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A
BRIEF REVIEW OF THE WORK
OF
DONDERS

BY
ERNEST CLARKE



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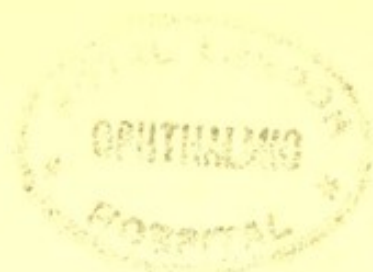
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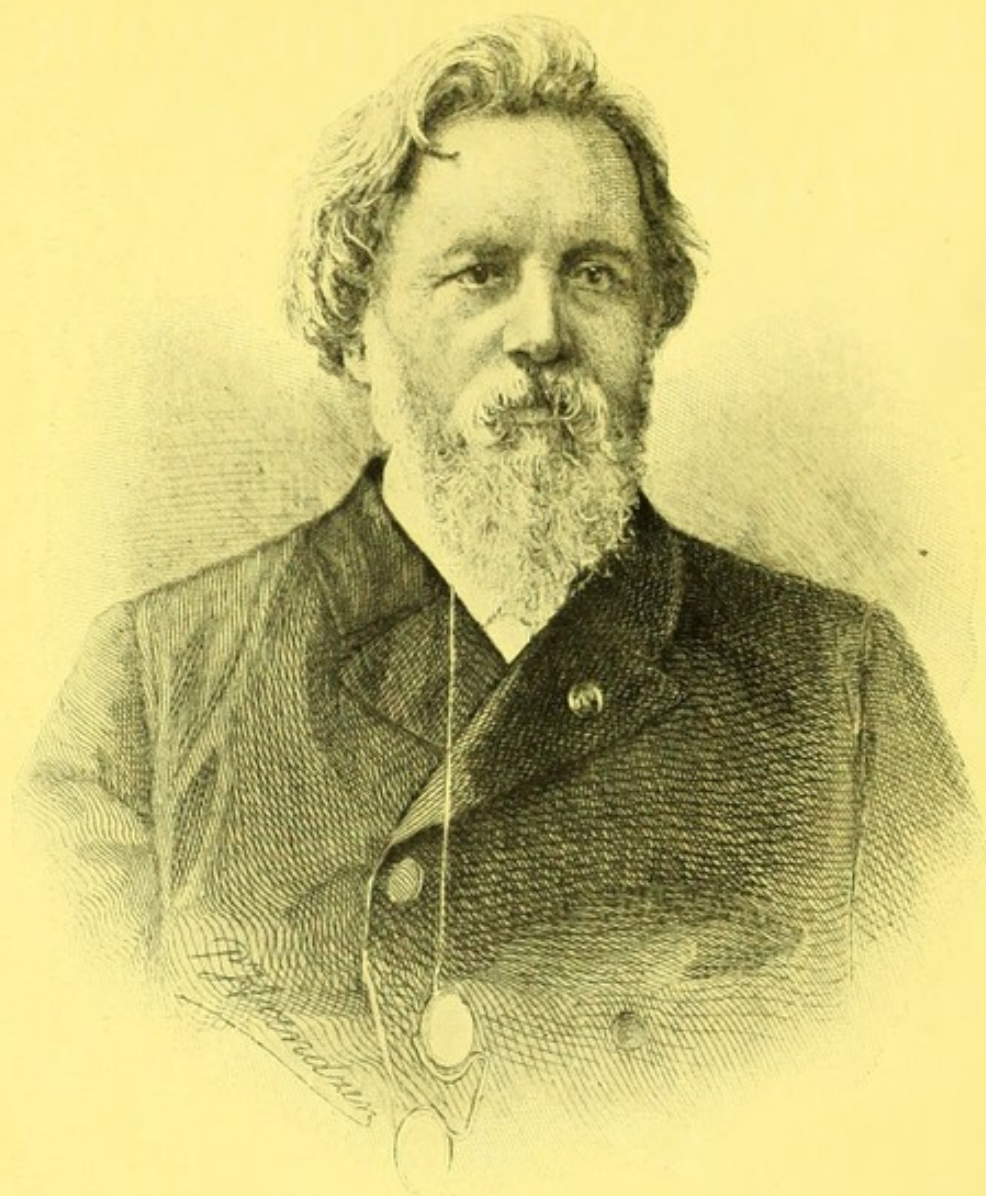
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J. C. Donners

PROBLEMS IN THE ACCOMMODATION
AND REFRACTION OF THE EYE

A
BRIEF REVIEW OF THE WORK
OF
DONDER'S

AND
THE PROGRESS MADE DURING THE LAST
FIFTY YEARS

BY
ERNEST CLARKE, M.D., B.S., F.R.C.S.

