improved, and the chief value which may be gained from the employment of the drug is not attained. At the age of 2 years the proper glasses should be put on the child, the lenses being selected by retinoscopy. Mr. Worth orders glasses at even a younger age, but Dr. Posey thinks that as the frames must be bound on the head by tapes, there is some danger in such young subjects, in interfering with the proper development of the bones of the face, and possibly with the eyes themselves, from pressure. The amblyoscope may be used at the age of 3 or 4 years, and if employed persistently, the results obtained are very gratifying.

DR. S. D. RISLEY, Philadelphia, declared that, in the management of squint occurring in early childhood we should not be unmindful of the somewhat limited group of cases in which the convergence is not due to the usual error of refraction, but is associated with, and probably caused by a central blind spot. It is not possible to demonstrate the presence of this in very young children, but its existence should be suspected when the well-known methods of treatment fail to correct the deviation. Dr. Risley has a number of times been able to verify this diagnosis after the child was old enough to permit a demonstration of a macular scotoma. He agreed with Dr. Posey and Dr. Moore that all cases of strabismus, when the deviation is due to refraction error, should be corrected by glasses very early in life. When seen early and when the correction is accurately made, there need be but little difficulty in securing binocular vision, even without the aid of the amblyoscope. Dr. Risley expressed surprise to hear Dr. Savage give credit to Dr. Worth for devising the use of atropin in the good, or fixing eye, to compel the use of the deviating eye. Dr. Risley has used the drug in this manner during his entire experience and the method did not originate with him so far as he knows. He called attention to the fact that in amblyopia of the deviating eye impaired vision may be due to the peculiar error of refraction, or to commencing disease in the macula, and the apparent amblyopia disappear as soon as these are corrected. Which eye shall deviate may be determined by the greater impairment of vision produced by the higher refraction error or by asymmetrical corneal meridians, for example; or it may be by a faulty attachment of one or more muscles to the globe, a condition which is often foreshadowed by certain deformities in the skull which lead to abnormalities in the shape of the orbit. It is obvious, therefore, that in the study of strabismus or other defects of ocular balance each case must be considered separately. We can formulate no law by which all cases are to be compared. We can not hope to secure by Worth's amblyoscope, or by any other instrument, binocular single vision until the refraction error has been accurately corrected, and not then in the presence of macular scotomata, or in cases where the anatomic relation of the muscles is at fault. In these last named cases the only hope is in prudent surgical interference.

DR. LEARTUS CONNOR, Detroit, stated that there is another class of cases, perhaps somewhat rare, in which nothing appears to account for the amblyopia occurring after previously good sight other than malnutrition, presumably of the retina. A year ago last October a man over 60 years old came to Dr. Connor with a history of good health previous to the past four months, when he had been gradually losing sight, until his vision was reduced to 6/200 in one eye and 8/200 in the other. The ophthalmoscopic image showed nothing except that the retinal artery was distinctly smaller in either eye than normal. The field of vision was very decidedly restricted. The refractive errors had been perfectly corrected. The only thing that constitutionally seemed to have any bearing on the case was a persistent indigestion. Dr. Connor employed vibratory massage on the eyeball and within a month vision was 20/200, and at the end of three months the patient could read Jaeger No. 1 and had 20/20 vision for distance. The only two factors observed in the case were the diminished blood supply to the retina and the poor digestion. The case puzzled Dr. Connor at the time and he is not absolutely clear about it yet, but the patient's improvement certainly followed the improved nutrition of the retina. Putting this observation with others. Dr. Connor is inclined to think

that in not a few cases there is impaired digestion and interference with nutrient processes which tend to diminish the power of the special senses.

DR. T. W. MOORE said that from Dr. Savage's remarks he inferred that Dr. Savage has been able to produce binocular vision in these cases in which the patients were over 6 years of age. Dr. Moore has tried it in a few cases, but has absolutely failed. He thinks that has been the usual result. Recently he has only tried to increase the vision in the blind eye in patients over 6.

EXPERIMENTAL AND CLINICAL EVIDENCE OF DYNAMIC (SPASTIC) ASTIGMATISM.*

F. B. EATON, M.D.

SAN FRANCISCO.

