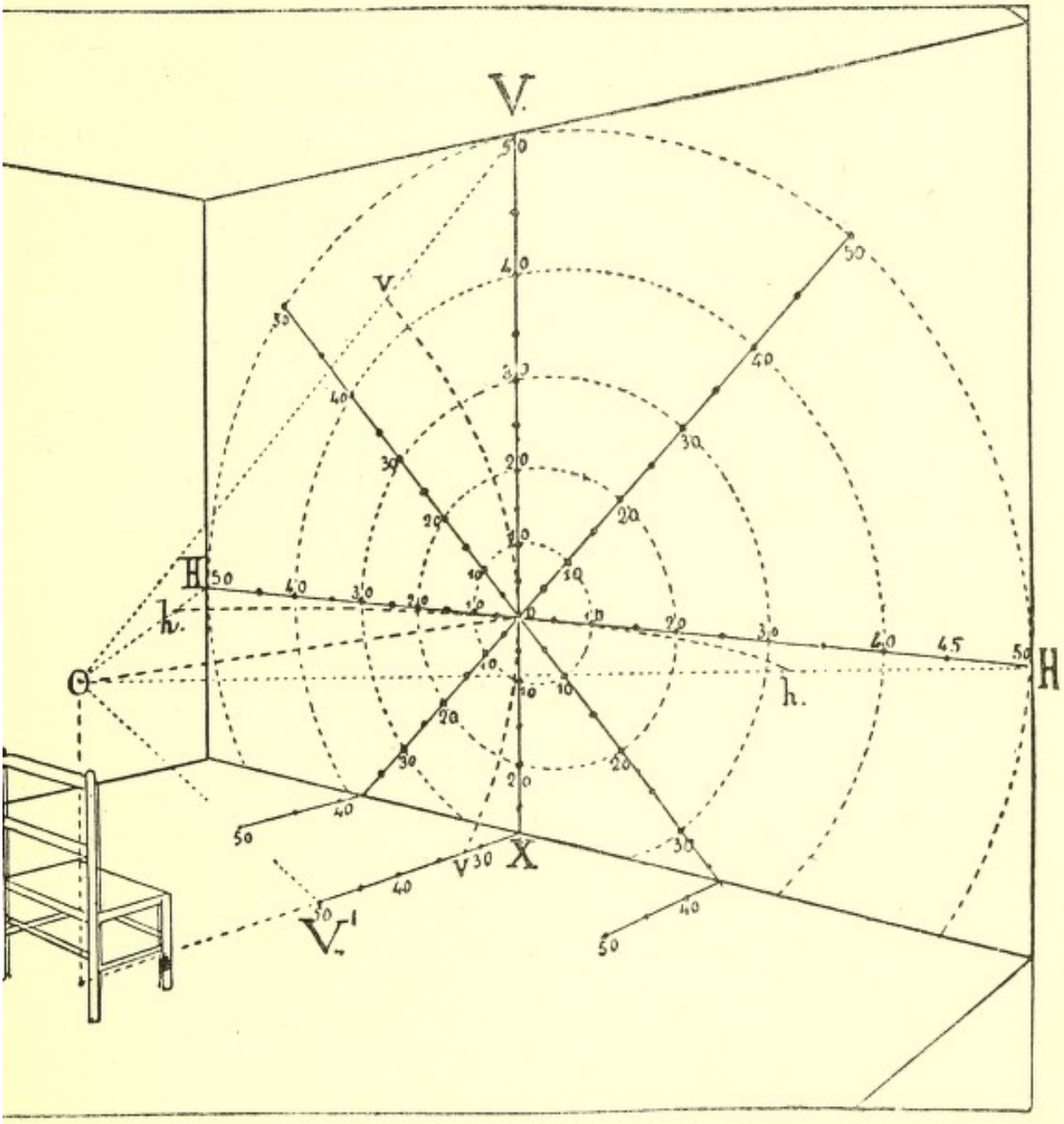


Fig. 10.

distance from it, and round the patient an imaginary sphere is constructed with O as centre.

At the point where the horizontal tangent from O meets the wall at right angles the object for fixation is placed, it

being a small candle, or better still a vertical luminous line, which can easily be made with an electric light, as the inclination of the false image is thus more exactly demonstrated. The points where the radii from the centre strike the wall are marked on the wall at 5° distances by means of brass-headed nails. Thus a star-shaped figure is formed round the point of fixation which gives the tangents at each 5°. The patient, wearing a red glass before one eye, projects the false image on this scale. The distance between the false image and the fixed object gives the amount of the angle of strabismus.

To simplify matters for anyone wanting to adopt our "mural scale," we give the values of the tangents for a radius of 225 cm., and one of 300 cms., *i. e.* the points of the wall which must be marked when the patient's head is 225 cm. or 300 cm. distant from the wall.

The point O is placed at 1 m. 20 cm. from the floor, which is the average distance of a seated person's head from the floor.

Tangents, *i. e.* distance from the point zero.

Angles.	r-225 cms.	r-300 cms.
5°	19·6 cm.	26 cm.
10	39·6	53
15	60	80
20	82	109
25	105	140
30	130	173
35	158	210
40	189	251
45	225	300
50	274	358
55	321	428

The meridans inclined at 45° meet the floor at 120 cm. from the point *x* (Fig. 10); that is, for a radius of 225 cm. between 30° and 40°.

From this point the tangents are as follows—

Angles.	r-225 cms.	r-300 cms.
25°		43 cms.
30	18	92
35	54	129
40	82	157
45	105	180
50	124	200
55	141	216

For the obliques—

30°		6 cms.
35		57
40	23	95
45	55	130
50	91	157

The value of the tangent is proportional to the distance of the patient from the wall. If, instead of 225 cms., he is seated at 450 cms., that is, at twice the distance, the figures of column I only correspond to half the number of degrees (*e. g.* 39 cm. only equal 5° instead of 10°), but the precision of the measurement is doubled.

Here we give the tangents for a radius of 1 metre. For the mural divisions we only require to multiply them by the distance at which the patient is placed from the wall expressed in metres.

Tangents.	Metres.
5°	0·087
10	0·176
15	0·268
20	0·364
25	0·466
30	0·577
35	0·701
40	0·839
45	1
50	1·192
55	1·428

The angle of strabismus and consequently the diplopia increases when the vision is directed towards the region of action of the paralysed muscle. Whilst the paralysed eye lags behind the normal eye, the false image corresponding to it seems to move more rapidly than that of the healthy eye.

In order to alter the direction of the eyes, we recommend that the position of the object of fixation (a lighted candle) be not varied, but be left on its place in the wall.

We produce the desired direction of the eyes by changing the position of the head, *e. g.* to make the eyes look to the *right*, the head is turned to the *left* (E. Landolt).

The more the direction of the eyes approaches the muscular plane of the *vertical* motors, the more the upward and downward rotatory effect of these muscles become manifest. At the same time their rotatory action round the antero-posterior axis and their effect in the lateral directions diminishes.

Conversely, the more the direction of the eyes deviates from this muscular plane, the more the vertical action

diminishes, and the rotation and abduction or adduction increases.

It follows that vertical diplopia, produced by paralysis of an *inferior* or *superior rectus, increases* in looking outwards (towards the *affected side*), and diminishes on looking towards the *healthy* side.

As the vertical motors are also rotators round the antero-posterior axis, the faulty direction of the eye (vertical divergence) which results from the paralysis of these muscles, becomes so much more manifest that the latter action comes into play. It is thus that the *inclination of the head towards the left shoulder* increases the defect of the vertical movement of the *right* rotators : left superior oblique and superior rectus, and right inferior oblique and inferior rectus.

The inclination of the head towards the right shoulder increases the defect of the vertical motility of the *left* rotators : right superior oblique and superior rectus, and left inferior oblique and inferior rectus.

The *inclination of the image* and the diplopia in the horizontal (crossed), due to paralysis of a vertical rectus, *decrease* in looking towards the *affected* side and *increase* in looking towards the *healthy* side.

Conversely, *vertical* diplopia in paralysis of an *oblique, increases* in looking towards the *nose* (*inwards*), that is, towards the *healthy* side, and *diminishes* on looking towards the *affected* side.

The *inclination* as well as the lateral diplopia (homonymous) increases in looking towards the *temple,* in other words, towards the *affected* side, and diminishes on looking towards the *healthy* side.

It is to be noted that the horizontal diplopia, which theoretically should accompany the vertical diplopia in paralysis of an upward rotator or of a downward rotator

is not an absolutely constant symptom. It is frequently absent in a paralysis of an oblique. Convergence and homonymous diplopia, which should result from paralysis of these muscles, may be neutralised and even over-corrected by the divergence, which the visual lines take up, when the eyes are in the state of rest—that is, when the fusion of the images of the two eyes becomes impossible, as is the case in paralysis of a vertical motor.

Position of the Head.

A patient with paralysis of an eye, usually turns his head in such a position that the binocular field of vision is, as much as possible, in front of him. To accomplish this he usually supplements the deficient rotation of the eye by rotation of the head.

As the image corresponding to the affected eye is in the direction of action of the paralysed muscle, we may say that the patient *turns his face towards the image.*

E. g. paralysis of a muscle rotating the eye to the right (right rectus lateralis, or left rectus medialis)—face directed towards the right.

Paralysis of a muscle rotating the eye to the left (left rectus lateralis, or right rectus medialis)—face directed towards the left.

The following exception to this rule must be carefully noted. It occurs in paralysis of muscles acting in the vertical which have at the same time a slight lateral action. The compensatory movement of lateral diplopia in such cases would increase the vertical diplopia, which is much more troublesome; it is on account of this that the patient turns his face in the opposite direction to that which it would otherwise take.

Thus the superior and inferior recti turn the eye slightly inwards (towards the nose). In spite of this, in paralysis

of these muscles the head is turned to the affected side, and not towards the healthy side. For the same reason, the patient turns his face towards the healthy side, and not towards the affected side when he is suffering from a paralysis of an oblique, though these muscles are abductors to a certain degree.

The increase of obliquity of the image which this movement produces is compensated for by the inclination of the head towards the shoulder of the healthy side in a case of paralysis of a superior rectus, or superior oblique, and towards the shoulder of the affected side in paralysis of an inferior rectus or inferior oblique. The inclination of the head on the shoulder at the same time diminishes the vertical divergence, *i. e.* the difference in height of the double images.

For upward and downward rotation and for torsion the rule previously given holds good :

Paralysis of an *upward* rotator (rectus superior, obliquus inferior)—face directed *upwards.*

Paralysis of a *downward* rotator (rectus inferior, obliquus superior)—face directed *downwards.*

Paralysis of a muscle that inclines the eye towards the *right* (superior rectus and superior oblique of left, and inferior rectus and inferior oblique of right)—inclination of the head towards the *right.*

Paralysis of a muscle that inclines the eye towards the *left* (inferior rectus and inferior oblique of the left, superior rectus and superior oblique of right)—inclination of the head to the *left.*

Briefly—Paralysis of a superior, that is to say of an inward rotator—head inclined towards the healthy side.

Paralysis of an inferior, that is to say, of an outward rotator—head inclined towards the affected side.

Excepting the lateral movement produced by the muscles acting in the vertical direction, the pathological position of the head corresponds to the normal action of the paralysed muscle : the *red lines* L R of the column VIII of our table indicate therefore also—except for the exceptions mentioned above—the position of the head when the corresponding muscle is paralysed.

Vertigo.

Vertigo is a symptom of paralysis of an ocular muscle which is very characteristic. It results from the apparent movement of surrounding objects, and more especially for those which are situated within the sphere of action of the paralysed muscle. Vertigo, moreover, is increased by the confusion which results from double vision. To avoid this movement of surrounding objects the patient is obliged to shut one eye. This is usually the eye which is paralysed, but if the patient habitually fixes with this eye, then he closes his healthy or normal eye and gradually learns to use the paralysed eye for going about.

False Projection or Localisation.

If the patient usually uses the non-paralysed eye for fixation, and if this eye be covered so as to oblige him to use the paralysed eye for fixation, instead of locating the fixed object in its real position he projects it towards the side to which the paralysed muscle should normally rotate the eye. He will estimate the situation of the object according to the amount of nerve energy which he requires to expend to direct the deviated eye towards that object. The more pronounced the paralysis, the greater is this expenditure of innervation. In reality the degree of false projection usually is in relation to the degree of paralysis.[1] This law may be somewhat modified in complete paralysis or in

[1] E. Landolt, *Arch. d'opht.*, xviii, May 1898.

paralysis of old standing, but chiefly when the paralysed eye is the eye which is habitually used for fixation.[1]

The measurements are made with Landolt's apparatus. It consists of a blackboard, placed vertically, and on which in investigating a case of a horizontal motor a white

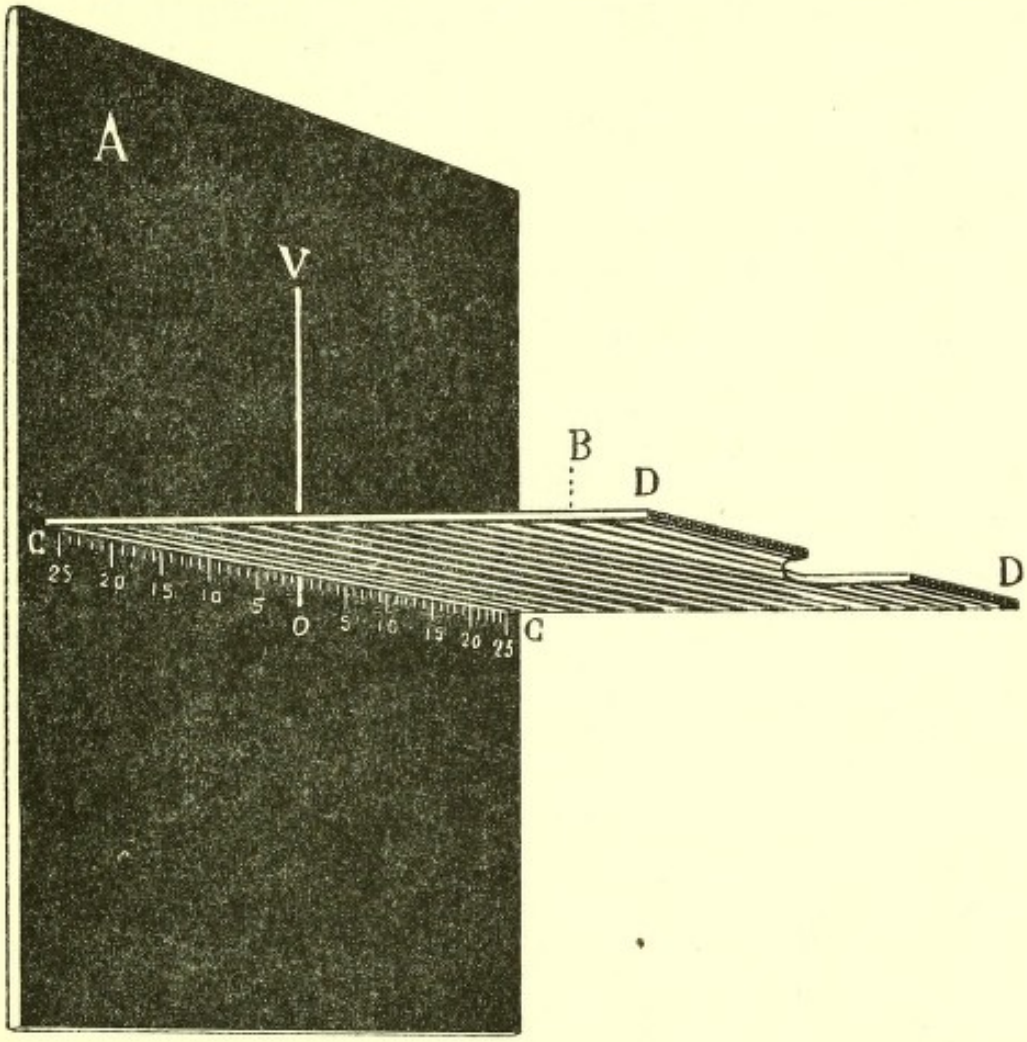


FIG. 11.

vertical line is drawn down the centre. A horizontal board is fixed to the vertical board by hinges, and has an indentation at its free border into which the patient's neck fits; thus once in position the patient cannot see the lower part of the blackboard. Just below the horizontal board is a horizontal white line which is divided to right and left into tangents by degrees for a radius of 70 cm. (distance of

[1] M. Landolt, *Arch. d'opht.*, xxii, Jan. 1902.

the eyes from the board). The patient is asked to touch quickly the lower continuation of the vertical line below the horizontal board, and the degree of error is the measurement of his false projection. To measure the false projection in paralysis of a vertical motor we use a vertical board and on it a vertical scale, the line of fixation being horizontal. The head must be kept looking straight at the board throughout, as this is important to avoid error, and the patient must rapidly touch the board with either hand alternately. This method has the advantage of giving an accurate estimation, as the patient cannot see his hand below the board, and cannot bring memory into play, thus his finger is guided by his muscular sense only.