SENIOR SURGEON TO THE CENTRAL LONDON OPHTHALMIC HOSPITAL
CONSULTING OPHTHALMIC SURGEON TO THE MILLER GENERAL HOSPITAL



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1914

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TO THE
UNDYING MEMORY OF THE
GREAT DONDEERS

PREFACE

As fifty years have elapsed since Donders gave us his classical treatise on "Anomalies of Accommodation and Refraction of the Eye," it seems a fitting opportunity to remind the medical profession throughout the civilized world in what that work consisted, and to instil into their minds the deepest feelings of gratitude and admiration. The practical portion of the subject has alone been treated. Added to this résumé of Donders' work is a brief record of the progress made since his time.

Donders' classical treatise was published first in English (it was never published in Dutch!), and, as a very small return compliment, this little book has been published first in Dutch.

The chapter on "Presbyopia" was read as a paper in the Ophthalmological Section of the Seventeenth International Medical Congress, August, 1913.

E. C.

CHANDOS STREET,
CAVENDISH SQUARE,
LONDON, W.

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ACCOMMODATION

THE MECHANISM OF ACCOMMODATION.

It seems almost a reflection upon physiologists and oculists that, although fifty years have passed, we are practically in the same position as far as our knowledge of the mechanism of accommodation is concerned as when Donders left it. With his clear reasoning, by a process of exclusion, he examined all the theories then extant, and stated that the ciliary muscle was the *only muscle* used in accommodation, and that the action of this muscle was to produce a greater convexity of the anterior surface of the lens, and that this surface approaches the cornea, that the posterior surface of the lens does become slightly more convex, but remains stationary, and consequently the lens becomes *thicker* in the centre (page 16).* He was quite positive that without a lens no accommodation is possible, and he says, "My investigations have led me to the conviction that in aphakia† not the slightest trace of

* The pages after Donders' quotations refer to "The Anomalies of Accommodation and Refraction of the Eye" (Donders, 1864). New Sydenham Society.

† "Aphakia" was the name given by Donders to the condition in which the lens is absent from the dioptric system, and the term has been universally adopted.

accommodative power remains" (page 320). How the ciliary muscle produces its action on the lens he declines to state. He mentions the Cramer and Helmholtz theory, which demands that at rest the lens is in a state of tension, and during accommodation is allowed to become convex by virtue of its elasticity; and Müller's theory, which asserted that during the accommodation the margin of the lens was squeezed by the ciliary muscle, making the centre of the lens thicker. Cramer, in "*Het accommodatie-vermogen*," Haarlem, 1853, published his investigations, which were followed the next year by those of Helmholtz.

Fifty years have passed, and the matter is still *sub judice*. The Helmholtz School, headed by Hess, maintain that during rest the lens is compressed by its capsule and kept flatter; and that when the ciliary muscle contracts, it pulls forwards and inwards this capsule, which thus becomes relaxed, and allows the lens to assume a more convex form.

Grossmann (*Ophthalmic Review*, 1904), gives the results of his investigations, and cites a case of *congenital aniridia* where the lens was observed to be tremulous under the strong action of eserine. A tremulous condition of the lens under strong accommodation would be fatal to the Tscherning theory.

It seems that all physiological proof is on the side of the Helmholtz theory, but there is no doubt that on the clinical side Tscherning's arguments assume a very strong position.

Tscherning says* that the action of the ciliary muscle is to increase the tension on the fibres of the suspensory ligament, and to alter the lens from a spherical to a hyperboloid form. The lens thus becomes more conical under accommodation, and the contraction of the pupil that takes place at the same time masks the increased aberration which results from the flattening of the periphery.

The clinical proofs that favour the Tscherning theory are—

1. There is not the smallest possible doubt that very small errors of astigmatism (of the cornea) can be, and are, corrected by an unequal contraction of the ciliary muscle, producing an opposite astigmatism of the lens. It is difficult to understand how this can occur except on the Tscherning theory.