Dynamic astigmatism may be defined as a condition of the eye due to dynamic changes in its media which prevent homocentric rays from being brought to a focus on the retina.

That such dynamic changes occur there is a preponderance of evidence and opinion on the part of Javal,¹ Georges Martin,² Landolt,³ Dobrowolsky,⁴ Oliver,⁵ Noyes⁶ Panas⁷ and others. Among authors who deny this condition I note only Bull⁸ and Tscherning.⁹ The authors in the first group, while stating briefly a belief in the existence of the condition, are nearly all silent as to its clinical significance, implying, apparently, indifference to it in this respect.

As far as I know, no one has gone so carefully and exhaustively into the subjective phenomena pointing to dynamic astigmatic contractions as Georges Martin¹⁰ of Bordeaux, who published a series of papers entitled "Etudes sur les Contractions Astigmatiques du Muscle Ciliaire," which were afterward collected and published in a small volume of about 200 pages. He records in detail about 150 cases in 300 eyes, and the volume is dedicated to Javal and Schiotz. In it the subject is presented with the utmost minuteness of observation and

* Read in the Section on Ophthalmology of the American Medical Association, at the Fifty-sixth Annual Session, July, 1905.

1. Annal. d'Oculistique, t. 88, p. 33, July, 1881, p. i.

2. Ibid., 1886-1887.

3. Refraction and Accommodation of the Eye, pp. 301, 302.

4. Arch. f. Ophth., xix, p. 51, 1868.

5. Norris and Oliver, vol. iv, p. 420.

6. Diseases of the Eye, 1890, p. 111.

7. Traité des Maladies des Yeux, vol. i, p. 141.

8. Annal. d'Oculistique, Feb., 1892.

9. Optical Physiology (English Transl.)

10. Annales d'Oculistique, 1886-1887.

record in nearly every aspect in which it is possible to view it. When Martin's papers appeared, seventeen years ago, they attracted my attention because I observed facts clinically which apparently corroborated his. I was not convinced, however, though again and again, in the years following, in doing refraction work, I was reminded of the subject by phenomena similar to Martin's records.

It was not until 1902 that I began systematically to record these observations, and I was startled to find how accurate were his records when duly analyzed. In comparing his findings with mine, I found a difficulty owing to his peculiar method of marking his astigmatic dial and his trial-frame cells, and probably this has proved an obstacle to others in this country, since we follow the mathematical protractor.

To save time in recording cases, Martin adopted certain signs of abbreviation. To indicate that the dial lines appear equal, he wrote the sign $\odot$ which exists in typography, especially in works on botany. If a line on the dial appeared clearer than the rest, he wrote the sign $\bigcirc$, writing by the side of it the number of degrees of said line. The principal portion of Martin's treatise deals with three forms of dynamic contractions: *Contractions rénitentes*, which I translate tonic contractions; *contractions élastiques*, which I translate clonic contractions, and *contractions résistantes*, which, of course, are the same in English. About half of the

through the vertical, and b those through the horizontal meridian. Fig. 2 represents their path before the use of atropin with $-0.50c.$ ax. 180° before the eye. Here the $-0.50c.$ ax. 180° has thrown the vertical meridian rays back to that extent (myopia 1.00 D.) where, by a dynamic astigmatic contraction affecting the horizontal meridian rays, all rays meet at -1.00 D., the result being a myopia of 1.00 D. with equalized appearance of the astigmatic chart ($\odot$), but vision of $1/1$?

Fig. 3 shows the path of the rays with a $-0.75c.$ ax. 180° before the eye.

Now, the vertical meridian rays are thrown back to that extent, but the contraction affecting the horizontal meridian rays has relaxed; i. e., according to Martin the contraction is elastic (clonic); for, at the very first test, with no lens before the eye the chart lines were equal, and the myopia 1.50 D., the contraction has relaxed 1.50 D. -0.75 D. $= 0.75$ D., and the result is a myopia of 0.75 D. with the chart lines equalized ($\odot$) and $V. = 1/1$. Fig. 4 shows the path with $-1.00c.$ ax. 180° before the eye. Here the vertical meridian rays have been thrown back so that in that meridian there remains only 0.50 D. of myopia. But now the contraction affecting the horizontal meridian rays does not relax, the rays still meet at 0.75; hence the horizontal line

Fig. 5.