Secondary Deviation.

If each eye be alternately covered in a person with ocular paralysis, it is noticed that the deviation is greater in the normal eye when covered, the affected eye fixing, than in the affected eye when covered while the healthy one fixes. This deviation which occurs in the healthy eye is called *Secondary Deviation.*

It may be permanent when the paralysed eye is the better eye as regards visual acuity, and the one which is usually used for fixation. If the patient continues to use this eye, the healthy eye will take the position opposite to the paralytic deviation which should be present, *i. e.* it will take up the position of secondary deviation, and project its retinal image accordingly.

The secondary deviation is thus represented by the red lines of column VIII of the table, if we transpose L for R and R for L. The black line then corresponds to the affected but fixing eye, the red line to the normal but deviated eye.

For example, if the left eye, affected with paralysis of the superior rectus fixes the object, the black line of column VIII L in the third division corresponds to the left eye, and the

red line to the right eye. The normal right eye will then be higher, slightly to the right, and also inclined towards the right.

The normal eye deviated in this manner projects its image in the opposite direction; thus, in the example given the image is projected downwards and to the left. In other words, when the healthy eye is situated in the position of secondary deviation, its image corresponds to the position taken by the paralysed eye when the normal eye is used for fixation.

There are exceptions to these rules in lateral deviation and inclination of the eyes and the images in such cases, but in horizontal and vertical diplopia, which are by far the most important forms, these rules are correct without exception.

Briefly we can say: the phenomena following paralysis of an ocular muscle are manifest both in the normal direction of action of the muscle and in the opposite direction.

Thus we have—

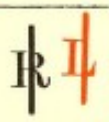
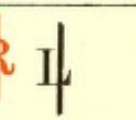
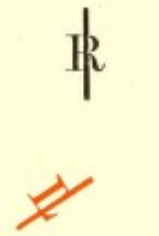
In the direction of the **Physiological** Action of the Muscle—	In the **opposite** direction to the Physiological Action of the Muscle—
(*a*) Diminished excursion of the eye (limitation of the field of fixation).	(*a*) Deviation of the eye.
(*b*) Appearance and increase of diplopia.	(*b*) Decrease of diplopia and even fusion of the double images.
(*c*) Apparent movement of surrounding objects.	(*c*) Projection of the retinal images of the secondarily deviated non-paralysed eye when the paralysed eye is used for fixation.
(*d*) False projection.	
(*e*) The direction of the face and inclination of the head.	
(*f*) Secondary deviation of the non-paralysed eye.	

These symptoms may be made more apparent in changing the direction of vision by rotating the patient's head.

I. The Paralysed Muscle.	II. Deviation of the Eye (Strabismus).	III. False Image of the Affected Eye (Diplopia) (Red line of Col. VIII).	IV. Limitation of Movement and False Projection (Red line of Col. VIII).	Increase of D (Red line
Rectus Lateralis	Towards the normal side; convergent strabismus.	On the affected side; homonymous diplopia.	Towards the affected side.	On lookin affected s
Rectus Medialis	Towards the affected side; divergent strabismus.	On the normal side; crossed diplopia.	Towards the normal side.	On lookin normal s
Rectus Superior	Downwards; somewhat towards the affected side; rotated to the temporal side; strabismus dorsum vergens with slight divergence.	Higher; on the normal side, and inclined towards the normal side; vertical and slightly crossed diplopia.	Upwards and somewhat towards the normal side.	The differ is increa and towa side; an tion of th the *affe* horizonta the incli creased normal s
Rectus Inferior	Upwards; somewhat towards the affected side; rotated towards the nose; strabismus sursum vergens with slight divergence.	Lower; on the normal side, and inclined towards the affected side; vertical and slight crossed diplopia.	Downwards, and somewhat towards the normal side.	Difference creases d towards side; an tion of wards th the horiz and incli towards t
Obliquus Superior	Upwards; somewhat towards the normal side; rotated to the temporal side; strabismus sursum vergens and slight convergence.	Lower; on the affected side, and inclined towards the normal side; vertical and slight homonymous diplopia.	Downwards, and somewhat towards the affected side.	The differ increases and towa side; an tion of th the *affe* horizonta inclinatio wards th
Obliquus Inferior.	Downwards; somewhat towards the normal side; rotated towards the nose; strabismus deorsum vergens and slight convergence.	Higher; on the affected side, and inclined towards the affected side; vertical and slight homonymous diplopia.	Upwards and somewhat towards the affected side.	The differ increases towards side; an tion of th the *heal* horizonta inclinatio wards the

[1] In secondary deviation the only reliable test is the difference in height between the eyes, *i.e.* the images; the differe

[2] The red lines of Column VIII, which show the physiological action of the vertical motors, as well as their ac of the eye would occupy after a rotation of 40° around the axis of the muscle referred to. We have obtained their By comparing the red and black lines, we obtain the exact amount of rotation around the horizontal, vertical an

...on and of ...VIII).	VI. Position of Head (Direction of Face) (Red line of Col. VIII).	VII. Secondary Deviation of Normal Eye.	VIII. Diplopia.[2] The red line corresponds to the false image of the paralysed eye; the black of the image of the normal fixing eye.	
			L.	R.
...ards the	Towards the affected side, without inclination.	Towards the affected side; convergent strabismus; homonymous diplopia.		
...ards the	Towards the normal side, without inclination.	Towards the normal side; divergent strabismus; crossed diplopia.		
...n height ...upwards ...affected ...inclina- ...towards ...ide, the ...opia and ...are in- ...rds the	Outwards; towards the affected side, and inclined towards the shoulder of the normal side.	Upwards and towards the normal side; rotated towards the normal side; vertical and slight crossed diplopia; the image of the normal eye lower and inclined towards the affected side.[1]		
...eight in- ...ards and *affected* ...inclina- ...head to- *...thy* side, ...diplopia ...increase ...mal side.	Downwards; towards the affected side, and inclined towards the shoulder of the normal side.	Downwards and towards the normal side; rotated towards the affected side; vertical and slight crossed diplopia; image of normal eye higher and inclined towards the normal side.[1]		
...n height ...wnwards ...e normal ...inclina- ...towards ...ide, the ...opia and ...rease to- ...ted side.	Downwards; towards the normal side, and inclined towards the shoulder of the normal side.	Downwards and towards the affected side; rotated towards the normal side; vertical and slight homonymous diplopia; image of normal eye higher and inclined towards the affected side.[1]		
...n height ...rds and ...normal ...inclina- ...towards ...ide, the ...pia and ...rease to- ...ted side.	Outwards; towards the normal side, and inclined towards the shoulder of the affected side.	Upwards and towards the affected side; rotated towards the affected side; vertical and slight homonymous diplopia; image of normal eye lower and inclined towards the normal side.[1]		

...he horizontal plane and the inclination of the images does not always correspond with what would be expected. ...paralysis, are not drawn at haphazard; they correspond exactly to the position which the vertical meridian ...by calculation, and controlled it experimentally with our ophthalmotrope.
...-posterior axis produced by each muscle.

Symptoms of total paralysis of the Oculomotor Nerve.

Ptosis.—Mydriasis.—Paralysis of the *sphincter of the pupil* and of the *ciliary muscle*, i. e. paralysis of accommodation. Paralysis of the *rectus medialis*, *rectus superior* and *inferior*, and of the *inferior oblique*.

Divergent strabismus, often with slight downward rotation, with *inclination* towards the *normal* side.

Therefore, *crossed diplopia*.

The image of the affected eye is *inclined* towards the *affected side*, and is sometimes slightly higher than that of the one on the normal side.

Rotation of the eye towards the *normal* side, *upwards* and *downwards*, is limited.

Inclination of the image is more accentuated in looking towards the *normal* side.

Increase of *horizontal* diplopia in directing the vision towards the normal side, and increase of both upward and downward *vertical* diplopia. On looking upwards the image of the *affected* eye is the *higher*, while on looking downwards it is the *lower*. The inclination of the image increases on looking towards the *normal* side.

When paralysis of the oculomotor nerve is very marked, the *rectus lateralis* of the same side frequently has its action slightly limited; this is shown by the fact that the field of fixation is more or less limited on the temporal side. The other eye also may show some other oculomotor disturbances.

Fields of Fixation in different cases of Paralyses.

The dotted curve indicates a normal field of fixation of minimum extent.

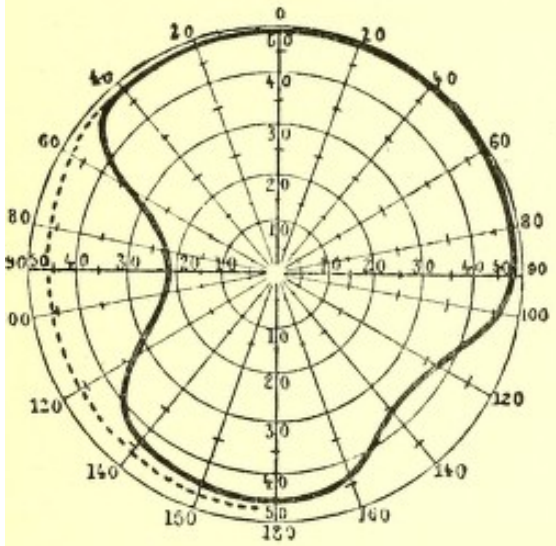


FIG. 12.—Field of fixation of a *left* eye affected with *paralysis* of the *lateral rectus*. (Strab. conv. 8°).

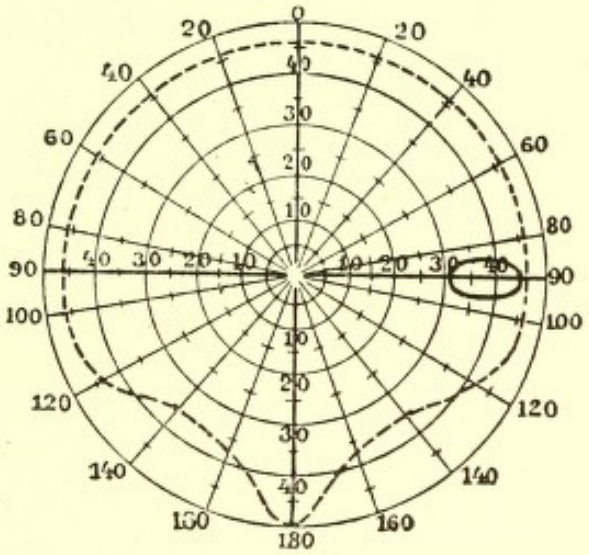


FIG. 13.—*Binocular* field of fixation of a patient suffering from *paralysis* of the *rectus lateralis of the left eye*. Dotted line corresponds with normal *binocular* field of fixation.

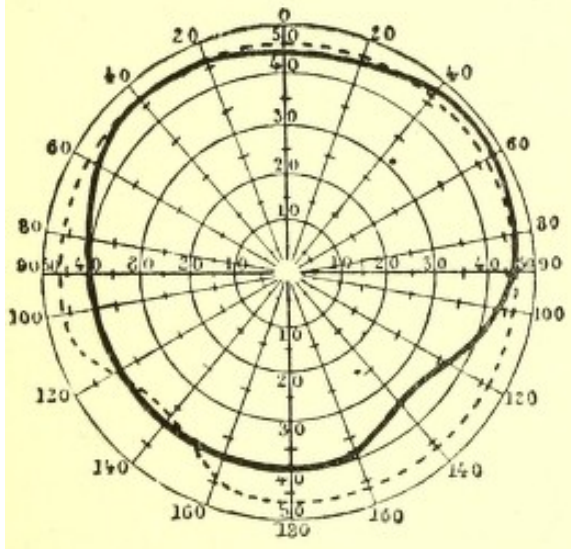


FIG. 14.—Field of fixation of a *right* eye affected with paresis of the *obliquus superior*.

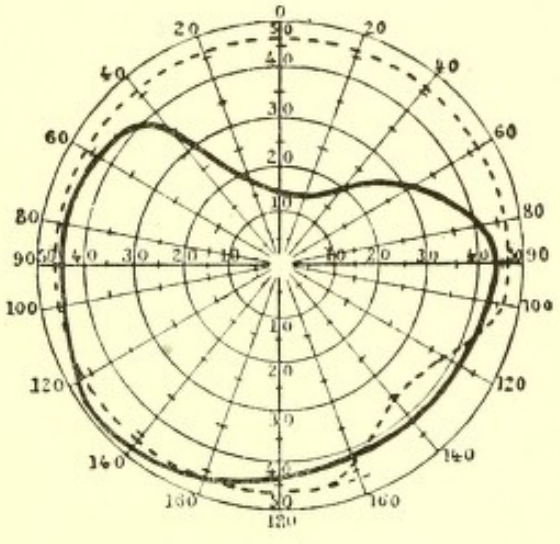


FIG. 15.—Field of fixation of a *left* eye affected with paresis of the *obliquus inferior*.