2. We know that some young subjects, by powerful effort, can produce an accommodative power equal to 17 D or 20 D! Is it possible to get this effect by a simple relaxation of the lens capsule? Does it not point rather to a squeezing of the periphery of the lens, which thus causes the lens—which is so soft in youth—to bulge anteriorly and centrally?

3. And, finally, Is not the Helmholtz theory totally against our conception of the meaning of rest.

But, after all, we *know* that it *is* the ciliary muscle

* Tscherning, "Physiologic Optics," page 183. Enlarged English edition, 1900.

that does the work of accommodation, and we know what that work is, and for all practical purposes that is enough. It is only a matter of interest to know by what mechanism the work is performed.

RANGE OF ACCOMMODATION.

At rest the eye is adapted for the most distant point it can see distinctly—viz., its punctum remotum (R) (page 31), while the greatest possible contraction of the ciliary muscle adapts the eye to the nearest point it can see distinctly—viz., its punctum proximum (P). The force required to change the eye from R to P is called the “Range or Amplitude of Accommodation,” and is represented by the *difference* between the refraction of the eye at rest and when doing its utmost work.

The range or amplitude of accommodation

$$= \frac{1}{P} - \frac{1}{R} \text{ (page 28).}$$

Expressed in dioptries Donders' equation is $a = p - r$, where a equals the number of dioptries representing the accommodation, p equals the number of dioptries represented by the eye when in a state of maximum refraction, and r equals the number of dioptries represented by the eye at rest—*i.e.*, when adapted for its farthest distinct point. Hence p equals the dynamic refraction of the eye, and r the static refraction.

When the eye is emmetropic $a = p - \frac{1}{\infty} = p$,

that is, the amplitude or range of accommodation is represented by its nearest distinct point.

When the eye is hypermetropic, as R has a negative value, the equation is—

$$a = p - (-r) = p + r.$$

In myopia, as R is positive, the equation remains

$$a = p - r.$$

THE INFLUENCE OF AGE UPON THE ACCOMMODATION.

Fig. 1 is Donders' well-known diagram altered by Nagel to the metrical system. It shows the

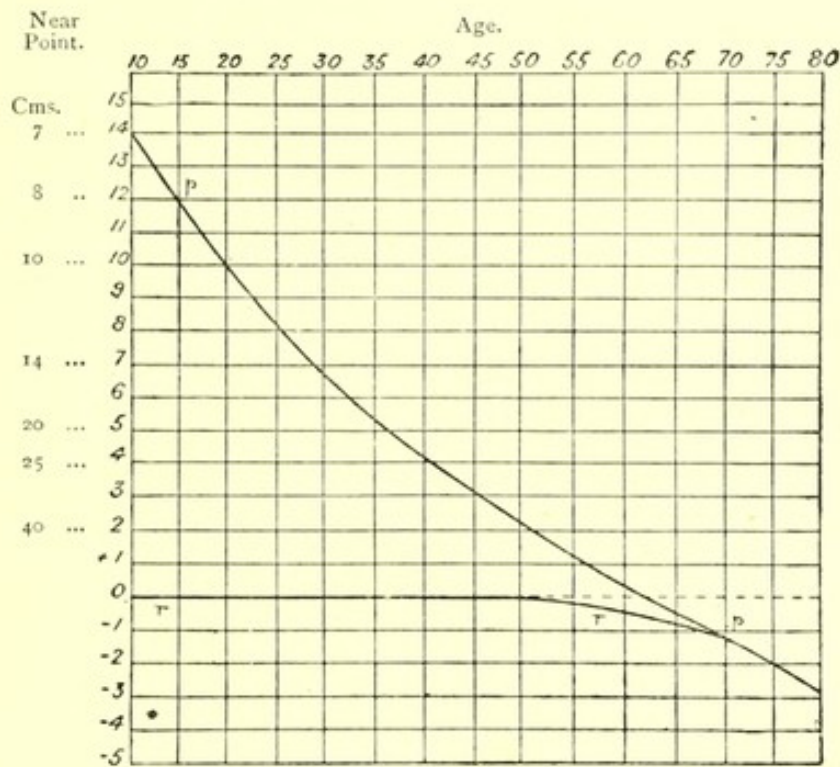
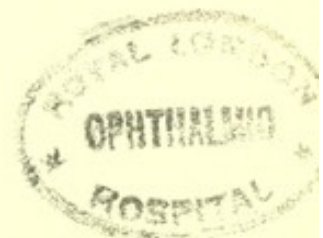


FIG. 1.

average range of accommodation of an emmetrope at different ages. r is the far-point line, in this



case coinciding with the zero line, until acquired hypermetropia commences at age fifty-three, and p is the near-point line curving down and meeting r at age seventy, when all accommodation ceases. The subject is more fully discussed under Presbyopia (page 16).