Fig. 6.

treatise deals with what Martin terms "associated partial contractions," or astigmatic contractions in an astigmatic and "active" eye, producing, by the law of associated movements (Vulpian), a functional astigmatism in the other, "passive" and non-astigmatic eye.

This paper permits me only to outline Martin's clinical methods and findings, to adduce some of my own, and to describe briefly certain subjective and objective investigations of my own and other eyes, which apparently prove dynamic astigmatism caused by the accommodative apparatus on the one hand, and by the extraocular muscles on the other.

I present first some simpler examples of Martin's cases, using the notation of our own charts and trial frame, but premising that I do not assume the correctness, as he did, of Javal's dictum of 1887, that the corneal astigmatism constitutes, almost without exception, the total astigmatism. I therefore select some of the cases in which atropin was used three or more days.

Miss —, aged 17. Intense chorionic blepharitis. Javal (ophthalmometer), 1.50 D. to rule. O. D. V. $1/1$? Em. $\odot$ w. + 0.50s. $\odot$ 90.

w. $-0.50c.$ ax. 180° $\odot$ $1/1$?

w. $-0.75c.$ ax. 180° $\odot$ $1/1$

w. $-1.00c.$ ax. 180° $\odot$ 180 $1/1$?

Under atropin, w. $-1.50c.$ ax. 180° $\odot$ $1/1$

Martin's tests may be graphically analyzed thus:

Under atropin the course of the rays is as in Fig. 1, a being the position of the focal line of those passing

Fig. 7.

Fig. 8.

Fig. 9.

on the chart becomes the clearest. According to Martin, there is, therefore, a tonic (rénitente) contraction of 0.75 D. affecting the horizontal meridian. The result is astigmatism and $V.$ lessened to $1/1$.

A more striking case of Martin illustrates a much higher degree of tonic contraction:

L., aged 9. Corneal astigmatism 2.50 D. to rule.

Without atropin O. D. V. $= 2/5$? $\odot$; w. $-1.00c.$ ax. 180° $\odot$ V. $1/2$.

Without atropin O. D. V.; w. $-1.25c.$ ax. 180° $\odot$ V. $1/2$.

Without atropin O. D. V.; w. $-1.25c.$ $\odot$ $-1.00c.$ ax. 180° $\odot$ V. $2/3$.

Fig. 5 shows the path of the rays under atropin, producing simple myopic astigmatism.

Fig. 6 shows the path of the rays before the use of atropin, with $-1.00c.$ ax. 180° degrees before the eye. The cylinder has thrown the vertical meridian rays back to a focal point of 1.50 D. But the test now showed equalized chart lines, and vision only improved to $1/2$. Martin therefore concludes that an astigmatic contraction of 1.50 D. affected the horizontal meridian lines, producing $\odot$ but leaving a myopia of 1.50 D.

Fig. 7 shows the path of the rays with $-1.25c.$ ax. 180° degrees before the eye. This cylinder has thrown

the vertical meridian lines back to the focal point, 1.25 D., but the contraction affecting the horizontal meridian lines has relaxed (clonic), and the vertical line of the chart is the clearest.

Lastly, with $-1.25s. \ominus -1.00c. ax. 180$ degrees, the chart lines are equalized and vision becomes $\frac{2}{3}$, equal to the vision under atropin with full correction by $-2.50c. ax. 180$ degrees. The contractions of the horizontal meridian thus neutralized are shown in Fig. 8; the black lines indicate a myopia in the vertical meridian of 2.50 D., and the tonic contraction affecting the horizontal meridian rays, of 1.25 D. The dotted lines indicate both the effect of the cylinder on the vertical meridian rays, and the disappearance of all contraction in the horizontal meridian under atropin.