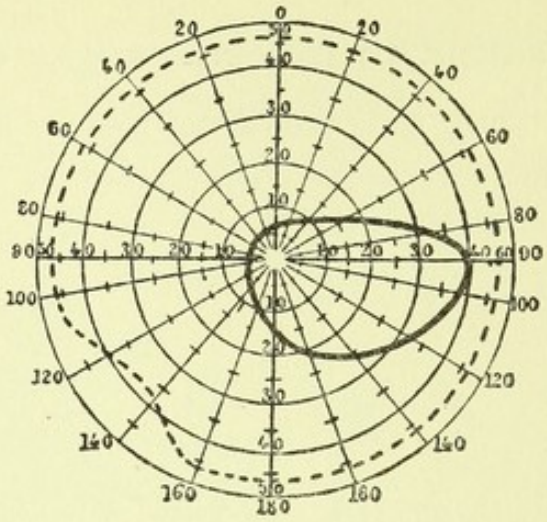


FIG. 16.—Field of fixation of a *right* eye affected with paralysis of the *oculomotor nerve*.

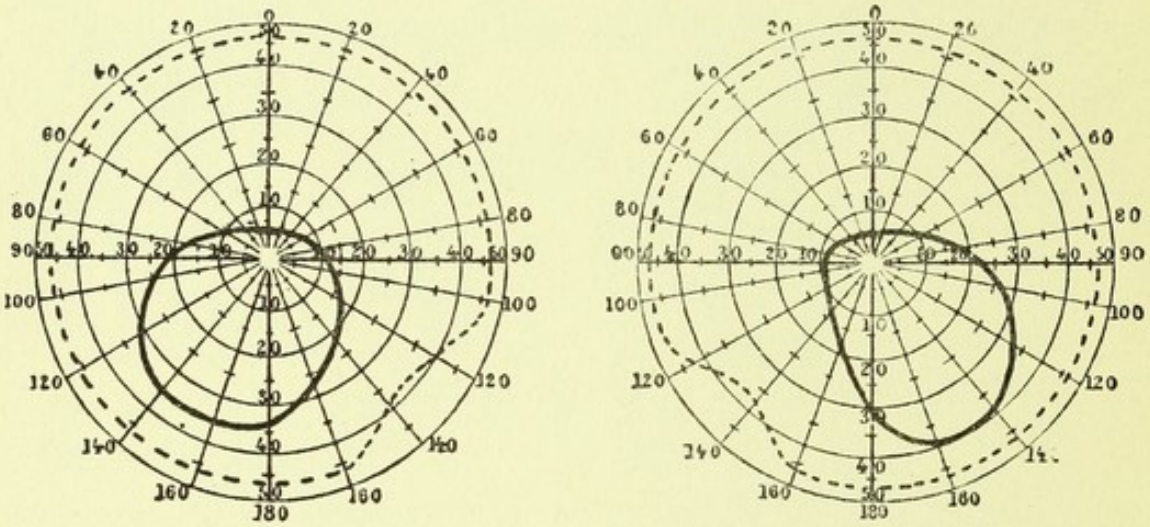


FIG. 17.—Fields of fixation of bilateral paralysis of the oculomotor nerve.

Spasms of the Ocular Muscles.

Simple spasm of an ocular muscle must not be confounded with the contracture which frequently develops in the course of an ocular muscle paralysis of long standing. Spasm is a contraction which ceases with enervation, whilst in contracture the muscle becomes modified in its structure and is permanently shortened. Contracture is thus a secondary lesion, whilst spasm is a primary one.

The symptoms of spasm of an ocular muscle are exactly opposite to those produced by paralysis of that muscle, and are similar to those which paralysis of the antagonistic muscle would produce. We must not forget that the medial and lateral recti are each opposed by a single muscle, whereas each of the vertical rotators has at least two corresponding antagonistic muscles.

Spasm is differentiated from paralysis of the opposed muscle by a lesser uniformity. Muscular cramp or spasm cannot remain constantly at the same degree for days or weeks, and can be overcome by narcosis. At intervals it disappears, or is at times more pronounced than at others. This variation in degree can be demonstrated not only in the variations in the angle of strabismus, but also in those of the excursions of the eye affected with spasm: in paralysis the angle and excursions are more constant or gradually modified according to the improvement or development of the illness.

Tonic spasm of a single muscle is seemingly very rare. In making a diagnosis the general condition of the patient must receive special attention.

Spasms of the motor functions and of the associated movements of the eyes are met with much more frequently: lateral, upward and downward rotation are all encountered, but chiefly convergence, and less frequently divergence.

We shall discuss these in the chapter on affections of *associated* movements.

Some spasms of this class when affecting the symmetrical movements resemble a concomitant strabismus and are met with in spastic rigidity of the limbs (Little's disease), and also in hysteria. They do not come under the heading of the second form of strabismus, but under the third form (p. 62).

Clonic spasm may accompany certain paralysis of isolated muscles, especially during their period of recovery.

Thus, in nuclear affections, we may meet with a

contracture of the levator palpebræ (which was previously paralysed) when other ocular muscles become innervated.[1] Possibly Graefe's sign in Graves's disease may be considered as a spasm of the levators of the lids; this sign, as is known, depends on the fact that the upper lids are not lowered when the patient looks down towards the ground.

In 1882 I was consulted by the celebrated oculist Desmarres on account of a rhythmic spasm of his right *superior oblique* which had been in a state of paresis as the result of a cerebral hæmorrhage with right-sided hemiplegia and aphasia. As the two images of the diplopia were close together, distortion and apparent movements of objects resulted which was very troublesome. In his many years of large practice he had never seen an analogous case, and I myself have never come across such a case again.

Such nystagmic tremors are observed in many different diseases of the nervous system. They can, however, be produced simply by overwork or weakness of the muscles, and are especially observed when the eyes are rotated into their extreme limit of excursion laterally or vertically (see p. 67).

DIAGNOSIS OF OCULAR PARALYSIS.

Diagnosis in a case of ocular paralysis is most easily made by means of diplopia if it be present. To make use of this the patient is seated facing a wall on which a lighted candle is fixed at the height of the patient's eyes. A single vertical incandescent wire is still better than a candle, as with it the inclination of the image can be more accurately judged.[2]

[1] Wilbrand and Saenger, *Neurologie des Auges*, i, p. 77.

[2] If the wall has our tangent scale marked out on it (*vide* p. 33), the distance of the patient's head from the wall should be equal to the radius of the division. Thus the *quantitative* as well as the qualitative diagnosis of the deviation can be made; in other words, we can measure the angle of strabismus.

In front of one of the patient's eyes we place a red coloured glass. The head and the body must be kept perfectly straight facing the wall.

First, we note the nature of the diplopia—*homonymous*, *crossed* or *vertical*. If it be mixed, that is, if the images are not on the same level or not exactly one above the other, we first direct our attention to the most pronounced character of deviation; to the horizontal if the images are further apart in the horizontal, and to the vertical deviation if they are further apart in the vertical.

To diagnose a muscular paralysis, we must first determine *which is the paralysed eye*. In doing this our first law helps us. "The affected eye is recognised by the fact that the diplopia increases when the direction of vision is turned towards its image."

To change the direction of vision the object of fixation is not moved, as we have said previously; it remains fixed to the wall, but we turn the patient's head in the opposite direction to the direction of vision we wish to obtain: to direct the eyes to the left we turn the patient's head (or face) to the right; to make him look upwards the head is lowered, and vice versa (see p. 36).

For example: the image of the *left* eye is the *lower*, that of the *right higher*. If the patient's head is moved backwards to make him look *downwards*, the two images seem to separate from one another. Therefore the *left* eye is the one affected. (In lowering the head, that is to say, when the vision is directed upwards towards that of the right eye, the images approach one another.)

The affected eye being thus determined we shall find the *paralysed muscle* by our second law: "The paralysed muscle is the one which would have placed the eye in the position and direction of the false image." These two laws suffice for finding the affected muscle acting in the horizontal plane.

The same is the case in vertical diplopia when, besides difference in height, there is well-marked lateral separation and inclination.

If in our example the image of the affected left eye is not only lower but also deviated to the temporal side (homonymous diplopia) and inclined towards the inner side, we have clearly to do with paralysis of the *superior oblique*. This muscle turns the eye downwards, outwards, and the vertical meridian is inclined inwards.

Frequently the patient cannot give precise statements as to the lateral deviation, and especially regarding the obliquity of the double images.[1]

It sometimes happens that the horizontal diplopia is the contrary to that which paralysis of the muscle would lead one to expect. Thus, instead of being homonymous in paralysis of an oblique it may sometimes be crossed. This may occur when the direction of rest or equilibrium of the eyes (p. 25), is divergence. As the eyes are, on account of the paralysis, incapable of fusion of their images, they diverge instead of converging, as is the case in paralysis of an oblique when the direction of rest is parallelism.

From the patient's replies we can only conclude that there is paralysis of an upward or downward rotator.

As each eye has two upward and two downward rotators,

[1] The inclination is much more distinct when the object of fixation is a luminous wire instead of a small flame. Usually, however, lateral and oblique deviation which accompany paralysis of a vertical rotator are not well marked in the primary position of the eyes. The images of column VIII (p. 45) represent a rotation of 40° round the axis of the vertical rotators. In reality such a pronounced diplopia never occurs. If we reduce this to 10°, which is already a considerable amount, that is reducing the vertical and horizontal distances as well as the inclination to a fourth, the two lateral and oblique deviations will become scarcely perceptible. The usual diagrams, which are absolutely schematic for representing diplopia, greatly exaggerate the horizontal and oblique directions in relation to the vertical, and thus give a very erroneous idea of the real symptoms.

we must resort to another method for finding out whether the case be one of paralysis of an oblique or a rectus; in our example whether it is the *superior oblique* or *inferior rectus*.

To make a differential diagnosis between paralysis of an oblique and a rectus (superior or inferior), it is sufficient to represent the direction of the muscular plane of the motors in the vertical (Fig. 1) : towards the temple, laterally for the recti, inwards for the obliques. The more the line of vision approaches the normal direction of action of these muscles, the more their vertical action becomes manifest, and the greater the increase in vertical diplopia when they are paralysed; on the other hand, the lateral action and that round the antero-posterior axis diminishes, and proportionately so does the horizontal diplopia and inclination of the image (the contrary, of course, is produced in the opposite direction to the muscular plane).

If, then, by rotation of the head to the left, the direction of vision is turned towards the right and the vertical diplopia increases, we are dealing with a paralysis of an *oblique* of the left eye, or the *rectus* superior or inferior of the right eye. On the contrary, vertical diplopia, which increases in vision towards the left, is caused by paralysis of a *rectus* of the left eye or an *oblique* of the right eye. Or, again :

Increase in vertical diplopia in vision towards *the side of the normal eye* is caused by paralysis of an *oblique*.

Increase in diplopia in vision towards the *affected* side is caused by paralysis of a *rectus*.

Let us again take our example (paralysis of a *downward rotator* of the *left* eye). If we turn the patient's head towards the left, so that he must direct his vision towards the right to fix the object, and if the vertical diplopia increases, we diagnose paralysis of the *superior oblique*.

In directing his vision to the opposite side the images

approach each other in the vertical, but separate in the horizontal. By this method we frequently can make manifest a horizontal diplopia and an inclination of the image which would be latent if the patient looked straight in front of him.

The difference in height of the double images (vertical divergence) is also influenced by the inclination of the head on the shoulder. Thus—

Increase in vertical diplopia produced by inclination of the head towards the *left* shoulder indicates paresis of a rotator towards the *right*.

Left *superior rectus* or *superior oblique*.

Right *inferior rectus* or *inferior oblique*.

Increase of *vertical diplopia* produced by inclination of the head on the *right shoulder* indicates paresis of a *left* rotator.

Right *superior oblique* or *superior rectus*.

Left *inferior oblique* or *inferior rectus*.

When, in a case of muscular paralysis, the subjective symptoms, notably the *diplopia*, are wanting, the diagnosis is based on the *objective symptoms* only. Thus we can make use of the *position of the patient's head*. Now as the patient has but one head and two eyes, this symptom can only aid us in determining the kind of paralysis with which we are dealing. Thus—

The face is turned to the left = paralysis of muscles rotating the eye to the left, *left rectus lateralis, right rectus medialis*; also in paralysis of the left recti superior and inferior and the right superior and inferior oblique, though they have a slight action towards the right (because in this manner the patient diminishes the vertical part of his diplopia).

Face turned towards the right = paralysis of a muscle rotating the eye towards the right, *left rectus medialis, right rectus lateralis*, or the right recti superior and inferior,

or the left superior or inferior oblique though they have a slight rotatory action towards the left.

Face directed downwards = paralysis of a downward rotator, superior oblique, or inferior rectus.

Face directed upwards = paralysis of an upward rotator, inferior oblique, or rectus superior.

Inclination of the head towards the *left* shoulder = paralysis of a rotator towards the left, *left inferior rectus or obliquus; right superior rectus or obliquus.*

Inclination of the head towards the *right* shoulder = paralysis of a rotator towards the right, *left superior rectus or obliquus; right inferior rectus or obliquus.*

Objective diagnosis is made most surely by following the same method as we have indicated for subjective diagnosis. First we ask, *Which is the affected eye?* and then, *Which is the paralysed muscle?*

(1) As regards the first question we must note that the affected eye is not always the deviated eye, but that which makes the lesser corrective movement towards the fixation object when the other eye is covered. If the patient habitually fixes with the paralysed eye, then he continues to fix with that eye, and thus it is the other eye which assumes the secondary deviation.

(2) To determine which muscle is the paralysed muscle the excursions of the eye are examined. They are necessarily limited in the sphere of action of the weakened muscle. The objective symptoms of the paralysis result then directly from what we have said (p. 43) regarding their normal action, and also from the red lines of our synoptic table, which represent the effect of the normal action of each muscle on the direction of the eye.

The limitation of the excursion of the eye is just the reverse of the normal action of the muscle. The only exception is made by the horizontal component of the angle

of strabismus due to paralysis of a vertical motor, as we have already explained.

All that we have said (p. 51) regarding the analysis of the subjective symptoms of an ocular paralysis applies equally to the objective symptoms; we need only remind ourselves that the direction of the eye is opposite to that of its image, the deviation the opposite of the diplopia, *e. g.* convergent strabismus = homonymous diplopia; divergent strabismus = crossed diplopia. Thus our law on p. 51 is transformed into: The paralysed muscle is that whose defective action brings about the deviation of the eye.

To find most rapidly which is the paralysed muscle we take into account the following facts—

Convergent strabismus = paralysis of an externus rectus
Divergent ,, ,, ,, internus ,,

Vertical strabismus = paralysis of a muscle for upward or downward rotation. Limitation of excursion of an eye downwards (upward strabismus) = paralysis of a downward rotator. If the strabismus increases in looking towards the healthy side and with inclination of the head towards the shoulder of the affected side, the *superior oblique* is affected. If the strabismus increases in looking towards the affected side, and is accompanied by inclination of the head towards the shoulder of the healthy side, then the inferior rectus is affected.

Limitation of the excursion upwards (strabismus deorsum vergens) = paralysis of an upward rotator. If the strabismus increases in looking towards the healthy side, or with inclination of the head on the shoulder of the healthy side = inferior oblique. If the strabismus increases in looking towards the affected side and with inclination of the head on the shoulder of the affected side = superior rectus.

In the newborn who does not yet fix objects, one can recognise paralysis of a horizontal motor by the following method shown by Bartels. When a newborn child is rotated laterally, for example, to the right, the eyes at first remain directed to the left, and then are rapidly turned to the right if the rotation of the body continues. If this rotation is stopped the eyes again turn in the opposite direction, that is, to the left in our example. If this movement of the eyes which normally accompanies the rotation of the body is wanting in one or other of the eyes, one can conclude that there is paralysis of the muscle which should have brought about the rotation.