So far each eye has been examined alone; when examined together, we get what Donders called the *binocular range of accommodation*.

AMPLITUDE OF CONVERGENCE.

We still use Donders' formula, and expressing the equation in metre angles, we get—

$$Ca = Cp - Cr,$$

where Ca is the amplitude of binocular convergence expressed in metre angles, and Cp and Cr the near-point and far-point also so expressed.

Further, Donders supplied us with a graphic diagram (Fig. 2) illustrating that, although accommodation and convergence are intimately connected, still, "without a change of convergence, the accommodation can be modified" (page 110), and also that "the convergence may be altered without modifying the accommodation."

We can experimentally prove to ourselves the truth of Donders' teaching by altering our accommodation without changing our convergence, as, in looking at an object with both eyes, before which we place weak convex and concave glasses; and also by altering our convergence without changing our

accommodation, by placing before the eyes weak prisms, base in or out.

The amount of dissociation between the accommodative and convergence efforts is limited, and varies *with* and *in* the individual; it can be increased by practice, and it differs for varying degrees of accommodation and convergence. Fig. 2 shows

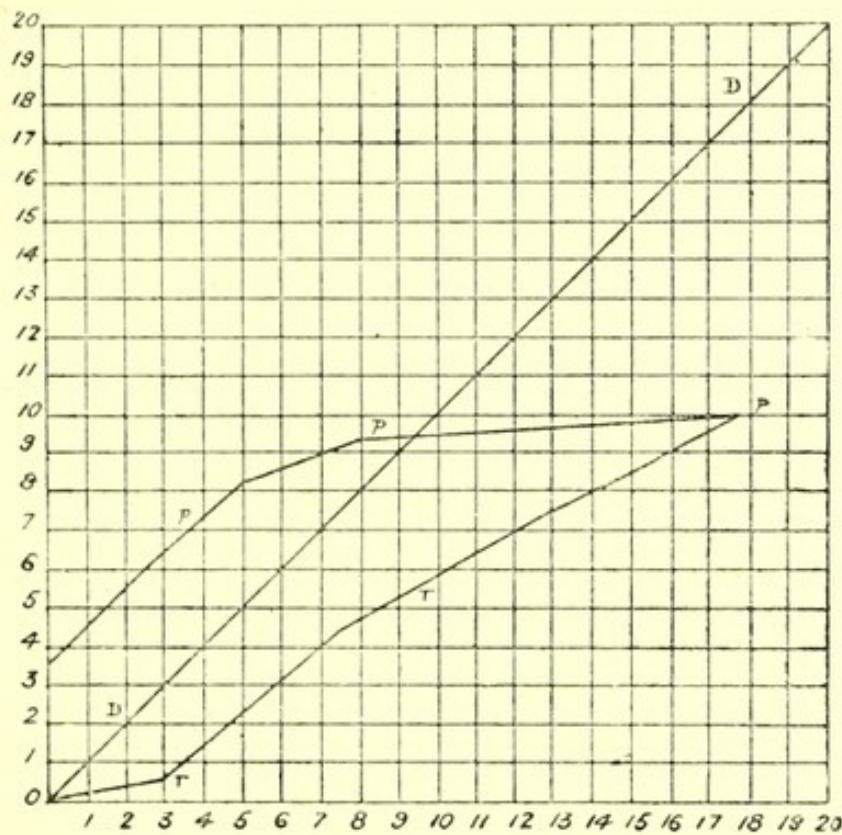


FIG. 2.

(Donders' diagram altered to the metrical system by Nagel.)

the relative amount of accommodation that can be used with different degrees of convergence in an emmetrope aged fifteen.

The horizontal figures record the degrees of convergence in metre angles, and the vertical figures record the degrees of accommodation in dioptries.