The highest degree of dynamic astigmatism I have seen was in the following case:

W., aged 41. April 4, 1904, complained of defective vision and severe headaches. He has never worn glasses. He has been exposed late at night to electric light. Eyes are very irritable.

Examination.—V. O. C. = 21/29 $\odot_{no}$. Neither minus or plus cylinders or spheres improved vision. Hyoscin hydrobromate .25 per cent. for 48 hours.

V. O. D. w.—3.00 cyl. ax. $5^\circ = 21/10???$, $\odot$

V. O. S. w.—3.00 cyl. ax. $175^\circ = 21/16$, $\odot$

The path of the rays with cycloplegic in O. D. is shown in Fig. 9.

Since both spherics and cylinders were refused before the use of the cycloplegic, there was a tonic dynamic contraction affecting the 5° meridian rays of 3 D.

Fig. 10

Fig. 11.

Fig. 12.

Fig. 13.

Among a large number of my own cases of the condition under consideration I select two examined after twelve and eleven years, respectively, as of value in showing how persistent are dynamic contractions, and how they may mask the true static refraction.

S. S., girl, aged 17, was seen first in 1892. Subjective tests then gave myopia corrected by: O. D.—5.00 sph. $\odot$ O. S.—2.25 sph. $\odot$

In 1895, aged 20 by skiascopy: O. D.—5.00 sph. O. S.—2.25 sph.

January, 1895: Atropin 1 per cent. for four days. Skiascopy: O. D.—4.50 sph. $\ominus -0.25c. ax. 90^\circ$! O. S.—2.25 sph.

Trial case: O. D. V. w.—5.00s= $20/20??$ $\odot$. O. S. V. w.—2.75s= $20/20$ $\odot$

In 1904, nine years later, aged 29, Javal: O. D. 10 ± 1.37 D. O. S. $150^\circ \pm 0.50$ D.

O. D. V. w.—4.00 sph. $\ominus -0.75$ cyl. ax. $10^\circ = 21-19???$ $\odot$

O. S. V. w.—2.50 sph.= $21/19, 21/13???$

The path of the rays in O. D. in 1895 is shown in Fig. 10; in 1904, in Fig. 11.

Comparison of cuts shows a disappearance in 1904 of a tonic contraction affecting the meridian of 10 degrees, of 1.00 D. which atropin had not uncovered.

N. L., girl, aged 10, 1894. Asthenopia. Atropin 1 per cent. instilled for one week:

O. D. V. w.+1.75s. $\ominus -0.50$ cyl. ax. $10^\circ = 6/5$, $\odot$

O. S. V. w.+1.75s.= $6/5$, $\odot$

A year later, skiascopy gave the same refraction in each eye, and Javal O. S. 1.25 to rule. Fig. 12 shows the path of the rays under atropin in 1894, in O. D.

January, 1905, aged 21. She has worn, for near only, a pair of +1.25 cylinders, axes 90° ; has done much drawing. O. S. mucopurulent conjunctivitis.

Trial case: O. D. V. w.+1.50s. $\ominus -1.25c. ax. 170^\circ = 21/16$, $\odot$
O. S. V. w.+1.50s. $\ominus -1.25c. ax. 10^\circ = 21/16$, $\odot$

The path of the rays in O.D. in 1905 is shown in Fig. 13, which, when compared with Fig. 12, shows that in 1905 there was a contraction in the O. D., changing the principal meridians from 10 degrees and 100 degrees to 170 degrees and 80 degrees. Also that there was a general contraction of 0.25 D., and a tonic dynamic contraction in the meridian of greatest refraction of 0.75 D. It might seem well that a cylinder of 1.25 D. was accepted in 1905, as against one of only 0.50 D. accepted under long use of atropin in 1894. But the discomfort and irritation of the eyes and the conjunctivitis favor the original atropin correction. How often, after careful correction of compound hyperopic astigmatism, we are forced to increase the sphere, and to decrease the strength of the cylinder.