II. STRABISMUS RESULTING FROM DEFECTIVE BINOCULAR VISION.

(Concomitant Strabismus.)[1]

Binocular vision is the only sure factor which controls the correct relative direction of the eyes. If it be absent, or if the desire for binocular vision is only slightly developed, the relative directions of the eyes are controlled by other influences. Amongst these the most important are the relationship between accommodation and convergence, and the position of rest of the eyes.

In young hypermetropes, or even in patients who are not hypermetropic but have merely feeble accommodation, the mere effort of accommodation may produce convergent

[1] The expression "concomitant strabismus" is far from being the correct word to express this condition. It dates from a period when the cause of this form of strabismus was not properly understood. We now know that it neither depends on a fault in the muscle structure nor on a paralysis of its nerve; in other words, there is no organic lesion. We might differentiate it by calling it *functional* strabismus from *organic* strabismus due to lesion in the oculomotor system of the eyes.

squint, if the necessity for binocular vision does not produce a well-balanced association between convergence and accommodation.

Conversely, myopes of high degree, who require little if any accommodation, or patients who, though not myopic, are deprived of binocular vision for one reason or another, develop a *divergent strabismus*. They do not make the effort which positive convergence necessitates, and thus their eyes assume the reciprocal direction that is the most convenient position for them to take up, and in the great majority of cases this direction is divergence.

In concomitant strabismus the abnormal directions of the eyes almost always occur in the horizontal plane. Convergence, whether positive or negative, usually lies in the plane passing through the two centres of rotation. Only in very pronounced cases a deviation in height is present as well as the horizontal deviation. A *converging* eye is usually directed a little upwards, a *diverging* one slightly downwards.[1]

In concomitant strabismus the *visual* association between the two eyes does not generally exist. They see independently of each other, as two separate organs, but they cannot move independently one from the other. Their movements obey the laws of motion of the *double eye*, only the position of equilibrium of the eyes is no longer parallelism.

As we have already seen from its ætiology, concomitant strabismus always involves the two eyes to the same degree: it is not one or other eye which squints, but the error of the relative direction of the eyes becomes manifest in one only, because necessarily one of the eyes must be directed towards the object which attracts the attention of the individual. Therefore, instead of speaking of convergent

[1] E. Landolt, *L'Etiologie du strabisme*: *Arch. d'opht.*, xvii, p. 74. 1897.

strabismus of the left eye, one should say: convergent strabismus, the right eye fixing.[1]

Differential diagnosis of concomitant and paralytic Strabismus.

If the ætiology of strabismus is borne in mind, the differential diagnosis between concomitant and paralytic strabismus should not present any difficulty.

Paralytic strabismus usually appears rapidly, concomitant strabismus comes on slowly.

Concomitant convergent strabismus usually develops during infancy, and usually in hypermetropes. Accommodation influences concomitant strabismus to a very high degree, whereas it has very little influence upon paralytic convergent strabismus.

Convergent paralytic strabismus (paralysis of the rectus lateralis) is not so rare in children as is usually supposed, although it occurs much more frequently in adults. We need only remind the reader of its causes—syphilis, diabetes, rheumatism, etc., and the various cerebral diseases to which adults are much more liable than children.

Concomitant divergent strabismus may appear at any period of life as the sequence of loss of binocular vision.

In *concomitant strabismus* we do not find spontaneous diplopia corresponding exactly in position and degree to the deviation, which is so characteristic a feature in paralysis.

Diplopia, which can be made apparent in some cases of concomitant strabismus, differs from that in paralytic strabismus, and corresponds more to an alternating vision. The patient usually cannot indicate with certainty the reciprocal position of the two images.

It is true, that even in *paralytic* strabismus, diplopia

[1] E. Landolt, *L'Étiologie du strabisme: Arch. d'opht.*, xxii.

may be absent in cases where binocular vision has not previously existed, or where the deviation is one of very old standing; even in this last case we can, as will be seen later, reawaken this diplopia.

No matter which eye is covered, the degree of deviation is usually the same for each eye in *concomitant* strabismus, whereas in *paralytic* strabismus the deviation is greater when the patient fixes with his paralysed eye, than when he fixes with his normal eye, as we have already explained. In other words, *secondary deviation*, which is characteristic of paralytic strabismus, is absent in non-paralytic strabismus. Similarly false projection also is absent in *concomitant strabismus*.

As the seat of concomitant strabismus is not in one eye, but in both at the same time, so the after results are also found in both eyes. On both sides limitation of the field of fixation gradually develops, which is *temporal* in *convergent*, *nasal* in *divergent* strabismus. This limitation is usually more pronounced in the constantly deviated eye than in the eye used for vision.

In paralytic strabismus the limitation of movement corresponds to the sphere of action of the paralysed muscle, and the paralysis usually exists in one eye only. Even if both eyes be affected, the fields of fixation are not easily confounded with those in a case of concomitant strabismus. The fields of fixation in a case of *double paralysis of the recti externi* will differ on careful investigation from those of a *concomitant convergent strabismus:* on the temporal side they show a definite notch (Fig. 12), whereas in concomitant convergent strabismus there is merely a diminution of the convexity or a flattening on the outer side [1] (p. 61, Fig. 18).

Difficulty may be found in making a differential diagnosis

[1] E. Landolt, *Arch. d'opht.*, 1881.

between paralytic strabismus and concomitant strabismus in old-standing cases. The absence of diplopia does not

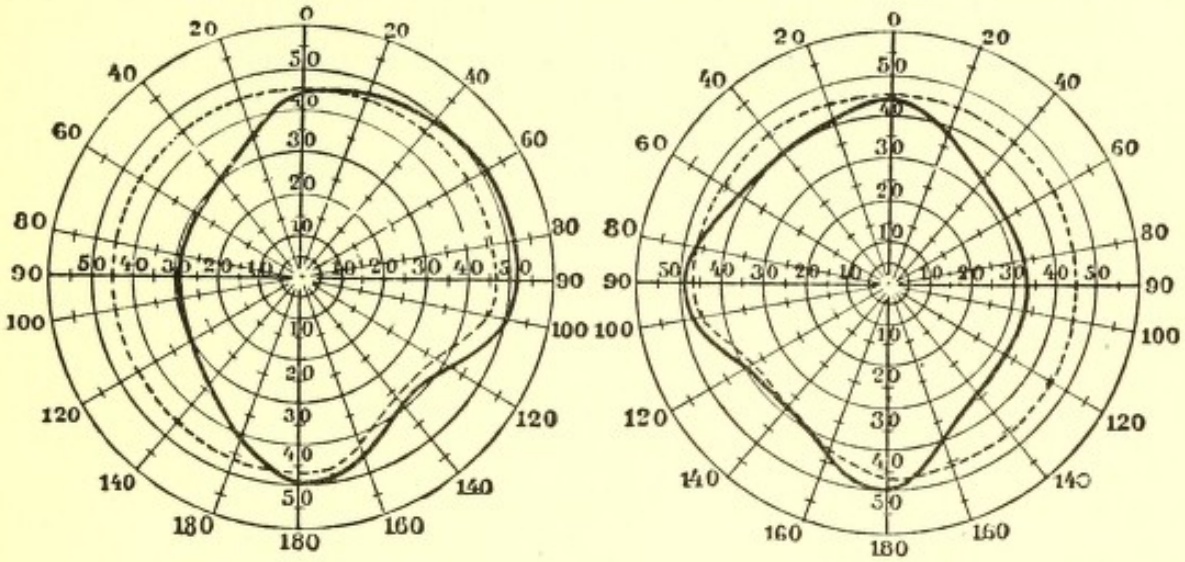


FIG. 18.—(Continuous line.) Fields of fixation of a severe concomitant convergent strabismus. Dotted line = normal fields of fixation of minimal extent.

suffice to exclude muscular paralysis. Patients with paralytic strabismus gradually learn to exclude the image

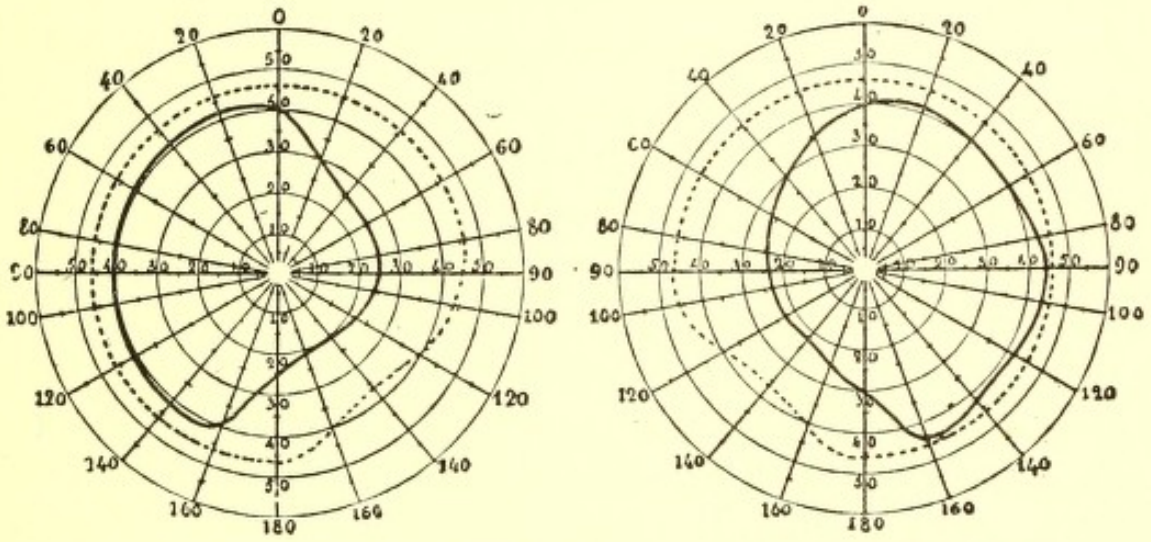


FIG. 19.—Fields of fixation of a severe case of myopia with relative concomitant divergent strabismus.

of the deviated eye from their consciousness, and thus avoid diplopia. Diplopia, however, may be reawakened with a little patience and in a characteristic fashion, especially by using a prism placed perpendicularly to the deviation.

The image of the fixed object thus falls, in the deviated eye, on a portion of the retina on which impressions are still perceived. If the deviated eye has very bad visual acuity we make use of a very brilliant source of light, the intensity of which is modified for the healthy eye by placing a smoked glass before it.

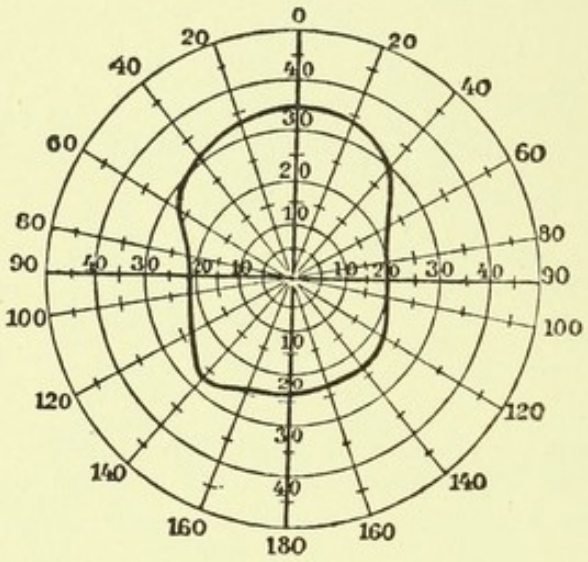


FIG. 20.—*Binocular* field of fixation of the patient of Fig. 19.

In eyes thus deviated for many years, the muscular structure becomes altered, and this modifies the fields of fixation in a somewhat peculiar manner.

If, therefore, the ætiology and all the diagnostic methods are gone into thoroughly, a definite conclusion may usually be arrived at, as the two forms of strabismus, by their nature alone, are so very different. There may be difficulty in diagnosing a case in which concomitant strabismus is combined with paralytic strabismus.

III. STRABISMUS RESULTING FROM A LESION OF THE CENTRES GOVERNING THE SYMMETRICAL OR ASSOCIATED MOVEMENTS OF THE EYES.

Although the governing centres of convergence and divergence are involved in concomitant strabismus, whether convergent or divergent, still the primary cause of these forms of strabismus do not always consist in a lesion

of these centres. Non-paralytic convergent strabismus caused by a spasm or exaggerated innervation of convergence usually results from spasm of accommodation, which in turn is the result of faulty refraction either static or dynamic.

Similarly in cases of *divergent* concomitant strabismus we usually have not to deal with a paralysis of convergence, but merely with an inactivity or weakness of convergence which may result in deficiency or even complete obliteration of this function. But in both cases, whether convergent or divergent concomitant strabismus, the primary cause lies in an insufficiency of the development of binocular vision.

The third form of strabismus originates quite independently of refraction, visual acuity, or binocular vision, but results from a primary lesion of the centres.

If the centre for the symmetrical movements is the seat of the lesion, the strabismus is always accompanied by *diplopia*; *homonymous* in spasm of convergence or in paralysis of divergence, *crossed* in paralysis of convergence or in a spasm of divergence. Here already lies a fundamental difference between this *central* form and concomitant strabismus.

If we examine the fields of fixation, after having previously diminished the spasm of convergence as much as possible by paralysing the accommodation with atropine, they are usually found to be normal.

This character again differentiates this form of strabismus from concomitant strabismus, and also from paralytic strabismus. False projection and secondary deviation also are both absent.

The diplopia in an affection of the associated movements cannot be confounded with that in the case of paralysis of a lateral or medial rectus; in the latter case it increases markedly in one direction and diminishes in the other; in

an affection of the associated movements it increases in both directions.

Simultaneous and isolated paralysis of the two *medial recti* is scarcely ever met with, whereas it is not rare to find the two laterals simultaneously paralysed. It is accompanied by temporal limitation of the excursion of the eyes and by homonymous diplopia. The cause and seat of the paralysis is diagnosed from the ætiology and accompanying symptoms.

Simultaneous deviation of both eyes towards the right or left, upwards or downwards, or affections of the associated movements, cannot be confounded with paralysis of isolated muscles nor with a concomitant strabismus. The nature of the primary lesion indicates whether we are dealing with a case of paralysis or spasm, with a destructive or irritative lesion, in the region of the nervous centre for the associated movements (pp. 90 *et seq.*).

IV. AFFECTIONS OF OCULAR MOTILITY FOLLOWING A LESION OF THE CENTRES OF THE EQUILIBRIUM OF THE BODY AND EYES (PARADOXICAL AFFECTIONS).

There exist certain, though rare, affections of ocular motility which are distinguished by their incoherence. They do not correspond to lesions of the centres of binocular vision, but are accompanied by most paradoxical combinations of vision. For example, when one eye is turned outwards the other turns upwards, or, again, if the patient is asked to look up, one eye is rotated upwards and the other downwards.

Frequently the levators of the eyelids participate in these incoherent movements. It may happen that when the patient opens his eyes widely the eyes involuntarily deviate outwards or downwards. Conversely, lateral and down-

ward deviation has been observed when the eyes are closed, this phenomenon frequently being accompanied by a facial paralysis.