The diagonal $D D$ represents the convergence, starting from zero—*i.e.* “infinity”—and stopping at 5 cms. (20 metre angles). The vertical divisions between the upper curved line $p p$, and the diagonal represent the amount of maximum, or positive part, of accommodation ascertained by the strongest concave glass that can be borne without prejudice to binocular and distinct vision, for any given point of convergence; and those between the diagonal and the lower curve $r r$ represent the amount of minimum or negative part of accommodation ascertained by the strongest convex glass. Thus, take convergence for 6 m.a. Above we have 2.5 dioptries of positive accommodation, and below 3 of negative accommodation—that is, the relative amplitude of accommodation for 6 m.a. of convergence is 5.5 in this individual. It will be seen that when the convergence has reached 10 m.a. the whole of the range of accommodation is negative.

Accommodation remaining fixed, we can estimate the amount of relative convergence by means of prisms; the strongest prism, base out, that can be borne with fusion represents the positive, and base in, the negative part of the amplitude of convergence; and, as Landolt pointed out, we find that Fig. 2 can be made use of to represent this. The diagonal $D D$ represents the accommodation starting with eyes adapted for infinite distance; the positive portion of the relative range of convergence is on the right of the diagonal, and is represented by the horizontal divisions between $D D$ and $r r$,

and the negative portion is on the left. Thus for accommodation at 25 cms.—*i.e.*, 4 dioptries—we see that we have 3 m.a. on the right, and 3.5 m.a. on the left—that is, while maintaining the same amount of accommodation, an adducting prism producing a deviation of 3 m.a., and an abducting prism requiring a diminution of 3.5 m.a., can be overcome by the eyes. Thus, for 4 dioptries of accommodative power in this individual, an amplitude of convergence of 6.5 m.a. exists.

It is fortunate for the ametropes that this dissociation between accommodation and convergence is possible. A hypermetrope of 3 D, who fixes an object binocularly 33 cms. off, must use an additional 3 of accommodation—that is, he must use 6 altogether—but he will only require to converge to 3 m.a. If the association between accommodation and convergence were absolute, he would either have to converge to 6 m.a. and consequently squint, and thus lose binocular vision, or he could keep binocular vision on the condition that he did not accommodate for this near-point—in other words, he has the choice between distinct vision and binocular vision; he cannot have both. Many hypermetropes dissociate these two efforts, and can by practice and “education” accommodate in excess of their convergence. The difference in the power to dissociate these two efforts is one of the explanations of the well-known fact, that of two individuals having the same refractive defect, one will squint and the other not.

The same necessity for dissociation between convergence and accommodation occurs in myopia. A myope of 3 D can see an object 33 cms. off without any accommodation, but must converge to the extent of 3 m.a. Thus he uses his *convergence* in excess of his accommodation.

Donders states that accommodation can only be maintained for a distance when, in reference to the negative, the positive part of the relative range of accommodation is tolerably great, and that the relative range of accommodation in ametropic eyes is quite different from that of emmetropic eyes, but that it tends to approach the latter when the correction of the error has been worn for some time (page 126).

It is hardly necessary to add that no improvement has ever been suggested on the foregoing simple and exhaustive explanations of the range of accommodation.

ERRORS OF ACCOMMODATION

SPASM OF ACCOMMODATION.

SPASM, or Cramp of the Ciliary Muscle. This is the opposite of cycloplegia, and occurs in two forms: (1) A temporary spasm, soon passing off with rest; (2) a permanent spasm, referred to on page 62 as *spasm of accommodation*, and generally associated with hypermetropia in young people, and producing an apparent myopia. Both forms are the result of strain of the ciliary muscle, and are, with rare exceptions, cured by the use of a cycloplegic and the correction of the error of refraction.

Donders says that in hypermetropia we find often "a permanent tension, which wholly or partially conceals the abnormal condition; it can exist only in virtue of the accommodation, and must therefore be destroyed by the paralyzing influence of the atropia" (page 622).

Spasm of accommodation often exists in young myopes, causing them to ask for a much stronger concave glass than they require; hence the extreme importance of examining all such cases under the influence of a cycloplegic.

PARALYSIS OF ACCOMMODATION (NON-DIPHTHERITIC).

Donders says of this defect : " Paralysis of accommodation as disease is by no means an unusual occurrence. Emmetropic and ametropic eyes are alike liable to it. It occurs, too, at every age, but in old persons, who have already lost their accommodation by senile changes, it is of little importance " (page 591).

It is generally accompanied by dilatation and immobility of the pupil, which is the only objective symptom. Myopes, of course, suffer the least disturbance, and hypermetropes the most.