That Javal was himself the subject of a high degree of astigmatism against the rule is, I think, perhaps fairly well known. That his eyes corrected this, in his opinion, is not, I think, so generally known. At the International Medical Congress at London in 1881 he cited these facts, stating that when 16 years of age he saw the stars as perfectly round points when he made

an effort difficult to maintain. When 24 his manifest astigmatism was about 2.00 D., and at 44 it was 2.50 D. Javal recorded one of his cases, M. L., whose corneal astigmatism at the age of 34 (1865) was 6.50 D., and the subjective error but 4 D., indicating to Javal a dynamic contraction of 2.50 D. In 1869 the subjective astigmatism was 6 D., and in 1881 7.50 D.

Samuel Theobald¹¹ in 1885 cited three similar cases in hyperopes observed during twelve and sixteen years in which the astigmatism increased from 1 to 3 D.¹⁰

FREQUENCY OF THE CONDITION.

Dynamic astigmatism, in my own experience, exists very frequently, and nearly always in tired, irritable eyes which are affected with organic (corneal or lenticular) astigmatism. It is often overlooked simply because it is so often thought that the ciliary muscle and the convergence excess have been paralyzed completely by some weak or strong cycloplegic.

We say: "The tongue can no man tame." Who can tell when he has subdued the ciliary muscle? There are times when no man can tame it; nor are such times infrequent. I have tested hyperopic eyes which accepted no cylinders, though the corneal astigmatism was 1.50 D., yet by patient, so-called "fogging" for

11. Amer. Jour. Ophth., July, 1885.

days nearly this whole amount became manifested, together with a higher degree of hyperopia.

EFFECTS OF AGE, STATE OF HEALTH AND TEMPERAMENT.

It has been generally noted that dynamic astigmatism predominates in youth and disappears at the age of 40 or 50. As to disappearance at the pre-byopic age, Javal and Martin note numbers of cases as exceptions, and I have seen undoubted ones between 50 and 65, and once in a woman of 73. General debility, the asthenic state, etc., prevent dynamic contractions, which, in the young, reappear when the patient recovers normal vigor. Martin exhibits a conservatism that points a lesson to some extremists of this day who would disenfranchise migraine as a distinctly nervous disease. He states: "The contraction alone is incapable of producing neuralgic crises; it is necessary that it occur in a subject constitutionally affected with a diathesis (arthritis, or herpeticism)."

MECHANISM OF DYNAMIC ASTIGMATISM.

This is not easily explained, for there is even yet no universal agreement as to the mechanism of accommodation. Martin² disavows any intention of asserting that certain fibers of the ciliary muscle contract, but only that the partial contraction in question causes a convexity of the lens in a certain meridian. Dr. Chas. A. Oliver¹² remarks that during astigmatic tests "it

Fig. 14.

will at times be found not only that the amounts of the defect change when the examinations are made with the two eyes simultaneously, but that the relative angles of the two principal meridians likewise change. This is possibly to be accounted for in a measure by the action of the extraocular muscles in producing, both directly and indirectly, different degrees of tension on the eyeballs during their combined functioning."

Priestly Smith¹³ cites a case in which in one eye there was total paralysis of the externus and internus, the other muscles being intact. The patient complained that vision of this eye was impaired. The ophthalmoscope showed no change in the fundus or media, but skiascopy showed 1.00 D. of astigmatism in the vertical meridian corrected with a -1.00 D. cylinder axis horizontal to normal vision. On recovery from the paralysis, all traces of astigmatism vanished.

This brings me to some investigations of my own, made on my own eyes and on those of others, in the endeavor to estimate the relative importance of the action of the accommodative apparatus on the one hand and of the extraocular muscles on the other, in producing dynamic astigmatism. These comprise two kinds of experiments:

1. The objective examination of the corneal curvature by the ophthalmometer with the eyes at rest, and the changes in curvature and direction of the meridians due to more or less convergence and accommodation.

2. Tests with the astigmatic chart at 20 feet in a static state of rest in the primary position, and the changes in the meridians and astigmatic curvature produced by concave spherical lenses resulting in increased accommodative effort with overaction of convergence (esophoria).

Subjectively, I have simple hyperopic astigmatism of 0.75 D. in each eye, accepting + 0.75c. ax. about 75 degrees, O. D., and same cylinder axes, exactly 105 degrees, O. S.