At the moment when the eyes converge the eyelids may open or shut involuntarily, or the eyelid on one side may be lowered when the eyes are directed to the opposite side.[1]

Goldsheider reported a case of hysteria in which spasmodic elevation of the left upper lid occurred when the patient looked downward and to the right.

Some of these phenomena may have their seat of origin in the region of the centres; the most complex phenomena occur in affections of the *labyrinth* and *cerebellum*. In these two localities, but chiefly in the cerebellum, centres are found for regulating the movements of the eyes; up to the present these centres have not been fully investigated.

According to Bernheimer, the motor affections noted in *lesions of the labyrinth* (lateral deviation, nystagmus, etc.) are merely irritative phenomena at the level of the motor centres of the eyes, the irritation being caused by the alteration of pressure transmitted from the internal ear by the subarachnoid space; or they may be the result of irritative reflexes produced by irritation of the roots of the auditory nerve which are closely related to the motor nerves of the eyes.

All the forms of strabismus, especially those which we have classed under the heading "Paradoxical Affections," can be produced by alterations of the internal ear which constitutes the principal regulator of the relative direction of the eyes.[2]

Nystagmus.

We may class *nystagmus* with the paradoxical affections. Nystagmus is a characteristic oscillating movement which may be either vertical, lateral, or round the sagittal axis.

[1] Wilbrand and Saenger, *Neurologie des Auges*, i.
[2] M. Bartels.

According to the form of the oscillations of the eye, the nystagmus is called REGULAR and pendulum-like (*ondulatoire*), when the eyes move equally both in quantity of movement and rate, to either side from the median position of rest. It is called REGULAR and jerky (*à ressort*), when the movements consist of a slow phase in one direction followed by a rapid phase in the other direction. There is a third form—IRREGULAR, when the direction and duration of the oscillations are constantly variable.

This affection, often congenital, may develop in infancy as the result of *corneal* opacities, or opacity of the *lens* (polar cataract), or may result from *amblyopia* from various causes.

It is then due to a defect in development of the co-ordinating centres for the ocular movements which has not been governed by the vision. This nystagmus is usually irregular.

Congenital nystagmus of pendulum-like form frequently accompanies hemeralopia (retinitis pigmentosa), and especially nyctalopia with total achromatopsia. The ocular movements in the latter case are due probably to a central scotoma.

It is not uncommon to meet with nystagmus in *albinos*.

Congenital nystagmus must be differentiated from acquired nystagmus. In subjects affected with the former, objects appear immobile in spite of the continuous movements of the eyes; in the subjects affected with the latter, objects seem to move in the same way as the movements of the eyes. Sometimes the head also takes part in the nystagmic rhythmical movements.

Nystagmus frequently affects miners. It is also found in other classes of workers—milliners, seamstresses, and those who work in bad light or in a strained position for hours at a stretch. This is a regular clonic spasm due to fatigue but an incomplete clonus (Coppez). A lowered state of vitality also predisposes to this condition.

In *miners'* nystagmus the eyes usually oscillate round the sagittal axis, and frequently round the vertical axis at

the same time. In directing the vision downwards the nystagmic movements cease; on looking upwards, and particularly obliquely upwards, the nystagmus increases; the same is true if the worker raises his head suddenly.

Lesions of the labyrinth are accompanied by an irregular (*saccadé*) jerky nystagmus. The excitation of the vestibular nerve produces an associated movement of both eyes, which would be continuous if anatomical conditions would permit of it.

When the eyes have reached the extreme possible position of their excursion they are pulled back to their original position, by the sudden contraction, to allow the movement to again recommence. It is a veritable kinetic conjugate deviation (Coppez).

Since fixation dominates the direction of the eyes, nystagmus only becomes apparent when the eyes are covered or are amblyopic.

Nystagmus also accompanies a large number of diseases of the *brain*: disseminated sclerosis, meningitis, hæmorrhages, emboli, cysticercus, and hydrocephalus. The regions affected are very various and may be very extensive: the lesion may lie in the fourth ventricle, the optic thalamus, corpora striata, corpora quadrigemina, cerebellum, restiform bodies, or the medulla oblongata. Variations in the pressure of the cerebrospinal fluid may also produce nystagmus (Uhthoff).

In *tabes* true nystagmus is rare; the tremors of the eyes met with in this disease, as also in *syringomyelia* and other affections of the cerebrospinal system, are not identical with nystagmus. Such tremors may be observed in all fatigued or feeble ocular muscles, and especially so in the extreme positions of rotation of the eyes.

An hypothesis has been put forward that certain toxic substances, such as carbonic oxide, can produce nystagmus by their action on the central nervous system.

SEAT AND ÆTIOLOGY OF THE OCULOMOTOR AFFECTIONS.

The lesions apt to interfere with the normal ocular movements may lie in the *orbit*, in the *superior orbital fissure*

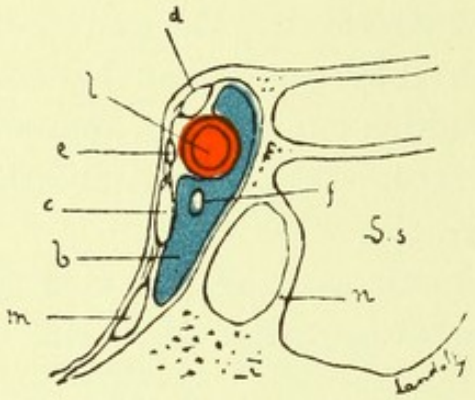


FIG. 21.—Vertical transverse frontal section passing through the posterior third of the cavernous sinus.

b, Cavernous sinus; *c*, Ophthalmic division of the oculomotor nerve; *d*, Trigeminal nerve; *e*, Trochlear nerve; *f*, Abducens nerve; *l*, Carotid artery; *m*, Maxillary division of the trigeminal nerve; *n*, Septum of the sphenoidal sinus; *ss*, Sphenoidal sinus.

through which the motor nerves pass in entering the orbital cavity, in the *cavernous sinus*, in the course of the nerves to *the base of the brain* between their exit from the brain and their *nuclei*, in the nuclei themselves, or even higher up in the *cortex* of the brain.

Finally, lesions of the *internal ear*, of the labyrinth, of the tympanic cavity, and especially *cerebellar* disease, may all give rise to oculomotor affections.

Lesions situated below the nuclei are called *peripheral*, those situated above *central*.

Peripheral lesions are classified as *orbital* (the sphenoidal fissure included), *basilar* and *fascicular*.

The central or supranuclear lesions may be subdivided into *subcortical* and *cortical*.

Amongst the lesions in the *orbit* itself which may interfere with ocular movements we find hæmorrhage, exudations, empyema, neoplasms originating not only from the orbital cavity but also from the accessory cavities (frontal, maxillary or ethmoidal sinuses); further, we may cite thrombosis of the orbital veins.

Injuries: Wounds from sharp instruments, gunshot wounds, or the cicatrices following such wounds may injure the ocular muscles either directly or indirectly by inflammation of neighbouring structures.

New growths: Carcinoma, sarcoma, gumma, etc., as well as trichinosis, may all affect the ocular muscles.

Frequently periostitis of the sphenoidal fissure produces ocular paralysis by compressing the nerves which traverse the fissure or by the extension of the inflammation.

Congenital malformations of the muscles, such as anomalies of length, insertion, or force, which were formerly believed to be causes of strabismus, we think to be exceedingly rare.

The first-mentioned causes, such as tumours, which by their volume limit the excursion of the eye, usually displace the eyeball in such a way that their situation can be diagnosed. Similarly wounds may be localised very exactly by noting which muscle is paralysed as the result of the wound.

We do not wish, however, to begin by pointing out the seat of the lesion and then describe the symptoms resulting, but, from the point of view of the practitioner's interest, we first detail the abnormalities found, and from this diagnose their cause. We shall begin with isolated paralysis of individual ocular muscles.

1. Paralysis of the Rectus Lateralis.

To investigate thoroughly a case of paralysis of the *lateral rectus* we must bear in mind the course of the nerve which supplies it from its origin to its entrance into the muscle.

The nucleus of the nerve to the lateral rectus (abducens, VI), is the lowest of all the motor nuclei of the eyes, and is situated behind the pons in front of the eminentia teres; it is surrounded by the primitive fibres of the facial, VII (Figs. 22, 23, 24). The root fibres of the sixth on one side do not cross with those of the other side. Some fibres, however, are detached from the nucleus of one side and pass towards the oculomotor nerve of the other side, doubtless for the rectus internus.

The fibres of the sixth united in a single bundle pass forwards, cross the medial longitudinal bundle, traverse the trapezium and appear at the base of the brain between the pyramids and the posterior border of the pons (Fig. 25).

From this the abducens is directed forwards and upwards for about 3 cm. along the median extremity of the petrous portion of the temporal, over the apex of which it curves to enter the cavernous sinus.

In the sinus it lies close to the internal carotid; it follows the ophthalmic division of the trigeminal and penetrates the orbit at the inferior extremity of the superior orbital fissure, and enters the muscular cone (Figs. 21 and 23).

In the orbit it lies on the medial side of the lateral rectus, which it penetrates a little in front of its posterior third.

Isolated paralysis of the lateral rectus, as of every motor nerve of the eye, may come on during an infectious disease or from some toxic cause, or from one or other of the general nervous affections which will be enumerated later (p. 96). In such a case the lesions affect the nerve in its nucleus as well as in its course.

Apart from these affections, isolated paralysis of the abducens nerve is met with frequently in cases of circum-

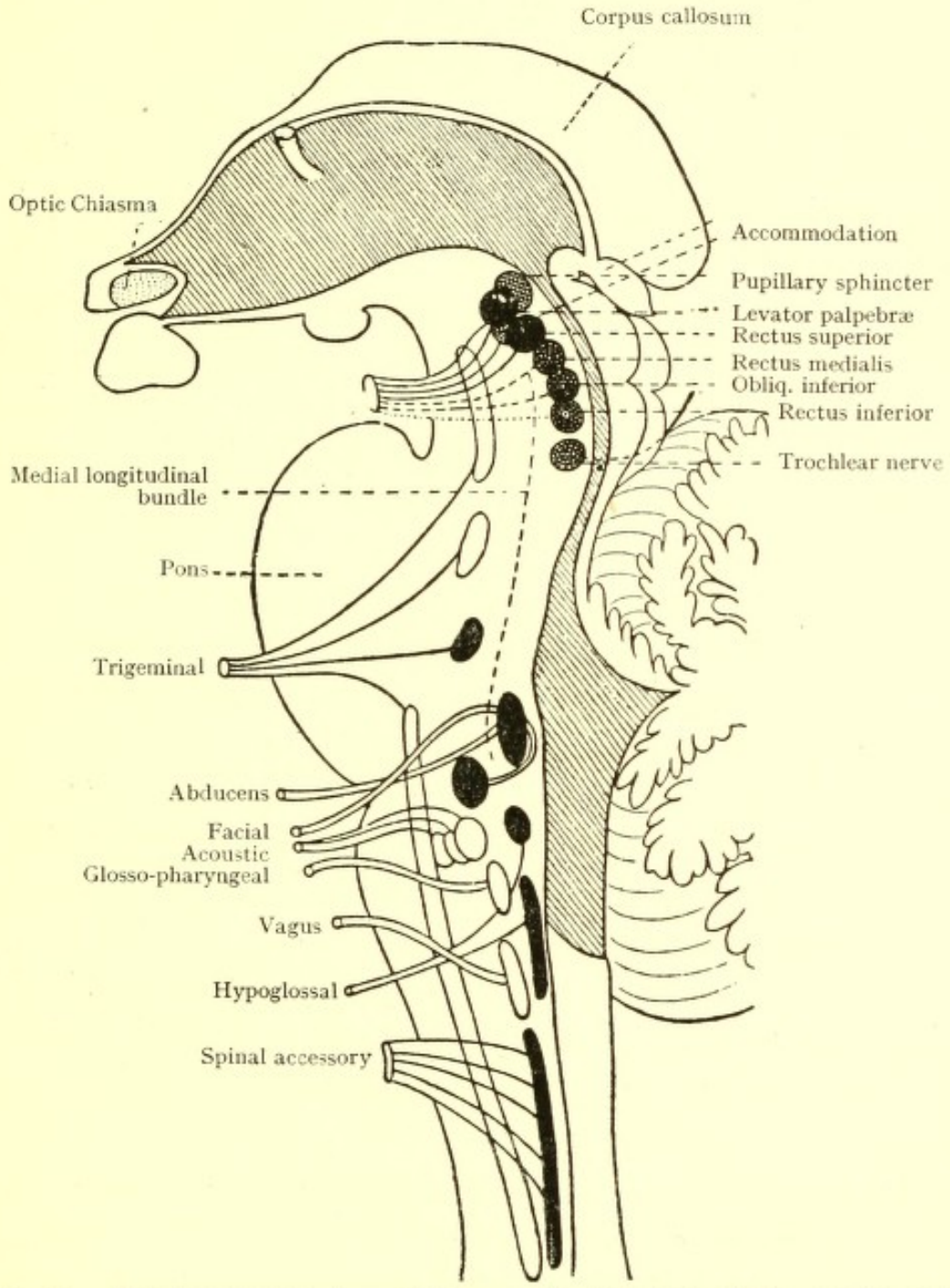


FIG. 22.—Diagrammatic view of the nuclei of origin of the ocular motor nerves, according to Poirier and Bernheimer.

scribed periostitis of the orbit; in aneurysm of the internal carotid in the cavernous sinus (in which in most cases the third, fourth and optic nerves also suffer); in fracture of the

petrous portion of the temporal (Panas); in lesions of the posterior portion of the pons, particularly circumscribed

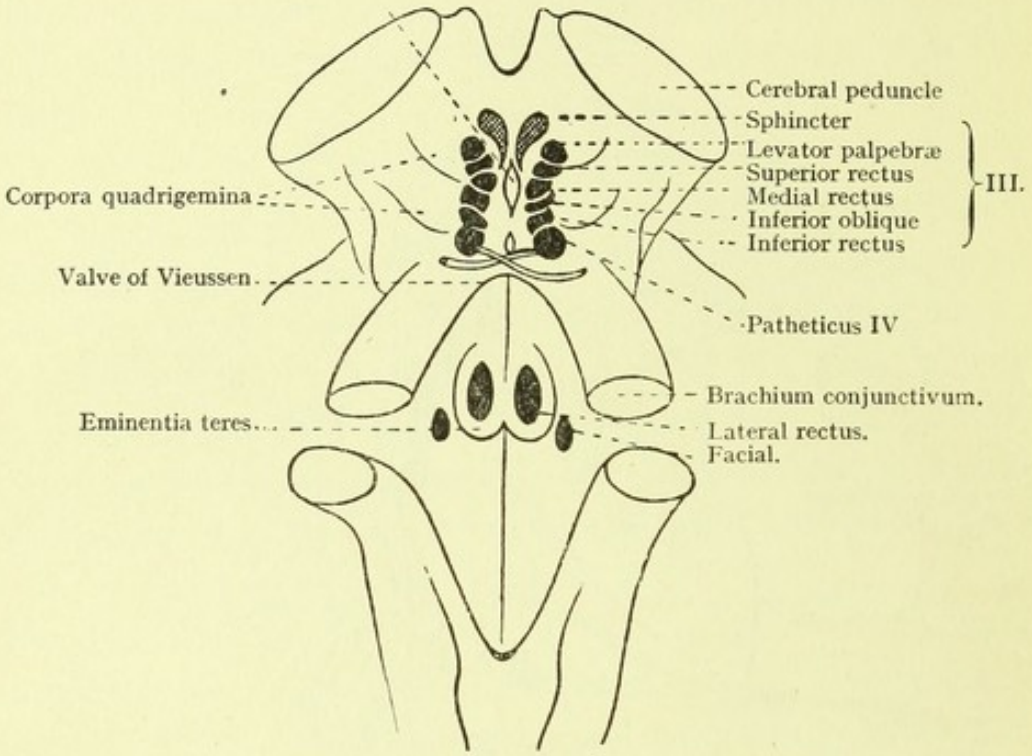


FIG. 23.—Diagrammatic view of the nuclei of origin of the ocular motor nerves, according to Poirier and Bernheimer. (Dorsal view.)

