microscopic examinations and substituted the anatomic dissections followed by the bacteriologic and animal tests of anatomically suspicious rats.

Of 14,184 rodents examined, 98.97 per cent. were *M. decumanus*; 0.73 per cent. *M. rattus*; 0.35 per cent. *M. rattus alexandrinus*, and 0.11 per cent. *M. musculus*. By far the greater number found infected were *M. decumanus*.

A brief summary of the anatomic and microscopic characters of acute and chronic plague in rats is given with the citation of specific instances.

The results of animal inoculation experiments show that the bacillus of natural rat plague in San Francisco is quite as virulent as the strains tested in Bombay by the last Indian Commission.

OCULAR ROTATIONS IN PARESIS.*

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At the last meeting of this Section our honored chairman, speaking of the ocular muscles, stated: "One . . . who has spent much time in investigation and has written largely in expression of his views recently said at the close of a most interesting address, 'I feel as if I were only on the threshold of this study,'" and also quoted another ophthalmologist as saying: "Ophthalmology is finished, you will find nothing new in it." These quotations are certainly reasonable cause for further thought on the ocular rotations, and perhaps the last word has not been said on this interesting subject. Carl Weiland may well ask: "Are our present ideas about the mechanism of the eye movements correct?" Furthermore, since the discussion of the paper, "Some Axioms Concerning Ocular Rotations," by our present chairman, Dr. G. C. Savage,¹ I have felt much interested in the subject of the rotations of the eye as it moves about in the field of version; the action of the ocular muscles in causing these rotations, their points of action and the phenomena presented by their pathologic condition—paresis. A careful study of the various writings on this subject has not rendered the matter at all clear to me, even though our chairman has stated that his conclusions are axioms.

To present one's individual opinions on this subject in a clear and concise manner is difficult, and to demonstrate any theory is more so, especially when we find so many diversified writings on the ocular rotations and the diagnosis of paresis. I may state that this paper will refer to eyes that have normal rotation about the center. It will deal with the subjects of increased or diminished rotation as found in that anatomic condition causing heterophoria and that pathologic condition causing paresis, with the phenomena pertaining thereto.

I wish to lay special stress on the term paresis in this paper. In complete paralysis of an eye muscle it does not seem to me that there can be any question as to the diagnosis, but in paresis the subject, according to the schemes of the writers of all our text-books, seems to be extremely complicated. I understand that neurologists make a clear distinction between these two terms, and, according to my views, the distinction between paralysis

and paresis of the ocular muscles should be clear and well defined.

In paralysis we have "the suspension or abolition of a functional power, especially in the nervous system, in which case there is a temporary or permanent loss of power of motion," etc. (Foster), while in paresis we have "an incomplete paralysis, especially when not associated with any demonstrable organic change" (Foster). Furthermore, I do not think that paresis should be classed under the conditions of heterophoria. I would consider heterophoria as an inability of the ocular muscles to perform a certain function. It is due to certain conditions, anatomic rather than pathologic, in which the muscle fails to act to its normal extent in the field of version. Hence I would call attention to the last two terms, as they apply with particular force in a study of the rotations of the eyes.

In paralysis the function of an ocular muscle is completely lost and the visual line must rotate or deviate in the direction of the muscles antagonistic and remain in that position. An exception to this is found in ophthalmoplegia externa, in which motion is completely lost in all directions and the eye remains fixed. In paresis the function of the ocular muscle is but partly destroyed and the visual line fails to turn in the direction of the paretic muscle beyond the median line of the orbit with a consequent diplopia. In heterophoria, with normal acuity of vision, we do not have diplopia when the look is in infinity. I state this again for the reason that it has been said that a diagnosis of paresis is made when the eyes are directed to the upper part of the field of version, with the visual line directed strongly to the right or left. It is also stated that in one of these positions the superior rectus acts as an elevator of only one eye, while the inferior oblique of the fellow eye raises the visual line (Duane, in Posey and Spiller). I can not agree with this proposition, as paresis of the inferior oblique, if it does occur, must cause diplopia with decided tipping of the image toward the side of the paretic muscle.

The most essential point to establish is the position of the eyes when at rest, with the look in infinity, in which the ocular muscles are in a state of normal tonicity. This position of the eyes is now well defined by all ophthalmologists as the first or primary position and one from which the eyes may be moved by the normal function of the ocular muscles to any secondary position in the field of version. Now, in this study of the ocular rotations and the primary position it must be understood that the position of infinity is not at twenty feet, but is any point in space, as a star. I wish to make this distinction, as practically we do have a certain amount of convergence at twenty feet, the usual distance for testing the vision. Furthermore, another essential is the visual acuity which must be at its best. In other words, in testing the eyes for the ocular rotations the visual acuity must not be reduced by any artificial means. Visual acuity reduced by mechanical means may be permissible at times, but it is not essential.