My corneæ have been repeatedly and carefully measured by different observers, and I have the faculty of converging moderately or excessively with either eye alone, the other remaining motionless in the primary position. On converging, the reflected and tangent images of the mires are always seen to separate in the meridian of least curvature 0.25 D., and to overlap in the one of greatest curvature from 0.12 to 0.25 D. Measurements on the arcs of the instrument give like results. Besides this, the principal meridians of each eye are shifted. Each nearly horizontal meridian inclining nasally 15 degrees, and each nearly vertical one outward 10 to 15 degrees, as shown in Fig. 14.

Taking the left eye, when converging the right on a point 8 to 10 inches in the median line, the tangent images at meridian 15 degrees of the left cornea are

Fig. 15.

seen to separate from 0.25 D. to 0.37 D., and to overlap about 0.12 D. near the meridian of 105 degrees. I say *near* these meridians because the principal meridian 15 degrees now becomes 30 degrees, and 105 degrees becomes 90 degrees; the more horizontal (15 degrees) inclining nasally 15 degrees, and 105 degrees inclining temporally 15 degrees.

A fellow physician has in the left eye a corneal astigmatism 15 degrees $\pm$ 0.75 D. On convergence, in contrast to my own case, the horizontal meridian inclines outward 6 degrees and the vertical 9 degrees inward, the curvature increasing slightly in each. (Fig. 15, the dotted lines indicate the meridians during convergence.)

In order to determine whether or not by subjective tests there is dynamic astigmatism produced in my left eye by simultaneous convergence and accommodation, and whether this bears any direct relation to the corneal asymmetry similarly induced, during three years I have proceeded as follows: With my correction, +0.75c. ax. 105 degrees, before this eye, the right covered, I regard an astigmatic chart and tangent scale at 6 meters. The dial lines appear equal and clear; exophoria 2 degrees. Now, placing a minus 3.00 D. sphere before the left eye and cylinder, and by a strong effort of accommodation overcoming the induced hyperopia, esophoria of 18 to 20 degrees is induced (9 to 10 degrees

12. Norris and Oliver, vol. iv., pp. 430, 431.

13. Ophth. Rev., vol. iv, p. 354.

of actual deviation, equivalent to convergence on a point of close to 8 inches). The chart lines 30, 45 and 60 degrees now alone appear distinct, and all the lines are equalized by $+0.50$ cyl. axis 135 degrees, indicating either a decreased relative curvature in the corneal meridian 45 degrees, or an increased one in meridian 135 degrees. The result of this experiment never varies, and it proves conclusively the production of dynamic astigmatism due either to accommodation or convergence, or both. Apparently, there is a direct relation between the corneal astigmatism produced in the left cornea by both objective and subjective experiments (Fig. 14).

The inclination of the principal meridians in my own eyes can not be explained by the outward physiologic torsion, which, according to Le Conte's laws, occurs in horizontal convergence from the primary position; for experiments show that this outward torsion of my eyes, converging to 8 inches, amounts to only 3 degrees, whereas, as seen in Fig. 14, the outward inclination of the nearly vertical meridians amounts to 10 and 15 degrees, while the nearly horizontal ones actually incline inward. There is, therefore, a distortion of the corneæ, and this, I believe, is due to pressure during convergence by the superior, inferior and internal recti. This pressure is quite as great in marked esophoria of accommodative origin.

In concluding this paper, I insist that dynamic astigmatism is of practical clinical importance, and the gist of my demonstration is a plea to consider in many cases the total refractive error in doubt until time, cycloplegic's rest, aided if necessary by the hypodermic use of hyoscin hydrobromate and bromids or other sedatives, with patient "fogging" at times, has eliminated all dynamic contractions. The condition is frequent, not rare, and failure to recognize it often accounts for the discrepancies in the refractive findings of competent ophthalmologists.

590 Sutter Street.

DISCUSSION.