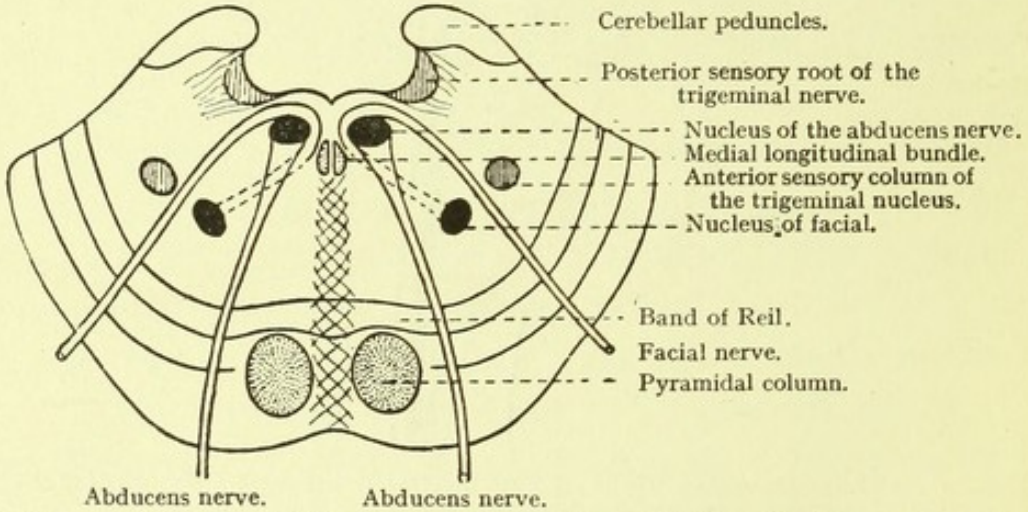


FIG. 24.—Section through pons and fourth ventricle at the eminentia teres (according to Poirier).

tumours near the median line, or in aneurysm of the middle cerebellar artery.

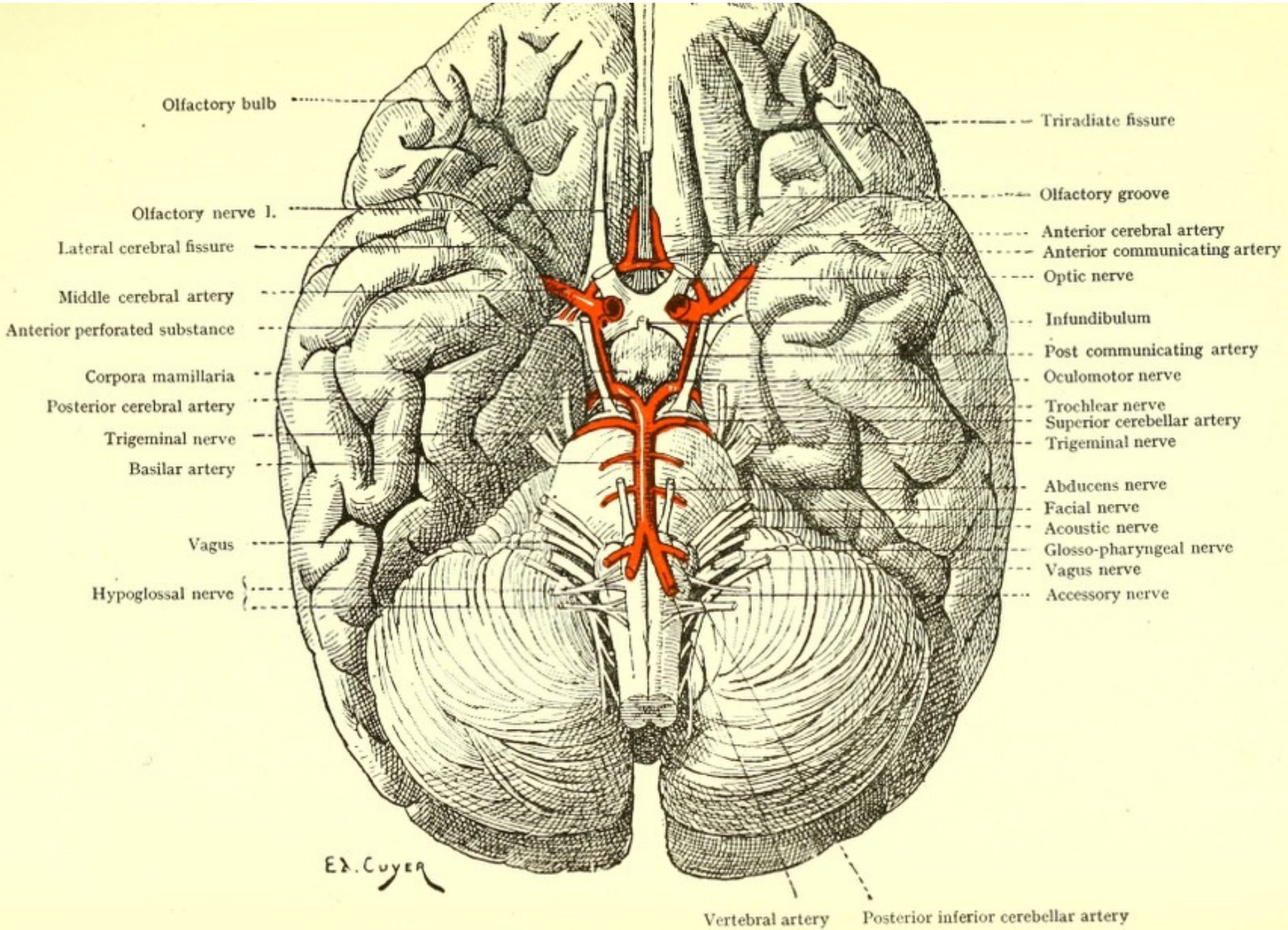


FIG. 25.—Base of the brain according to Poirier.

In simultaneous paralysis of one of the abducens nerves and the facial we conclude that there is a lesion in the nuclear region.

Congenital isolated paralysis of the lateral rectus has been observed in hereditary syphilis, or as the result of trauma from forceps.

Congenital affections of the motility of one eye are generally accompanied by defect in motility of the associated movements of the other eye.

Thus, for example, in paralysis of a left lateral rectus, if, when the right eye is covered, an object is displaced towards the left, the right eye remains stationary, whereas in its normal condition it would have followed the impulse given by the retinal image of the left (see Fig. 27).

Tumours of the acoustic nerve at the level of the pontine angle of the cerebellum can produce paralysis of the sixth nerve. In such cases not only are other nerves involved, acoustic, facial, or trigeminal, but nystagmus, vertigo, vestibular ataxia, or hemiataxia may occur, indicating that a cerebellar lesion or disease of the labyrinth is present. Paralysis of different ocular muscles frequently follows inflammation of the *internal ear*.

The inside of the tympanic chamber communicates directly by air cells with the apex of the petrous portion of the temporal, and otitis can easily extend to the latter and set up a circumscribed meningitis which affects the motor nerves in the vicinity. Infection may, moreover, spread by the lymph vessels which accompany the nerves and the vessels of the tympanitic cavity into the cavity of the skull.

Of all the motor nerves the abducens approaches the bony cavity of the ear most closely, and remains for the longest period in contact with it. It is therefore not surprising to meet with usually a paresis, frequently a

spasm, of the abducens nerves in an external otitis. The spasm becomes most apparent, seemingly, when the eye is closed (Gradenigo). The fourth pair can also be affected by an inflammation of the internal ear.

Paralysis of the lateral rectus with paralysis of the facial on the same side and hemiplegia on the opposite side (symptom-complex of Millard Gubler) indicates a lesion in the lower part of the pons. Decussation of the fibres supplying the face takes place at a higher point than those of the pyramidal fibres for the limbs.

Bilateral paralysis of the rectus lateralis must not be confounded with paralysis of divergence or a spasm of convergence, in which the temporal excursions of the eyes are normal. Double paralysis may be produced by a lesion of the base of the brain or of the nuclear region. The differential diagnosis is made by noting the accessory symptoms present. We have observed a double paralysis of the rectus lateralis in a case of tabes.

It must be noted that when both eyes are affected simultaneously with paralysis, only one is used for fixing objects usually, the other not participating in this function at all. The patient soon learns to direct this eye correctly, so that the false projection which one would expect is absent or at any rate not characteristic.

2. **Paralysis of the Superior Oblique.**

The nucleus of the trochlear nerve lies just in front of the Aqueduct of Sylvius and below that of the inferior rectus, so that it appears at the lower end of a group of nuclei for the oculomotor nerve (Figs. 22, 23). The crossing of the superior peduncles of the cerebellum is situated immediately in front of it.

The fibres of the trochlear nerve decussate completely. United into one trunk they leave the brain on the dorsal

side—contrary to the method of exit of the other cerebral nerves—behind the posterior quadrigeminal bodies. The nerves there pass in an outward and forward direction encircling the cerebral peduncles (Fig. 23), and appear at the base of the brain immediately in front of the trigeminal nerve (Fig. 25). After a short course on the superior petrosal sinus, by which it is separated from the pyramidal portion of the petrous bone, the trochlear nerve enters the lateral wall of the cavernous sinus. At this point it crosses the oculomotor nerve as it passes downwards. While this latter traverses the superior orbital fissure approximately in the centre, the trochlear nerve passes through it higher up and outside the region of the muscular insertions (Fig. 3). It follows the course of the superior oblique muscle and penetrates it on its upper surface.

As the fourth nerve remains at a distance from the base of the brain until it enters the cavernous sinus, it is much less exposed to basal lesions than the abducens, or oculomotor nerve, both of which lie close to the base for a considerable length of their course.

Isolated paralysis of the superior oblique is thus most frequently radicular or nuclear in origin, and results from the lesions already cited, or of which we have still to speak later; the muscle itself, of course, may be the seat of lesion in the orbit. This may occur frequently enough after the radical cure for an empyema of the frontal sinus.

3. **Paralysis of the muscles supplied by the Oculomotor Nerve.**

The *oculomotor nerve*, which supplies so many muscles, and which governs so many various functions, originates in a grey column which lies in front of the Aqueduct of the Brain and the Corpora Quadrigemina (Figs. 22 and 23). This column has been divided into nuclei which correspond

(so it is believed) to the following muscles in order from below upwards: *Inferior rectus, inferior oblique, rectus medialis, rectus superior, levator palp. superior.* Further forwards, or *in any case* nearer the median line, are said to lie the nuclei of the *sphincter of the iris* (contraction of the pupil) and of the *ciliary muscle* (accommodation).

The centre of convergence seems, moreover, to lie in the median plane between the nuclei of the two oculomotor nerves.

In any case it is certain that the *inferior* nuclei of the column correspond to the *external muscles of the eye,* the *superior* or *anterior* to the *internal muscles.*

The exact order in which the nuclei corresponding to each muscle lie in the large column of origin of the third pair is not yet exactly determined. Thus, for example, Bach and Tsuchida localise the nucleus of the inferior rectus uppermost in the column, whereas it is usually considered to be the lowest of the group.[1]

There is the same uncertainty about the decussation of the fibres of the oculomotor nerve in man. It is usually admitted that the decussating bundle originates in the dorsal cellular group of the inferior (caudal) extremity of the oculomotor nucleus.

According to Von Monakow, Bach, Juliusburger, Kaplan and others the crossed fibres, though constantly decreasing in number, extend as far as the upper third of the cell column. As, however, it is yet uncertain as to the different relative positions of the nuclei of the various muscles, it is also not known exactly which muscles receive innervation from crossed fibres, which from partially crossed fibres, and which from uncrossed fibres only.

Bach holds that the fibres of the *rectus superior* are

[1] An article on all the different opinions on this subject is found n Willbrand and Saenger, *Neurologie des Auges,* i, p. 111.

crossed fibres; Bernheimer those of the *rectus inferior* and *inferior oblique*; Pangrossi and Spitzka those of the *rectus internus*.

Tsuchida[1] believes that in man there is no real crossing of the oculomotor fibres, and that consequently the nuclei of one side may be totally destroyed without producing paralysis in the region governed by the nerve on the opposite side. The apparent crossing or rather interweaving of the fibres which is observed, particularly in the lower part of the nuclei, is in his opinion formed by commissural fibres or higher projection fibres.

As has already been stated, the nuclei of the *oculomotor nerve* of one side are connected with the nucleus of the *abducens* on the other side.

The nuclei of the motor nerves of the eye may suffer not only from direct lesions, but also from disturbance of their vascular system. The peculiarities of their blood supply explain the lesions found in vascular disease. Thus the anterior portion of the nuclear column is supplied by the *posterior cerebral artery*, the posterior portion by the *basilar artery*. Some nuclei may be involved, whereas others may remain intact.

These vessels are in addition terminal arteries. This explains their great susceptibility to variations in the blood pressure.

After leaving their nuclei the fibres of the oculomotor nerves pass through the crus, and are united into a single nerve trunk shortly before their exit from the base of the brain.

The two nerves leave the brain close to each other at the anterior part of the Pons (Figs. 22, 25 and 26). Each one is directed forwards, upwards and laterally through the sub-

[1] Tsuchida, *Die Ursprungskerne der Augensbewegungsnerven: Arbeiten aus d. hirnanatomischen Institut in Zürich*, ii, s. 154, 1906.

arachnoid space, between the two last branches of the *basilar artery* (Fig. 25), and reaches the external wall of the cavernous sinus; this it penetrates at the level of the *posterior clinoid process*, and proceeds under the small wing of the sphenoid to the *superior orbital fissure*.

In the posterior portion it is separated from the cavernous sinus by a thin membrane, whilst in front, according to certain writers, it lies free in the blood-stream, as does the abducens nerve.