Bearing these two points in mind, we are now prepared to consider the rotations of the eyes, not from the standpoint of the rotations of a rubber ball or other artificial apparatus, but simply from an anatomic and a clinical point of view.

It seems to me like "carrying coals to Newcastle" to come before this association of ophthalmologists and speak of the anatomy of the orbits, but I wish to make some suggestions for your consideration in reference to

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1. THE JOURNAL A. M. A., Aug. 4, 1906.

the points of action of the ocular muscles as given in the latest publications on this subject. We find in the text-books that the origins of the ocular muscles, except that of the inferior oblique, are at the optic foramen, at the posterior part of the orbit, and that the muscles are inserted into the sclera at their respective points of attachment. All this is true, but are these points of origin and insertion also their points of action? This seems to me an important question and on its correct interpretation will depend our ideas of the mode of action of the ocular muscles. It is conceded that the origin of each of the four straight muscles is at the optic foramen, but let us trace the course of one of these muscles to its insertion. For illustration I shall refer to the superior rectus. This muscle and the other recti occupy almost the entire apex of the orbit, but quickly separating from the others the superior rectus passes upward toward the upper part of the orbit just beneath the palpebræ superioris and, surrounded by its sheath, it soon becomes tendinous. This sheath is formed of fibrous, inelastic tissue, from which strong expansions of the same inelastic tissue pass off laterally and in all directions to the bones of the orbit and to the lids and conjunctiva (Dwight in Norris and Oliver's System). The external portions of these expansions form the external palpebral ligaments. As the superior rectus reaches the back part of the eyeball it becomes tendinous and, entering Tenon's capsule, passes to its insertion into the

Fig. 1.—Frozen section of orbit 5 mm. behind globe. (From Norris & Oliver's system.) L. P., levator palpebræ superioris; S. R., rectus superior; S. O., obliquus superior; In. R., rectus internus; E. R., rectus externus; O. N., optic nerve; Inf. R., rectus inferior; F. N., frontal nerve.

sclera, 7 mm. from the upper edge of the cornea. From this we may note that a section of the orbit made 5 mm. behind the globe of the eye will present the appearance as shown in Figure 1.

The orbit being filled with fat, connective tissue and the expansions from the sheaths of the muscles and Tenon's capsule, it seems to me that the muscle and tendon of the superior rectus can have little, if any, lateral movement when the muscle contracts or when the visual line is rotated inward. The diagram (Fig. 1) taken from frozen sections shows that the superior rectus, while it can move its insertion backward or forward, moves the central part of the muscle but very slightly in a lateral direction as it is held in its position by the sheath which is attached to the walls of the orbit by these expansions of inelastic fibrous tissue. If this idea of the anatomy of the orbit is correct, and I see no reason why it should not be, then one of the points of action of the superior rectus and the other straight muscles of the eye, can not be the fibrous ring that surrounds the optic foramen, but must be at the point of the sheath just behind the globe, as shown in the section. Now when the eyeball is strongly adducted, or, in other words, turned to the limits of the inner part of the

field of version, the tendon of the superior rectus will pass through its sheath at the back part of the globe almost like a pulley at right angles to its primary line of action. We have, then, these two points of action of the superior rectus: Its insertion into the sclera anteriorly and the position of the sheath at the back part of the eyeball posteriorly. In this connection it is to be noted that the same argument will apply to all the straight muscles of the orbit which take their posterior points of action from their respective sheaths about 5 mm. behind the eyeball.

It seems to me that this is a most important anatomic proposition and one that, so far as I have discovered, is not mentioned by any of the writers on the rotations of the eye in the orbits. I have been led to a study of these anatomic conditions because, I think, they have special importance in the consideration of the rotations or movements of the eye in the field of version and particularly so in the phenomena that may be presented in all parietic conditions of the eye muscles. Furthermore, I quote from one who has written extensively on this subject this statement in reference to the action of the superior rectus: "When the eye is adducted the muscle (superior rectus) will be nearly at *right angles* to the anteroposterior axis of the eye and hence will have little or no action as an elevator."² I would also refer to an article by Dr. D. K. Shute³ on ocular rotations, in which is found the same statements in reference to the anatomic arrangements of the eye muscles, giving the muscle plane of the superior rectus as follows: "The lower edge of the muscle plane extends from one immovable point (the center of origin of the muscle) to another immovable point the center of rotation of the eye. But the upper edge of this muscle plane makes varying angles with the median plane, "as while the center of origin is immovable its center of insertion is very movable." Shute furthermore states: "The lower edge of this muscle plane forms a fixed angle with the median plane (27 degrees for the recti) whether the eyes be in the primary position or whether they be abducted or adducted."