DR. WILLIAM CAMPBELL POSEY, Philadelphia, declared that the paper offered a satisfactory explanation to him of his failure to relieve some three or four cases during the last two or three years. He had refracted each eye separately very carefully, under a mydriatic, and had established the fact to his satisfaction that the axis of astigmatism in each eye was, say, at 90 degrees. The lenses were then ordered with the cylinders at this axis. In a few days, however, the patients returned, saying that the glasses did not give comfort, and that they noticed that if the lenses were slightly tilted they gave better vision and comfort. The work was then gone over a second time, only to find that when tested monocularly the axis was at 90 in each eye; with both eyes open, however, and the lenses in position, it was found that the axis of the astigmatism in one of the eyes had shifted about 10 degrees. That this change was due to an unusual action of one of the extraocular muscles on the eye Dr. Posey thinks there can now be no doubt.

DR. D. DE BECK, Seattle, Wash., related that during college life he was perfectly well in every way, but suffered from asthenopic headaches and in the beginning of his medical studies he still suffered; when he began eye work he found that either eye could read 20/20 readily. His eyes were tested under atropin by his late teacher, Dr. Seeley, who found that he had a simple hypermetropic astigmatism of 1.50 D., with an inclined axis, 105 in the right and 75 in the left. With these he saw perfectly well and has worn that correction for twenty-five years. He also had a slight but well marked cyclophoria. On taking off these glasses at that time he would find that he had a vision of 20/70, but that in a little while he could work down the line until he could read 20/20. Putting on the

glasses would for a few moments blur the vision and then he would again have 20/20 vision. In other words, he could adjust for the defect, but as years went by the adjustment became less and less perfect. This personal observation Dr. de Beck thought was in line with Dr. Eaton's experience.

DR. MARK D. STEVENSON, Akron, Ohio, said that when lenses having strong cylinders are prescribed, he rotates them in their frames until they are in the correct position before the eyes, so that the axis of each cylinder may correspond to one of the chief meridians of each eye. In doing so he has observed that the directions of the principal meridians sometimes change when binocular single vision replaces the vision of one eye. He has also observed this change during the act of accommodation.

THE INCIDENCE OF HEART DISEASE IN SAN FRANCISCO.*

WILLIAM FITCH CHENEY, M.D.

Professor of Principles and Practice of Medicine, Cooper Medical College, and Physician to Lane Hospital.

SAN FRANCISCO.

My object in writing this paper is to throw some light on the frequency of organic heart disease in San Francisco; on the relative frequency of the various forms; and on the factors that make the frequency what it is. In order to accomplish this purpose I have drawn on two sources of information. The first of these is the records of the General Medical Clinic in the dispensary of Cooper Medical College; and the second is the autopsies performed by my colleague, Dr. William Ophüls, professor of pathology in Cooper Medical College. In the medical clinic I have had opportunity for personal investigation of the cases concerned in the preparation of this paper, and know the facts to be as stated. As regards the autopsies, they were all made by Dr. Ophüls himself, who has kindly placed his personal records at my disposal.

Some explanation must be given at the outset as to what has been included under the term "heart disease." It is so often difficult to decide that the heart is really unsound, and that the heart symptoms of which complaint is made are not, in fact, due to disease of some other organ. Every clinical investigator must have certain rules for his guidance, and it is only fair to state first of all what rules have been followed in determining the presence of heart disease in the cases herewith reported.

First.—Although it may seem superfluous to state that no case has been diagnosed on symptoms alone, yet this statement is not superfluous, because such diagnoses are sometimes made.

Second.—No case in the group reported has been diagnosed on physical signs alone, especially not by the mere presence of murmurs. Nothing is more familiar to the teacher of medicine than the certainty with which a medical student rushes from the discovery of a murmur to the conclusion that valvular disease exists; while with increasing experience the murmur itself becomes less and less important as an indication of organic disease. I would not have it suspected, therefore, that diagnoses have been made in this way among the cases collected. On the contrary, so great has been the anxiety for conservatism in diagnosis, that no cases have been set down in the records as heart disease unless besides the murmur there was definite evidence present of alteration in the area of cardiac dullness, with symp-

* Read in the Section on Practice of Medicine of the American Medical Association, at the Fifty-sixth Annual Session, July, 1905.