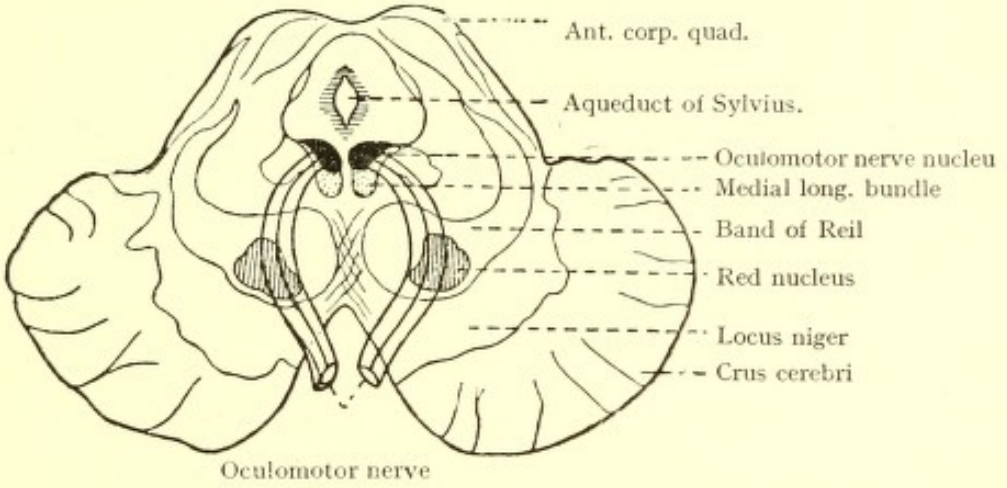


FIG. 26.—Section through the anterior corpora quadrigemina. (Déjerine.)

It enters the orbit by the inner portion of the superior orbital fissure (Fig. 3) below and to the medial side of the optic foramen, and to the medial side of the sixth nerve, and divides immediately into two branches, the upper for the *superior rectus* and *levator palpebræ*, the lower for the *medial rectus*, the *inferior rectus* and the *inferior oblique*.

A small bundle is detached from the nerve to the inferior oblique and goes to the *ciliary ganglion;* it is the short or motor root of the ganglion, and contains the fibres for supplying the *internal muscles* of the eye.

In the cavernous sinus the oculomotor nerve receives a sensory anastomosis from the *ophthalmic division* of the fifth and a sympathetic branch from the *carotid plexus*.

The proximity between the nuclei of origin of the oculomotor nerve explains the close interdependence of the muscles which are supplied by it; still, isolated paralysis of a muscle is not impossible, and as a matter of fact is actually found. Usually in such cases there is only paresis, and this paresis is frequently the beginning of a disease which will later affect other muscles, perhaps even all the other muscles supplied by the oculomotor nerve.

When one isolated muscle is paralysed the cause may lie, if high up, in the first nuclear or radicular region, in other words, at a point where the fibres have not yet united into a bundle; or it may be quite at their peripheral extremities, in the orbit where the nerve roots are already divided.

There is a tendency to admit, moreover, the possibility of a dissociated paralysis following a nerve lesion of the trunk itself.

Small hæmorrhages and areas of softening, but principally all infections and intoxications, can bring about dissociated central paralysis. Peripherally in the orbit a traumatism, compression produced by a neoplasm, an inflammation of the neighbourhood propagated from a sinus, can affect a nerve or a muscle of the group supplied by the third pair. The rectus superior and the levator palpebræ usually are affected at the same time.

When paralysis of an isolated nerve bundle of the oculomotor nerve is accompanied by motor affections of the face or the limbs, the cause is usually radicular, that is, it lies between the nuclei and the point where the fibres reunite into a trunk. Thus a lesion of the *crus cerebri* may simultaneously implicate some root fibres of the oculomotor nerve and some fibres of the pyramidal bundle going to the limbs.

As the former go to the eye of the same side, whereas the latter only cross lower, ocular paralysis occurs on the same side as the lesion, whereas paralysis of the limbs occurs

on the opposite side: *superior alternate hemiplegia* (symptom-complex of Weber).

Ptosis, or drooping of the upper eyelid, is a frequent symptom in many different affections of the central nervous system. It is usually due to a paralysis of the levator of the upper lid. The nerve for this muscle belongs to the oculomotor nerve, and its origin is found almost certainly in the most anterior portion of the nuclear group of the third nerve. It appears to have a very extensive cortical centre, and the cerebral paths which control the levator appear to take another course than those which govern the movements of the eye. The exact position of the cortical centre of the levator is not yet definitely ascertained. The most reliable researches made up to the present point to it being situated in the parietal lobe.

The differential diagnosis between a supra- and infra-nuclear lesion is made in the following manner: The patient fixes an object, his head being slightly bent backwards, and then the head is slowly brought into the normal upright position. If the eyes remain fixing the object and the upper lids are raised, the lesion must be higher than the reflex tracts, that is, between the cortex and the nuclei; if, on the contrary, the upper lids hide the fixed object the lesion is infra-nuclear; the paralysis is complete.[1]

Ptosis is frequently the precursor of paralysis of other nerves belonging to the third pair. The seat of lesion in such cases is frequently *nuclear*.

A *unilateral* incomplete ptosis may be a symptom of increased intracranial pressure in cerebral diseases (Uhthoff).

Bilateral ptosis is met with in asthenic bulbar paralysis, in most cases with remission, and is usually followed by external ophthalmoplegia, weakness of the limbs and of the muscles of mastication; it is also met with in polio-encephalo-

[1] Landolt, *International Clinics*, iii, p. 284. 1910.

myelitis. The differential diagnosis between those two diseases depends upon the electrical muscular reactions. In the former there is a myasthenic muscular reaction, in the latter qualitative alterations of excitability (Willbrand and Saenger).

A peculiar form of *double ptosis*, whose cause is not yet well understood, and not yet well localised, has been observed by Gerlier, in the symptom-complex, which he has called " vertige paralysant " ; it is accompanied by vertigo, muscular weakness, and pain in the back.

Double *isolated congenital ptosis* has been described; this form may also develop gradually, and is chiefly found in women; it is probably dependent upon an incomplete development or degeneration of the levator muscles, or may be due to an arrested development of the nuclei.

Ptosis is frequent in hysteria. Though usually due to a *spasm* of the orbicularis in such cases, it may, however, be found as a *true flaccid hysterical ptosis*.

Slight *unilateral ptosis* is observed in paralysis of the sympathetic, and is due to paralysis of the unstriped muscular fibres. The lower lid is then slightly raised towards the upper. The accompanying symptoms—miosis, enophthalmos, and hyperæmia of half of the face—leave no doubt as to the cause of the affection. To demonstrate more clearly the difference between the healthy eye and the one supposed to be affected with paralysis of the sympathetic, a solution of cocaine is instilled into both eyes. Whilst this produces dilatation of the palpebral fissure and the pupil of the former, no such reaction is shown in the latter.

Ptosis may accompany a total facial paralysis. Possibly the levator of the lid gets some fibres from the nucleus of the facial nerve; as a matter of fact, when the upper lid is raised to its maximum the frontalis muscle contracts (Brissaud). There exists a certain number of observations where a

paralysis at first complete of the oculomotor nerves has been followed by paradoxical affections which have been nominated the pseudo-sign of Graefe. The upper lid, still more or less paralysed for voluntary movements, lags behind the other when the patient looks downwards, it raises markedly in looking inwards, and especially inwards and downwards (adduction and convergence). It is supposed that during the regeneration of the nerve some strands have followed a false direction.

Moreover, it must be borne in mind that even a slight increase in weight of the upper lid (œdema, conjunctivitis, etc.) may bring on a certain degree of *ptosis*, no lesion existing in either the muscle or the nerve.

In rare cases, isolated paralysis of a muscle supplied by the third group is met with, usually groups of muscles, or all the muscles supplied by the oculomotor nerves, are affected at the same time; it also happens that other muscles of the same side, or even of the opposite side, are simultaneously paralysed.

Ophthalmoplegia is the name given to simultaneous paralysis of several ocular muscles.

To simplify the classification of paralysis of entire groups we have placed them in the following order :—

1. Unilateral partial ophthalmoplegia—paralysis of certain muscles of one eye.
2. Unilateral total ophthalmoplegia—paralysis of all the muscles of one eye.
3. Bilateral partial ophthalmoplegia.
4. Bilateral total ophthalmoplegia.

Ophthalmoplegia may be complete or incomplete according as the muscles are entirely or only partially functionless.

I. Unilateral Partial Ophthalmoplegia.

In this class we include, in the first place, paralysis of muscles innervated by the *oculomotor nerve*.

Two distinct forms of partial paralysis of the oculomotor nerves are described: 1st, that in which the external muscles alone (those governing the rotation of the eyeballs) are affected (*External ophthalmoplegia*); 2nd, that in which the internal muscles (the sphincter of the pupil and the ciliary muscle) are paralysed; this form is called *Internal ophthalmoplegia*.

By referring to our diagram (Fig. 22) we see that the former is due principally to a lesion of the inferior portion of the grey column of the oculomotor nucleus, whereas the latter form is due to a lesion of the superior portion of this column.

Sometimes an external ophthalmoplegia is only accompanied by a partial paralysis of the levator; this doubtless is due to the fact that the levator receives, as already stated, a nerve supply from the facial (VII), and perhaps also from a cortical centre in the angular gyrus.

Obviously an ascending or descending progressive lesion will convert either an external or internal ophthalmoplegia into a *total ophthalmoplegia*. If such a paralysis be accompanied by *glossolabio-pharyngeal paralysis*, *glycosuria*, or *polyuria*, the case will be one of a very extensive polioencephalitis.

There is a form of ophthalmoplegia interna which does not originate in the nucleus, but in the orbit, as is shown by the co-existing paralysis of the inferior oblique; in such a case the lesion lies in the *short root of the ciliary ganglion*.

In these cases the ethmoidal cells are frequently the primary seat of lesion.

If, as Bernheimer states, the fibres supplying the sphincter of the pupil form an isolated bundle situated in the centre

of the oculomotor nerve bundle, we should meet with paralysis of the sphincter alone in lesions of the inner part of the oculomotor nerve, and paralysis of the oculomotor nerve with the sphincter intact in lesions of the outer portion of the nerve.

Paralysis of *all the muscles* supplied by the third nerve without any other muscle being affected, is caused by a lesion of the nerve before its entrance to the sphenoidal fissure, and even above the cavernous sinus. If the seat of the lesion be in the fissure itself, other nerves passing through it would probably also be involved.

On the other hand, in a lesion affecting all the nuclei of the oculomotor nerve, the crossed fibres, that is, those passing to the other eye, would be involved.

In *total* paralysis of the oculomotor nerve the lateral rectus and superior oblique on the same side are slightly affected. Figs. 16 and 17 show this clearly by the fact that the field of fixation is somewhat limited in the lateral and infero-lateral part of the field of vision.[1]

A *recurring ophthalmoplegia* has been observed in neoplasms of the trunk of the oculomotor nerve (Uhthoff).

A *total* but *transitory* ophthalmoplegia is recognised in France and called "Migraine Ophtalmoplégique," because it is accompanied by severe pains and sometimes by vomiting; usually it affects the same eye repeatedly, but sometimes it affects both alternately. The seat of the lesion in such cases lies in the nuclei, and it is probably due to a congestive process in this region.

Paralysis of the oculomotor *nerve*, without involving the trochlear and abducens, and associated with *paralysis of the opposite side of the face and body* (symptom-complex of Weber), is suggestive of a lesion (hæmorrhage, softening,

[1] E. Landolt, *Étude sur les mouvements des yeux*, etc. : *Arch. d'opht.* 1881.

tubercle) of the inner side of the crus cerebri. As the fibres of the oculomotor nerve are more or less separate in their course through the crus cerebri, only some of them may be affected at one time. The more complete the paralysis of the oculomotor nerve the greater is the lesion, or the nearer it is to the surface of the crus cerebri.

The degree of the hemiplegia also depends upon the size of the lesion. Frequently partial paresis of the oculomotor nerves is met with accompanied only by paralysis of the lower part of the face on the opposite side.

When paralysis of the face is more complete than that of the limbs the lesion lies on the medial surface of the crus cerebri.

Instead of paralysis, symptoms of *irritation* may exist in the region supplied by the oculomotor nerve and on the opposite side of the body, with contractions or tremor of the opposite limbs (Benedikt's symptom-complex). In such a case there is an irritative lesion in the crus cerebri.

Paralysis of the *oculomotor, abducens, facial and trigeminal* associated with paralysis of the *opposite* side of the body, or of all the limbs, occurs in tumour of the pons cerebri encroaching on the nuclear region of the motor nerves of the eye, on the aqueductus cerebri and the corpora quadrigemina, or there may be multiple tumours in this region.

II. Unilateral Total Ophthalmoplegia.

Paralysis of all the motor nerves of one eye, without involving the other eye, can only be caused by a lesion in the *orbit* in the neighbourhood of the *superior orbital fissure*, or in the *cavernous sinus*.

Cellulitis of the orbit secondary to an affection of one of the sinuses may produce complete immobility of the eye.

Lesions due to trauma, tumours, etc., affecting all the

motor nerves of the orbital cavity, may also implicate other nerves in the vicinity; the lacrimal, frontal, naso-ciliary (neuro-paralytic keratitis) and even in certain cases the optic nerve may be involved. Syphilitic periostitis in the region of the superior orbital fissure will produce similar symptoms.

Lesions of the cavernous sinus are accompanied by symptoms of venous congestion as well as sensory and motor symptoms.

If pulsatile exophthalmos is present it indicates rupture of the internal carotid into the cavernous sinus (arterio-venous aneurysm).

Some writers attribute unilateral total ophthalmoplegia to the destruction of all the motor nuclei on one side. In such a case, however, a careful examination would reveal more or less accentuated affections of the motility of the other eye, as the nuclei of both sides have fibres of connection. A destructive lesion of all the nuclei of one side should not produce a total unilateral ophthalmoplegia, but rather—

III. Bilateral Partial Ophthalmoplegia.

The cause of paralysis of several muscles of both eyes lies most frequently in the nuclear region or in the base of the brain; some peripheral lesions, such as those of the frontal or sphenoidal sinus, may, however, simultaneously affect the contents of both orbits.

Thus, *paresis* of the *recti laterales, recti mediales, rectus superior, the sphincter of the pupil, and ciliary muscle* may occur during frontal or sphenoidal sinusitis, or from inflammation in the ethmoidal cells (Bernheimer, Kuhnt).

Bullets not infrequently injure both orbital cavities simultaneously.

If, in conjunction with slight motor disturbances of one

eye, there be total ophthalmoplegia of the other eye and hemiplegia of the opposite side of the body, we are dealing with a lesion of the pons. If, moreover, there exist a lesion of the optic tract, of the chiasma, or of the optic nerve, the cause will very probably lie in the base of the brain.

If the symptoms of a basal lesion are wanting, whilst the ophthalmoplegia is accompanied by *bulbar* symptoms (glosso-labio-pharyngeal paralysis), we must look for the seat of lesion in the nuclear region. This is particularly the case when the external muscles are affected whilst the internal ones remain intact (*inferior polio-encephalitis*).

If the ophthalmoplegia is associated with neuralgia of the trigeminal nerve and disturbances of equilibrium, the lesion (usually a tumour) lies in the region of the crus cerebelli. A tumour in this situation need not necessarily be large to produce signs of compression, as these do not result directly from the tumour, but from hypertension of the cerebrospinal fluid. A small tumour of the pineal gland or in the vicinity of the Aqueduct of the Brain can produce a marked increase in pressure as the result of venous congestion.