This is undoubtedly true, but does the upper edge of this muscle plane always form the same angle or any other angle to the median plane? This is the important point and the sum and substance of this paper, because I am convinced that, while the points of this vertical plane of the superior rectus are as above stated, yet when the eye assumes any secondary position the upper edge of this plane then becomes completely obliterated and the essential points of the vertical plane of the superior rectus become, first, a fixed point, the center of rotation; second, a slightly movable point, situated in the sheath of the muscle a short distance behind the eyeball, and, third, a freely movable point, the insertion of the muscle into the sclera. Now, under these anatomic conditions, if my contention is correct, while the posterior portion of the muscle plane changes very slightly, the anterior portion or that portion anterior to a point in the sheath about 5 mm. behind the eyeball, changes according to the rotation, but in all positions of the look is practically in the vertical plane of the globe. I am fully aware that this assertion may deny that the superior and the inferior recti, to which the same argument applies, have only a slight tendency to tort or turn the eye on its sagittal or anteroposterior axis even when the eye is adducted thirty or forty de-

2. Duane, in Posey and Spiller, p. 190.

3. N. Y. Med. Jour., Aug. 18-25, 1906.

gress inward. In Figure 2 the conditions as interpreted by Duane and others are shown at A; at B is shown my idea of the anatomic conditions.

In explanation of these drawings it will be noted that in A, the two points of action of the superior rectus, we find to be the optic foramen at x and the insertion of the muscle at y' and y'' , so that, when the eye is adducted 50 degrees from the primary position, the portion of the superior rectus just back of the eyeball must pass from the position a to b . If this be true, I can not understand how this lateral motion can take place, considering the extremely slight resistance the globe of the eye, resting on a cushion of fat and tissue, can possibly offer against the pull of the superior rectus when contracted. It seems to me very doubtful if such action can occur, considering that fibrous bands pass from the sheath of the muscle to the bones of the orbit and the parts surrounding it. On the other hand, looking at Figure 2, B, we find that if these fibrous bands do tend to keep the muscle sheath almost stationary in the upper part of the orbit, that is to say, so that the muscle can not move in a lateral direction at a , then the points of action of the superior rectus must be at a and y' and y'' , as shown in Figure 2 B. Consequently, when the superior rectus is contracted by the stimulation of the third nerve, steadied by the tonicity of the internus and the externus and its vertical meridian always corrected by the action of the obliques, it must act as an elevator in all parts of the lateral field of version.

Now, all this brings forward the question: Does the inferior oblique tend to turn the visual line upward; that is, does it assist or take the place of the superior rectus when the visual line is turned in any part of the field of version? We may answer that question by asking another: Is the elevating action of this muscle needed or necessary in the ocular rotation? As I have just stated, the slightest touch on the eyeball will move it from any position, and consequently the slightest action of the superior rectus will raise the visual line to any part of the field. Therefore, the supposition that the inferior oblique of the left eye is an elevator when the eyes are turned to the right, and not the superior rectus, is beyond my understanding and hence, in my opinion, the position of the images, in the upper part of the field of version, can not indicate a paresis of the inferior oblique. Thomas Dwight,⁴ writing of this, says:

Theoretically this is tolerably certain, but practically it is to be remembered that it is very unlikely that one, or even two, of these muscles ever act alone. The accuracy of any movement is due not alone to the pull of the muscle to which it is usually ascribed, but also to the graduated resistance of the antagonist. Probably the simplest movement of the eye is made by the more or less active concurrence of all the muscles.

This is probably true, but we must consider that this "active concurrence" means that, while certain muscles turn the globe, that the other muscle assists only so far as may be necessary to keep the eye steady and the vertical meridians parallel to each other in all parts of the field of rotation. Studying the question from this anatomic standpoint, we now come to the normal action of the ocular muscles in reference to the movements of the eye in certain parts of the field of version, except those of the direct vertical and horizontal rotations. As an illustration of what I consider the true physiologic action of all the ocular muscles, I would refer to the action which takes place when the eye is rotated upward and

outward by the action of the superior and external recti. I can not improve on the excellent illustration⁵ of this action given by Dr. G. H. Price, Nashville, Tenn., at the last session of the Association during the discussion of Dr. G. C. Savage's paper, and to this explanation I would refer the reader, as it is too extensive for insertion in this paper.