It is often not complete, only a paresis being present (page 593), and then the myope probably suffers no inconvenience. Rheumatism and syphilis are the most common causes.

I have seen several cases which were due entirely to *eyestrain*, and the following is an extremely interesting and characteristic one :

Mr. S. H. Aged twenty. An Oxford man. Has been reading lately very hard, and ten days ago found he could hardly read at all, and since then can only read for a very short time. He has put nothing into his eyes, and has not been taking belladonna or any drug. He has been for a week under the care of a medical man, who could find no other trouble or disease. Vision $\frac{6}{6}$ in both eyes. Accommodative power = 4 D. Pupils slightly dilated.

He was put well under homatropine, when the pupils dilated more fully, and the following was the refraction :

$$\text{R.V.} = \frac{6}{9} \bar{c} \begin{array}{c} +.25 \text{ sph.} \\ -.50 \text{ cyl.} \end{array} \begin{array}{c} \diagup \\ \vdots \end{array} \begin{array}{c} 5 \\ \vdots \end{array} = \frac{6}{5} \quad \text{L.V.} = \frac{6}{9} \bar{c} \begin{array}{c} +.25 \text{ sph.} \\ -.50 \text{ cyl.} \end{array} \begin{array}{c} \vdots \\ \diagdown \end{array} \begin{array}{c} 10 \\ \vdots \end{array} = \frac{6}{5}.$$

He was given the cylinders to wear always. He returned in a fortnight quite well, and his accommodative power was then 10 D !

PRESBYOPIA.

In all the splendid work that Donders did to restore order in the chaotic confusion in which the subject of errors of accommodation and refraction existed up to his time, perhaps his best work was in connection with *presbyopia*.

In 1860, in his work "*Ametropie en hare gevolgen*," he first drew attention to the necessity of drawing an accurate distinction between anomalies of *refraction* and those of *accommodation*, and again in his classical work in 1864 he specially refers to this. He thus shows the mistake that had always been made in contrasting presbyopia with myopia. "So long as presbyopia was opposed to myopia, so long must myopia exclude presbyopia, and in old myopic individuals, to whatever degree the senile changes may have attained, the term 'presbyopia' could not be applied" (page 215).

By pointing out that presbyopia was a senile change accompanied by diminished range of accommodation, and that every eye was affected by it whatever its refraction, Donders "cleared the air" at once. He also pointed out that presbyopia was due to an increased firmness of the lens, and that although, as a disturbance, it did not make itself felt in the normal eye before the age of forty, it began before puberty.

He showed how the near-point (P) gradually

receded from the eye from the age of *ten* (or before) by means of very clear and carefully prepared diagrams, which diagrams have been quoted and reproduced in every textbook on the subject in every civilized language for the last fifty years.

He is quite willing to concede that the use of the term "presbyopia" should be a little more limited than the above definition would imply, and may be thus described: "Presbyopia exists when, in consequence of the increase in years, with diminution of the range of accommodation, the nearest point has been removed too far from the eye" (page 83).

Donders thought it necessary to fix the presbyopic point, and in doing so he thought it also necessary to be arbitrary, and as he had originally suggested that when P had receded to *22 cms.*, that should be considered as the presbyopic point, he proposed to adhere to that; but he makes a very important qualification which many writers, quoting him, omit to mention. He says that fixing this point at *22 cms.* *does not necessarily* involve the prescription of glasses (page 212).

In spite of the clearness and accuracy of the foregoing facts enumerated by Donders which have stood the test of time, there are still writers who want to do away with the term "presbyopia." One prominent ophthalmologist quite recently wrote: "Presbyopia is an infirmity of age which spares some old persons until the age of eighty, but may affect young persons of twenty-five years or even

less." *Presbyopia spares no one*. As Donders truly states, it is no more an anomaly than are grey hairs or wrinkling of the skin (page 210), and he concludes his chapter with these words: "The more I investigate the subject, the more fully I am convinced that at a given time of life the range of accommodation is an almost law-determined quantity" (page 214), words which those who have followed in his steps and investigated the subject with the same care and accuracy can absolutely and emphatically confirm.

THE INFLUENCE OF AGE UPON THE ACCOMMODATION.