Simultaneous paralysis of the two *lateral recti* or of both *oculomotor nerves* has already been cited as a symptom of a basal lesion (tumour, meningitis, hæmorrhage, vascular changes) or of tabes.

We must remember that a single muscle of the eye never contracts alone, but that all the muscles of both eyes come into play to carry out the movements of the eyes with mathematical precision which is necessary for binocular fixation and for retaining both eyes fixed in the desired direction. We shall presume then, even though anatomy has not yet proved it for us, that the nuclei of origin are not isolated units, but are associated in the same manner as the movements of the muscles governed by these nuclei.

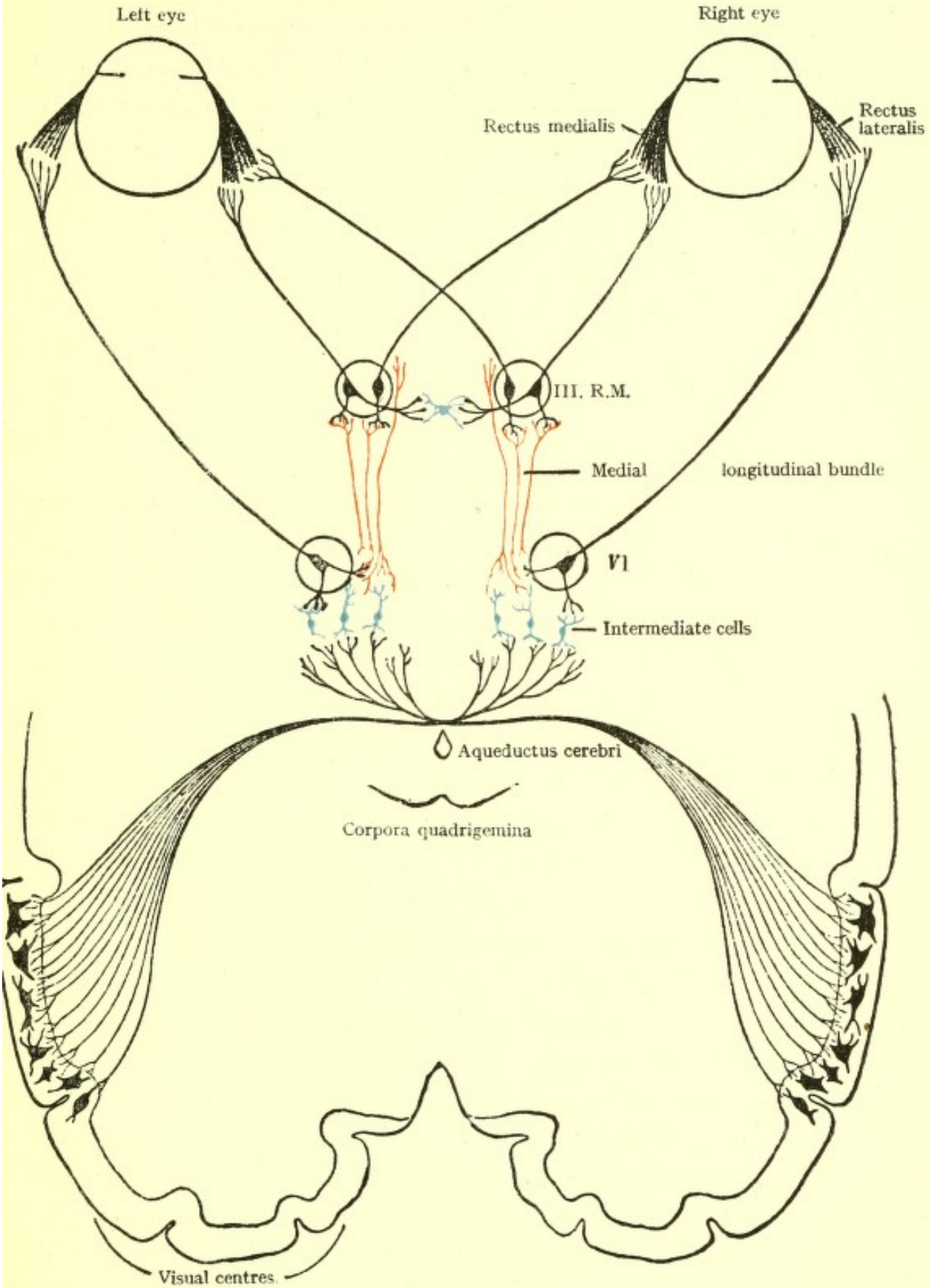


FIG. 27.—Diagrammatic representation of the innervation for the lateral and convergent movements of the eyes, according to Bernheimer.
III. R.M. Centre of the rectus medialis; VI. Nucleus of the abducens.

Thus it may occur, that in some cases of binocular ophthalmoplegia, the seat of the lesion really lies in the nuclear region, although one might be inclined to look higher up, in the sphere of volition, the region from which the impulses for binocular movements start.

As a matter of fact we meet with associated binocular affections of lateral movements, upward and downward rotation, of convergence or divergence, as the result not only of lesions in the pons, but also of lesions in the cortex, or the fibres which come from it.

The differential diagnosis is made by the already noted fact that in the former case all motility is abolished, in the latter case voluntary movements only are deficient. The patient is asked to look at an object and then his head is turned in the opposite direction to that of the affected muscle. If the eyes follow the direction of the head, the paralysis will be nuclear, if the eyes remain fixed on the object the seat of lesion is higher up in the brain.

Affections of the Associated Lateral Movements.

The cause of an affection of the *horizontal binocular movements* may lie in the region of the pons (longitudinal bundle), or may be cortical or subcortical, at the level of the centres which govern the lateral movements.

These were formerly regarded as situated in the angular gyrus, but are now, and probably more correctly so, localised in the second frontal convolution, in the inferior parietal lobe, and also in the neighbourhood of the calcarine fissure, which is close to the cortical visual centres (Tschermak).

It must not be forgotten that a defect of lateral rotation may not only be due to *paralysis* of the muscle on the side on which the field of fixation is reduced, but that it may also be due to *spasm* of the antagonistic muscle; that is, the one towards which the eye is deviated.

If the nature of the lesion (paralysis or spasm) as well as the seat of its cause (nuclear or supranuclear) have been discovered, there is no difficulty in determining the *side* on which the lesion lies.

In a *nuclear* affection with *paralytic* deviation, the lesion is found on the same side as the limitation of movement.

If the *nuclear* lesion produces *spasm* it is seated on the opposite side to the one on which limitation of excursion exists; *i. e.* it lies on the side towards which the eye is deviated.

The contrary is true when the lesion is situated higher than the nuclear region.

Frequently the face as well as the eyes is deviated towards one side (*conjugate deviation of head and eyes*). This symptom-complex usually accompanies hemiplegia. The following rules have been established by Prevost :—

(*a*) If the head and eyes are turned towards the side *away from* the *paralysed* limbs, that is, towards the lesion producing the hemiplegia, the lesion is *cerebral* and either cortical or subcortical.

(*b*) If, on the contrary, the patient *turns his eyes towards the paralysed side*, that is, away from the lesion, the lesion lies in the *pons*.

(*c*) If the limbs are *contracted* and the patient *looks away from the affected side*, and towards the lesion, the lesion is an irritative one in the region of the *pons*.

(*d*) If the patient *looks towards* the *contracted limbs*, and away from the lesion, the lesion is irritative and situated *higher than the pons* and on the opposite side to the contracted limbs.

These symptoms are explained by the fact that the motor fibres for the eyes and face cross before they reach the pons while the fibres for the limbs only cross lower down. A

lesion in the pons affects the former after they have crossed, the latter before they cross; on the other hand, a lesion higher than the pons affects botn before they cross.

It is not uncommon to find convergence retained while the lateral movements are abolished. In such a case the lesion is limited to the nucleus of the abducens nerve and to the fibres of the medial longitudinal bundle, which connect the nucleus of the abducens nerve (VI) with that of the medial rectus (III) of the same side. Convergence is governed by the cells of the nuclei of the recti mediales, which send uncrossed fibres to the muscle of the same side, and which are placed in relation with the cortex by cells of communication (Bernheimer, Fig. 27).

When deviation of the head and eyes is accompanied by epileptiform contractions (Jacksonian), the lesion lies in the posterior portion of the second frontal gyrus of the opposite side (Horsley).

A transitory conjugate deviation is sometimes also observed in deep affections of the parietal lobe.

Some lesions of the labyrinth may produce lateral deviations of the eyes which usually are spasmodic in character (Nystagmus).

Sometimes patients whose eyes are deviated laterally in the state of rest, converge when the patient is asked to look towards the opposite side. This is due to a lesion in the pontine path of the motor fibres.

In a case reported by Spiller [1] the rectus lateralis of one side was paralysed and the lateral rotatory action of the rectus medialis of the other side was abolished, but convergence remained intact. At the autopsy a tuberculous nodule was found which had destroyed the posterior longitudinal bundle on one side and compressed the fibres of the abducens nerve in the pons (Figs. 22 and 24).

[1] *Annals of Ophth.*, July 1904.

Paralysis and Associated Spasm of the Vertical Movements.

Paralysis of upward rotation only of the two eyes is rare.[1] It is usually accompanied by paralysis of downward rotation. *Downward rotation of both eyes* is seemingly never affected without upward rotation being limited at the same time.

Some tumours of the pons, the corpora quadrigemina (especially the posterior pair), the pineal gland, or the angular gyrus produce paralysis of the associated vertical movements (Grasset, Landouzy).

Organic Affections of the Symmetrical Movements.

(Paralysis and spasm of *convergence* and *divergence*.)

If a centre of convergence exists on the median line between the nuclei of the oculomotor nerves (III) of both eyes, an irritative or destructive lesion of this region should produce spasm or paralysis of convergence.

Paralysis of convergence has been observed in lesions of the posterior part of the third ventricle and in the region of the corpora quadrigemina; also in tumours of the thalamus, corpus striatum, and cerebellum (Uhthoff).

The seat of lesion in an affection of convergence or divergence may lie nearer the crus. Thus symmetrical lateral movements as well as vertical ones are frequently affected in combined sclerosis.

Analogous affections are observed also in hysteria; they may be permanent in character, or merely take the form of a hysterical attack following a fright or a pain in nervous subjects. To confirm the diagnosis we look for the classical symptoms of hysteria—anæsthesia, analgesia (abolition of

[1] M. Landolt, *Bull. de la Société de Neurologio*, Paris, 1908 and 1911.

the pharyngeal and conjunctival reflex), disturbances of taste and smell, concentric contraction of the fields of vision, ovarian tenderness, etc.

In affections of the symmetrical movements of the eyes, the investigation of the reflexes gives additional information regarding the seat of cause of the trouble.

If the reflexes are normal the lesion is situated higher than the nuclear region, and if they are absent the lesion lies below the nuclear region.

To make sure whether we are dealing with a paralysis, or with a spasm of the antagonistic muscle, a general anæsthetic may be required.

A case has been recorded where convergence was completely absent, whilst lateral movements of the eyes were normal; the affection was congenital, no record being given as to the seat of the lesion.

IV. Bilateral Total Ophthalmoplegia.

Absolute immobility of both eyes gives the face a peculiar expression which has been named " facies Hutchinson."

The cause of paralysis of all the muscles of both eyes may be seated in the base of the brain, as, for example, where an extensive neoplasm destroys all the motor nerves of both eyes. In such a case, however, other cerebral nerves are necessarily affected at the same time, and the symptoms accompanying the ophthalmoplegia leave no doubt as to the seat of lesion.

Usually total bilateral ophthalmoplegia is nuclear in origin, and caused by a polio-encephalitis of the motor columns of the isthmus, the pons, or medulla oblongata.

According to the part to which the lesion is limited, we have either a superior or inferior polio-encephalitis (glosso-labio-laryngeal paralysis). All degrees exist between these two forms up to total polio-encephalitis. Similarly the

extent of the paralysis may vary greatly; for example, the levatores palpebrarum or the internal muscles of the eye may remain intact; similarly the muscles of the face frequently retain their normal action. Sensation is not affected.

Acute superior polio-encephalitis may come on at any age, but is most frequent in children, as also is polio-myelitis, or infantile paralysis. It is accompanied by somnolence and usually terminates fatally, owing to the extension of the process to the medulla oblongata and floor of the fourth ventricle.

Chronic or subacute total bilateral ophthalmoplegia, whether primary or secondary, usually occurs in children.

In the early stages of these affections paralysis is frequently preceded by spasm.

CAUSES OF PARALYSIS AND SPASMS OF THE OCULAR MUSCLES.

The lesions affecting the oculomotor system and giving rise to the various diseases are very varied in their nature.

Trauma may either affect the muscle or nerve directly, or produce paralysis or pareisis indirectly by hæmorrhage or exudation. For instance, paralysis of the lateral rectus frequently follows fracture of the petrous portion of the temporal bone.

Periostitis of the orbit, superior orbital fissure, or bones of the base of the skull, frequently injures the motor nerves in their course.

Disease of the sinuses (empyemas, tumours, etc.) have been mentioned as causes of oculomotor disturbances originating in the orbit.

Neoplasms of every description (sarcoma, carcinoma, gumma, tubercle, lymphadenoma, osteoma, chondroma, hydatid cysts, etc.) may modify in different ways, according to their seat, the ocular movements. It must be remembered that a brain tumour may, even indirectly, produce diseases of the eyes or the oculomotor system by increasing the intracranial pressure.

It is self-evident that diseases of the *brain*, the *medulla oblongata, spinal medulla* and their *membranes* may react on the oculomotor system.

Thus in meningitis of the base of the brain the inflammation frequently spreads to the motor nerves of the eyes.

Amongst the diseases of the brain, besides the neoplasms already mentioned we have hæmorrhages, softenings, arterial aneurysms (particularly miliary aneurysms) and thrombosis, all of which may produce ocular changes.

The following may be causal factors: general paralysis, disseminated sclerosis, syringomyelia, paralysis agitans, combined sclerosis, and, most important of all, tabes.

Infectious diseases—diphtheria, erysipelas, septicæmia, pneumonia, typhoid fever, influenza, measles, scarlet fever, varicella, acute articular rheumatism, syphilis, tuberculosis—may all bring about various oculomotor disturbances.

Albuminuria may produce motor disturbance through hæmorrhages as well as anæmia.

Intoxications—alcohol, lead, quinine, carbon bisulphide, carbonic oxide, snake-bite poison, botulism and the *auto-intoxications* (diabetes, uræmia, septicæmia)—may affect the motor tracts of the eyes in any part of their course.

In Basedow's disease, besides limitation of movements due to exophthalmos, all manner of ocular paralysis may occur: partial or total ophthalmoplegia either unilateral or bilateral; bilateral external ophthalmoplegia with paralysis of the facial and trigeminal nerves, or with paralysis of the muscles of the pharynx and those of mastication; also paralysis of convergence (Schmidt-Rimpler, Vossius); limitation of upward rotation of the eyes has been described by Posey, due to paresis of the two superior recti. The causes of these paralyses should probably be considered as toxic in origin.

Finally, we must not forget *defect in development* of the centres, as well as of the nerves and muscles.

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