Now, following this theory of the eye movements, it is to be noted that as the visual line goes upward its vertical meridian tends to tort inward, which action is corrected by the inferior oblique, and as it goes outward the external rectus tends to tort the vertical meridian outward; this action being corrected by the superior oblique. Now, perhaps this action takes place according to the law of Listing, which I will discuss later; that is to say, about an axis at right angles to its lines of direction. But it is by the action of the superior and the external recti alone that this movement is accomplished, and the only action of the oblique muscles is to correct any torting of the vertical meridians from the vertical plane of the orbit, and in this function these oblique muscles keep the vertical meridians of the two eyes parallel.

In other words, the obliques do not tend to turn or

Fig. 2.—A. Action of superior rectus as interpreted by Duane and others. B. Author's idea of the action of the superior rectus.

to move the eyeball on its center of motion either upward or outward, but simply correct the tendency of the vertical meridians to deviate inward or outward as the contraction of the straight muscles moves the visual line in the desired direction or from point to point. I fully concur with Dr. Price in his exposition of this subject.

It is natural to suppose that the eye when changing the line of vision from one point to another will move the visual line along the shortest route, and probably it does, but this condition has not yet been fully proved, though often asserted, except for the horizontal and vertical meridians. In these two meridians we know the direction of the eye movements from the after images produced by looking at a small but intense electric light. When the eye is moved horizontally the line of after images will always be perfectly horizontal and we find the same phenomenon in the vertical meridian. When, however, the eye seeks some other position in the point of vision as in an oblique direction, it is doubtful if it does move along the shortest route. Tschering⁶ says:

5. Trans. of Section on Ophthalmology of A. M. A., 1906, 432; and THE JOURNAL A. M. A., Aug. 4, 1906.
6. Physiological Optics, p 293.

4. In Norris and Oliver's System, p. 98.

The best method of studying this question (ocular rotations) would probably be to bring the look quickly from one point to another, leaving the eye exposed to a pretty intense light. The after images of the luminous source then assume the form of a line which permits some conclusions as to the nature of the movement.

Why Tschering did not illustrate this phenomenon in his classic work on physiologic optics I do not know. Following out his suggestion and my own previous experiments, it seems to me that when the look passes in an oblique direction, as up and out, the line of after images is not a straight line from point to point, but forms a slight curve with its concavity downward. When the look is directed from the first position to a point upward and to the right the line of after images is curved downward and to the left (Fig. 3). Each minute after image shows the several positions at which the eye must have made an exceedingly momentary stop. This impression of the after images was fully confirmed at the same time by Dr. E. M. Alger at the New York Postgraduate Hospital.

Now, this seems to show that the look does not always seek the shortest route when passing from point to point nor will it follow a line. It has been proved that if we draw a long curved line, and as the look follows it photograph a spot of light on the cornea, it will be found that the look does not follow the line according to its curvature, but jumps from point to point in an irregular manner until the image of the complete line has

Fig. 3.—The eyes going up and to the right along the line of the diagonal of the square, the after images present the curves shown—down and to the left.

been impressed on the retina. It may be noted, therefore, that, except in a perfectly vertical and horizontal direction, when the eye movements are controlled by two or more of the straight muscles, all the lines of direction are exceedingly irregular.

These irregular movements, it seems to me, are fully and completely accounted for by the action of the straight muscles only, the function of the obliques being simply to hold or to control the vertical meridians and by this function always to keep these vertical meridians parallel to each other during these movements. In other words, I can not consent to the proposition that the obliques are endowed with two distinct functions, that of moving the eyes in the field of version and that of keeping the vertical meridians in their correct position. They have only one nerve supply and, as Flint says, "We can not, as a rule, cause only a portion of a single muscle to contract." How, then, can a muscle have two distinct functions?

As a possible exception to the above I may mention the action of the internal recti. The action of adduction of convergence being normal, there may be an evident paresis. But this action, while unique, may readily be understood from the fact that we do have two distinct central nuclei controlling the action of this muscle,

namely, that of adduction and that of convergence. Tschering⁷ says "the muscles make possible the rotation of the globe around any axis. . . . It seems, indeed, that all six muscles are concerned each time the eye makes any motion." Very true, but, as Price has shown, the straight muscles can "make possible the rotation of the globe around any axis" and the only physiologic action of the obliques is to correct any tendency to tipping of the vertical meridians as the globe turns about any possible axis by the action of any one, two or three of the straight muscles of the eye.

It does not seem to me that these propositions can, in any way, be antagonistic to the well-known law of Listing, which says: "The eye may be brought from the primary position to any secondary position by a rotation around an axis perpendicular to the two successive directions of the visual line" (Tschering⁸). Hence a careful inspection of B (Fig. 2) will show that the obliques are not essential to any necessary rotation nor can I conceive of any possible axis around which the eye can not turn by the action of the straight muscles alone.

Now, what is the normal rotation of the eyeball with reference to the movements of the visual line in the various parts of the field of version? The examination of this field should be principally objective and must be scientifically and accurately measured, not by the usual method of having the eye follow the finger or other object, as all these methods, while useful, are not strictly accurate. For this purpose I know of no instrument so useful, accurate and scientific as the tropometer of Stevens, with which we find the direct inward rotation to be 55 degrees, the outward 50 degrees, the upward 33 degrees and the downward 50 degrees. I think that we may accept this as the standard of normal rotation and any material change in these figures in our examinations must indicate some condition of heterophoria or a paresis.

In this connection it should be fully understood that in certain cases of muscular anemia these figures may be much reduced, but always show a relative change; that is to say, a little more inward rotation than outward and somewhat more downward rotation than upward without any pathologic condition whatever.

For the objective examination of the power or equilibrium of the obliques, both in normal and in pathologic conditions, I think the clinoscope is best. It will demonstrate any tendency to declination of the vertical meridian from the normal, but I have not found these measurements of practical value in my clinical work, though they are so strongly advocated by Stevens.

Paralysis, according to the definition previously given, may be left out of this question, as it is evident that a complete paralysis of any of the ocular muscles is quickly and objectively shown in all cases. I wish to refer to the common use of this term, however, as frequently given in text-books, in which the writers refer to paralysis of a muscle when there may be only a paresis or a deficiency of rotation in certain extreme parts of the field of rotation. Furthermore, for the purpose of correct clinical diagnosis, I would draw a clear and distinct line between the terms paresis and heterophoria. Paresis is an incomplete paralysis, in which there is some pathologic condition as its primary and essential cause. This cause may exist at any point throughout the course of the nerve supply from the cerebral center to the peripheral ends of the nerve which controls the action

7. *Physiological Optics*, p. 289.

8. *Physiological Optics*, p. 200.

of the muscular structure. On the other hand, in heterophoria, as I understand it, there is an abnormal functional condition of a muscle, not due to any pathologic condition of the muscle, but to an abnormal development or an abnormal insertion of the muscle into the sclera.

Now we may not study a paresis of an ocular muscle simply from a clinical standpoint in reference to any deviation from the primary position? We must then consider the various phenomena that may be presented in our patients, with a full realization that we are examining the functions of a human eye and not the possible rotation that may be shown by a rubber ball with a knitting needle axis.

Consider the eyeball as a distinct human organ, controlled by a psychologic process not fully understood, delicately poised against its cushion of fat and tissues so that the slightest disturbance of any portion of its muscular apparatus will disturb the entire economy of the organ. Consider, further, these photographs of the ocular movements when the eye follows a curved line and these after images when the eye moves its visual line in space and then we shall make our differential diagnosis of paresis and of heterophoria much more exact and complete, though we may come in opposition with Listing's law and the schemes of our text-books.

The diagnosis of paresis of the different muscles of the eyeball, according to the various methods and schemes as laid down for our study in writings and text-books, is complicated and difficult to understand. I do not think that mnemonics will help us much, as, to my mind, they seem to be arguments made and elaborated from a mechanical device and not from clinical conditions.

I feel assured from my past limited experience that in the early stages of all cases of paresis there must be a decided diplopia if the eyes are fixed in the first position; that is, when the look is in infinity. I know that this assertion has been previously advanced by other writers, notably Linnell, and I would insist on its being the first and most important point in the diagnosis of the slightest paresis of any ocular muscle. We must concede this point, as, I think, it must be the crucial test between that of a pathologic condition and that of an abnormal anatomic condition; in other words, the differential diagnosis between paresis and heterophoria. I would say, too, that when the eyes are in the primary position, with full acuity of vision, there must be paresis if diplopia is proved, while in certain abnormal conditions, as in heterophoria, diplopia is present or may be produced by certain procedures that reduce the visual acuity, but that this diplopia does not in any way indicate paresis. I would illustrate this by the diplopia in cases of heterophoria shown by the use of the red glass or even by the Maddox rod.

Certain other phenomena have a prominent place in the text-books in reference to paresis. I refer to the crossing of the images and to the tipping of one of the images in certain pareses when diplopia is present in certain parts of the field of vision. I may well ask if these phenomena, when present in a case of paresis, are worth our serious consideration. The latest testimony on this subject says that the tipping in many cases may be due to a primary declination of the vertical meridian. Consequently "the failure of uniformity (in this symptom) may militate against the correctness of the diagnosis," and "unfortunately . . . these conditions (also crossed diplopia) are not always uniform from the

fact that homonymous diplopia is sometimes found with paresis of the inferior rectus and crossed diplopia with paresis of the superior oblique." Fuchs states that "all parietic conditions may be complicated by heterophoria." This complication makes all schemes for the diagnosis uncertain. Swanzy, in reference to the diagnosis of paresis of the superior oblique and the inferior rectus, says: "It is only necessary to remember that in one case the images are crossed, but that the tipping of the images is the same."

In a case of paresis of the inferior rectus recently examined I found no tipping of the image of the parietic eye, yet in a case of paresis of the superior oblique I found very decided tipping. This patient was seen at my clinic at the New York Post Graduate Hospital, and at the examination the images of the candle were slightly crossed with decided tipping of the image of the parietic eye. This was shown with the upper end of the candle leaning inward toward the median line, which, according to the laws of projection, shows a rotation of the upper part of the vertical meridian outward. The superior oblique is the only muscle that controls this function and the diagnosis was confirmed.

I shall not dwell on the diagnosis of paresis of the internus, nor that of the externus, as in a parietic affection of these muscles the diagnosis is comparatively easy by the usual subjective and objective tests. This is particularly so when the tropometer is used to confirm the subjective symptoms. I would state, however, that I have never noticed the tipping of one of the images so frequently referred to in the text-books.

We have remaining the elevators and depressors of the eye or the superior and the inferior recti, and the diagnosis of paresis of these muscles, according to the formulas or schemes usually given, is difficult and it is hard to determine just which muscle is affected. Yet to me it does not seem that it should be so nor that the diagnosis should be so complicated if we leave out of the question the tipping and the crossing of the images.

Take, for instance, the diagnosis of a paresis of the inferior rectus from a study of the schemes of the text-books in paresis of this muscle and that of the superior oblique. Starting from the first or primary position, we have diplopia in both conditions, but in the case of a paresis of the oblique there is also a decided tipping of the image. In paresis of the inferior rectus, Mauthner and others state that we also have tipping of the image toward the temple; but does it tip in paresis of the inferior? Only lately I examined a case of paresis of the inferior rectus, and at no position, near the region of the primary position, was there any tipping of the image of the parietic eye. I know that one case does not prove a condition; but if we do have this tipping of the image must we not eliminate any cyclophoria as a complication? My experience has shown that the examination of the field of version with the tropometer will at once locate the parietic muscle.

Granting the above, it seems to me that we can consider a paresis of the obliques in a few words. It is almost universally conceded that the essential action of the obliques is to keep the vertical meridian of each eye parallel to the median plane of the body, or, in other words, one vertical meridian parallel to the other. Consequently the slightest pathologic condition, at any part of their nerve supply, must result in a loss of this verticality of one of the meridians with consequent tipping

of one of the images. Now this may be associated with some slight horizontal diplopia due to the disturbance of the entire ocular apparatus from the impairment of so important a function by the paresis. Whether the diplopia is homonymous or heteronymous makes little difference, as I think that this will depend simply on some pre-existing heterophoria and consequently is not essential to a correct diagnosis.

The nerve supply of the superior oblique comes from the fourth nerve or patheticus, while that of the inferior oblique comes from a branch of the third nerve or motor oculi. Hence we can readily appreciate that a paresis of the superior oblique will be much more frequent than that of the inferior oblique and clinical statistics prove this. In my experience, considering this contention as to the diagnosis, I doubt some of the reports of a paresis of the inferior oblique. I have never seen such a case; why, I do not know, as even in paresis of all the other branches of the third nerve I have not noticed any special tipping of the image of a candle.

Considering the unique action of the obliques, and apart from any action they may have in moving the visual line in the field of version, the diagnosis of a paresis becomes comparatively easy. In such a case as paresis of the superior oblique, even if it be complicated with positive declination (Stevens) there may possibly be a slight vertical or crossed diplopia. But there is a very decided tipping of the image of the candle when this is used as a test of the diplopia; one image will appear vertical and the other leaning or tipped toward the median line of the head. We then rapidly decide to which eye the candle that leans belongs and we have only to remember the laws of projection for the indications of the muscle affected.

Suppose there is a paresis of the right superior oblique, then the vertical meridian of that eye must lean outward from the action of the inferior oblique, and as the rays from the upper end of the candle will fall on the lower and outer part of the retinal field they will be projected upward and inward so that, to the patient, it will appear to lean to the left. This is a subjective test, but it can be fully and objectively verified by the examination of the declination of the vertical meridian with the clinoscope of Stevens. All this will apply to a paresis of the inferior oblique, but this condition is too infrequent to require a detailed description. Linnell says: "If the left eye sees the leaning candle and the inclination is toward the right, the affected muscle is the superior oblique of that eye. If it leans toward the left the affected muscle is the inferior oblique of that eye."

If we accept the foregoing method of diagnosis we may leave out of consideration the action of the obliques in paresis of the straight muscles, except so far as that the paresis of any eye muscle may disturb the harmonious action of all the muscles.

SUMMARY.

All tests for paresis must be made with the look in complete infinity and with full acuity of vision.

We must have a standard of the field of version for comparison of any deficiency of rotation.

The straight muscles are capable of moving the eyes in any part of the field of vision and from one part of the field to another; in other words, in no part of the field will the straight muscles fail to act according to their usual function.

The obliques take no part or action in the rotation of

the eyes in the field of version; in other words, the oblique muscles do not elevate or depress the visual line.

A distinct line must be drawn between paresis and heterophoria as shown by the diplopia; in the first by diplopia in infinity and in the second by the possible diplopia with reduced vision.

In the consideration and diagnosis of paresis we may exclude the condition of crossed or homonymous images and also the tipping, except in a pathologic condition of the obliques.

The individual muscles of the eyes are not endowed with two distinct functions, as they have but one nerve supply, except possibly in the action of convergence and inward rotation due to two distinct nerve centers.

The points of action of the straight muscles of the eyes are the insertions and the sheaths of the tendons as they are situated in the orbit just behind the globe.

CONCLUSIONS.

I feel that these conclusions are correct and offer them as my personal experience in the field of ocular rotations and paresis, because in this manner they have impressed me during my clinical work. As they are so much at variance with the usual teachings of the textbooks and of the writers on ocular paresis, I trust that they will meet with an open discussion. If I have written a word that will make the subject more clear I shall feel that this paper and work have not been in vain.

I close this paper with these words of Gasser: "In the physical architecture of the eye alone is written large that Nature is an idea; a constructive, creative development; an evolution of which we are a related part."

DISCUSSION.

DR. ALEX. DUANE, New York City, said that while the sheaths of the muscles are connected with the orbit by delicate septa, and anteriorly by various fibrous bands (check ligaments), this fibrous apparatus is not so arranged as to form an unyielding plane close behind the eye, such as there would have to be for the muscle to remain rigid in its posterior portion and be reflected in its anterior portion, in such a way as Dr. Valk depicts in his Figure 2B. What, he asked, are the actual functions of the muscles, as determined by the sure test of clinical experience? If the superior oblique is not a depressor, how does it happen that a traumatic paralysis, which, it is absolutely certain, is limited to this muscle, causes a marked vertical diplopia indicating that the affected eye is too high? And if the inferior oblique is not an elevator, how does it happen that a tenotomy of this muscle causes the eye to drop, as shown by the marked and characteristic diminution produced thereby in a vertical diplopia already present? A farther and convincing proof that the inferior oblique is an elevator can be had, if the tendon of origin is picked up and traction made on it before it is divided. Such traction causes the eye to roll decidedly up and moderately out.

Again, he asked, if, as Dr. Valk's theory demands, the superior rectus acts as an elevator equally whether the eye is directed in or is directed out, how does it happen that when a tenotomy or similar cause produces a paralysis surely limited to the right superior rectus, the limitation of movement and the vertical diplopia are mainly confined to and increase very greatly in the right upper quadrant of the field of vision and are comparatively slight and ultimately *nil* in the left upper quadrant? This is a fact which can be verified, and surely indicates that the superior rectus lifts the eye but little when the latter is directed inward. And if this muscle does not act to lift the right eye when directed inward, what muscle can lift it except the inferior oblique? The same query holds for the inferior rectus, paralysis of which causes a diplopia which is marked in the outer lower quadrant and is almost *nil* in the inner and lower quadrant. In this latter part of the field the eye can still move freely down, although

the inferior rectus is paralyzed. How can it do so, unless there is another depressor besides the inferior rectus? Dr. Valk, he said, further argued that the superior and inferior recti have no torsion action, this being the exclusive property of the obliques. But Dr. Duane has repeatedly seen a pathologic torsion of at least 10° develop as the immediate result of an isolated traumatic paralysis of the superior rectus, and in one case, at least, this was one of the most disturbing effects of a complete tenotomy of the muscle. If the muscle has no torting action, how can paralysis of it produce torsion?

DR. G. C. SAVAGE, Nashville, Tenn., finds the diagnosis of paralysis and paresis of the ocular muscles exceedingly easy. It can be made in the shortest kind of time and with the greatest certainty. There must be double vision in some part of the field, when there is a paralyzed or paretic muscle. This doubling always takes place in the direction of the action of the diseased muscle. All one needs in making a diagnosis is a candle and a horizontal line. The candle alone is necessary in the paralysis or paresis of a lateral muscle. Both a candle and the line should be used in diagnosing a paresis of the elevator or depressor muscles of the eye. If the candle doubles in the right field horizontally, the doubling becoming more and more as the candle is moved towards the right, one knows that the diseased muscle is on that side of its eye, corresponding to the direction of doubling; that is, it is either a right externus or a left externus. The eye that sees the candle furthest removed from the median plane is the eye to which the diseased muscle belongs. If the right eye sees the candle furthest removed, it is the right externus. If the left eye is the one that sees the furthest removed candle, the diseased muscle is the left internus. If there is no lateral diplopia, then no lateral muscle is involved. If the doubling exists only beneath the horizontal plane, the affected muscle is a depressor, the eye seeing the lower candle is the one to which the diseased muscle belongs. If this be the right eye, then it is either the right inferior rectus or right superior oblique. To determine easily which of these two muscles is the one involved, the candle may be put aside and the patient asked to look at a horizontal line on the blackboard. With the head depressed, he will see only one line. With the head elevated, he will see two lines, the lower one being false. The false line, belonging to the right eye, will lean in one direction or another, and this leaning determines which of the two muscles is involved. If it leans towards the right, it is the inferior rectus, if it leans towards the left, it is the superior oblique that is paralyzed or paretic.

DR. WENDELL REBER, Philadelphia, called attention to the value of the Maddox rod in the estimation of paresis, especially as to torsion. It is a very easy matter, he said, to have the rods marked off with a little white ink, and estimate the torsion in all cases of paresis, particularly of the superior or inferior rectus or of the obliques.

DR. FRANCIS VALK, New York City, stated that he had tried to make the subject of his paper practical so that it could be understood. He has been in doubt over and over again in these cases, and that is what prompted him to bring this matter up for discussion. It seems to him that traumatism of, say the superior oblique, would so change the motility of the eye that that might give many symptoms. As to paralysis by complete tenotomy, Dr. Valk has seen complete tenotomies performed, and the motility remained just the same over and over again, so that he does not think that is a point in diagnosis. As to Dr. Savage's remarks, he said that it must be remembered that in all cases there may be cyclophobia complicating the paresis, then the tilting of the image amounts to nothing. Dr. Valk still holds to the opinion that the inferior oblique is not an elevator of the eye.

Sources of Tuberculosis.—It is the overcrowded dwellings of the poor that we have to regard as the real breeding-places of tuberculosis; it is out of them that the disease always crops up anew, and it is to the abolition of those conditions that we must first and foremost direct our attention if we wish to attack the evil at its root and wage war against it with effective weapons.—Dr. Robert Koch.

THE TREATMENT OF INTERSTITIAL KERATITIS.*

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I have not attempted an academic paper on the treatment of interstitial keratitis, but have rather endeavored to give a paper based on clinical observations; and in order to get the conclusions and opinions of others, the following questions were addressed to thirty representative ophthalmologists in as many different sections of the country:

1. *What local measures other than those for the alleviation of symptoms have you used in the treatment of interstitial keratitis?*
2. *To what extent have any of them contributed to the shortening of the inflammatory stage?*
3. *Do you administer iodids during the inflammatory stage? In your opinion do iodids, given during the inflammatory stage, aid or retard recovery?*
4. *What, in your opinion, is the best method of administering mercury?*
5. *What is the average duration of the inflammatory stage in your practice?*

The answers to questions one and two were surprisingly uniform. Subconjunctival injections, cold water dropping, massage, ointment of yellow oxid of mercury, electricity, dionin and many others received honorable mention, but doubtful recommendation; and not in one single instance was there a clear convincing statement that any of them had contributed to the shortening of the inflammatory or progressive stage of the disease, showing conclusively that I am not alone in the opinion that local measures (except those for the alleviation of symptoms and complications) are of no value during the stage of infiltration and vascularization.

The answers to the third question with few exceptions coincided also with my opinion that iodids internally are useless, if not positively harmful during this stage.

A large majority of the answers to the fourth question indicated preference for inunction in the administration of mercury.

The answers to the fifth question could be divided into two classes: Those from men who assume that congenital syphilis is the sole cause of interstitial keratitis and treat it accordingly, and those from men who assume that there are other causes for this disease and who do not rely on antisypilitic treatment alone. It is a significant fact that the former, as a rule, gave the average duration of the inflammatory stage in weeks, while the latter gave it in months; therefore, the majority, I think, will agree with me in the assertion that the treatment of interstitial keratitis resolves itself into the treatment of congenital syphilis, with appropriate measures for the relief of its most prominent local symptoms, the combating of complications arising from involvement of the uveal tract and for the more rapid and complete clearing up of the corneal opacities. The treatment of congenital syphilis as well as of the acquired variety consists mainly in the judicious administration of mercury with the most careful attention to diet and hygiene, and I contend that the most stubborn and persistent cases will yield to scientific endeavor in that line. Unfortunately, however, a great many of us have fallen into the habit of empirical application of routine methods in the treatment of syphilis instead of

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