Fig. 3 is a copy of Fig. 107 from Donders' treatise (page 209); it shows the near-point of 140

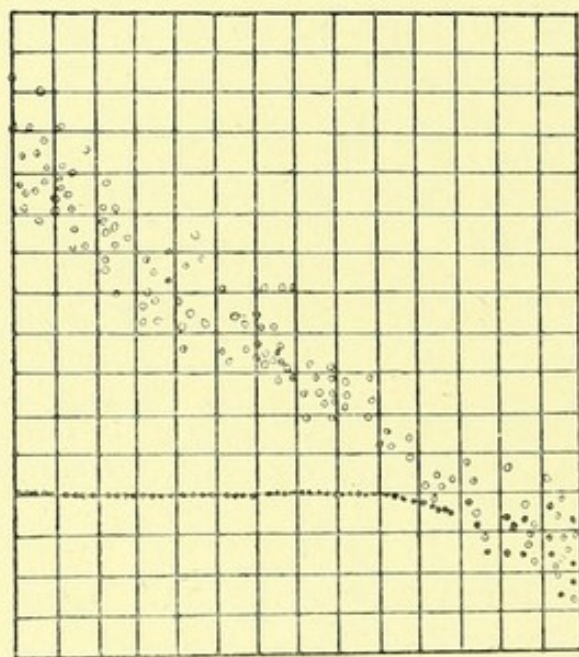


FIG. 3.

individuals, most of them so-called "emmetropes," with a few hypermetropes and myopes.

Taking the mean-point of these and arranging the diagram on the metrical system, we have Fig. 1 (page 7).

By referring to this diagram, we see that the average emmetrope at

the age of ten has 14 dioptries of accommodative power; at thirty only 7 (he has lost half); and 4.5

at forty, which means that his near-point has receded to 22 cms. ($\frac{100}{4.5} = 22$ cms.), the *Presbyopic Point* referred to above.

Most writers on refraction, taking the above diagram as the standard, give diagrams showing the range of accommodation of uncorrected hypermetropia and myopia. For instance, Fig. 14 (page 54) is the diagram of an uncorrected hypermetrope of 4 D, and Fig. 19 (page 68) is one of an uncorrected myope of 3 D. By comparing these diagrams with the emmetrope's diagram (Fig. 1), it will be seen that age for age all possess the same average accommodative power.

It is interesting to compare these results with my investigations when P was determined *after* the error had been corrected (see page 21).

Donders gives a diagram of an uncorrected hypermetrope of 6.5 D (on page 243), and one of an uncorrected case of stationary myopia of 1.75 (see Myopia, page 67), both of them showing the range of accommodation at different ages, and practically agreeing with the foregoing diagrams.

Donders did not use a cycloplegic in a large number of the cases from which he made his diagram, and thus many of the "emmetropes" were probably concealing some latent hypermetropia which he allows may have led to errors; and so to avoid these errors in my investigations every patient under forty-five was placed under a cycloplegic, generally *atropine* up to thirty, and *homatropine* over thirty.

My diagrams show a record of the near-point P of over 3,000 cases, and in every instance the smallest error of refraction was ascertained and corrected, and only normal cases were selected; the smallest trace of opacity of the cornea or lens, any abnormality of the fundus, even though it did not interfere with visual acuity, or the smallest amount of conjunctivitis, etc., was a cause for exclusion.

Tweedy's optometer was the instrument used for determining P, and the type was Snellen's D = 0.5.

The distance was measured from the anterior focus of the eye—viz., 13 mm. in front of the cornea.

In all cases where P had receded beyond 33 cms., convex glasses were added to the correction, and of course deducted afterwards.* During the examination the patient wore the correction of his defect—*i.e.*, the normal correction, not the cycloplegic correction. If there was any latent hypermetropia, this was allowed for in calculating the final result. For instance, a lad of sixteen under atropine was shown to have 3 D of H. The post-cycloplegic correction was +1.25. Assuming the eye to be never so relaxed as when under atropine, and allowing +1 D for the atropine, it left $2 - 1.25 = .75$ D to be added to the final result. With +1.25 in front of the eyes, the near-point P was at 10 cms., showing 10 D of accommodative power, and adding +.75 to this, made his total accommodative power = 10.75.

* In children where the accommodation was very powerful, -3 glasses were added to the correction, and, of course, 3 was added afterwards.

Fig. 4 shows the near-point P of 1,200 individuals of different varieties of refraction, in whom the error had been corrected previous to the determination of P.

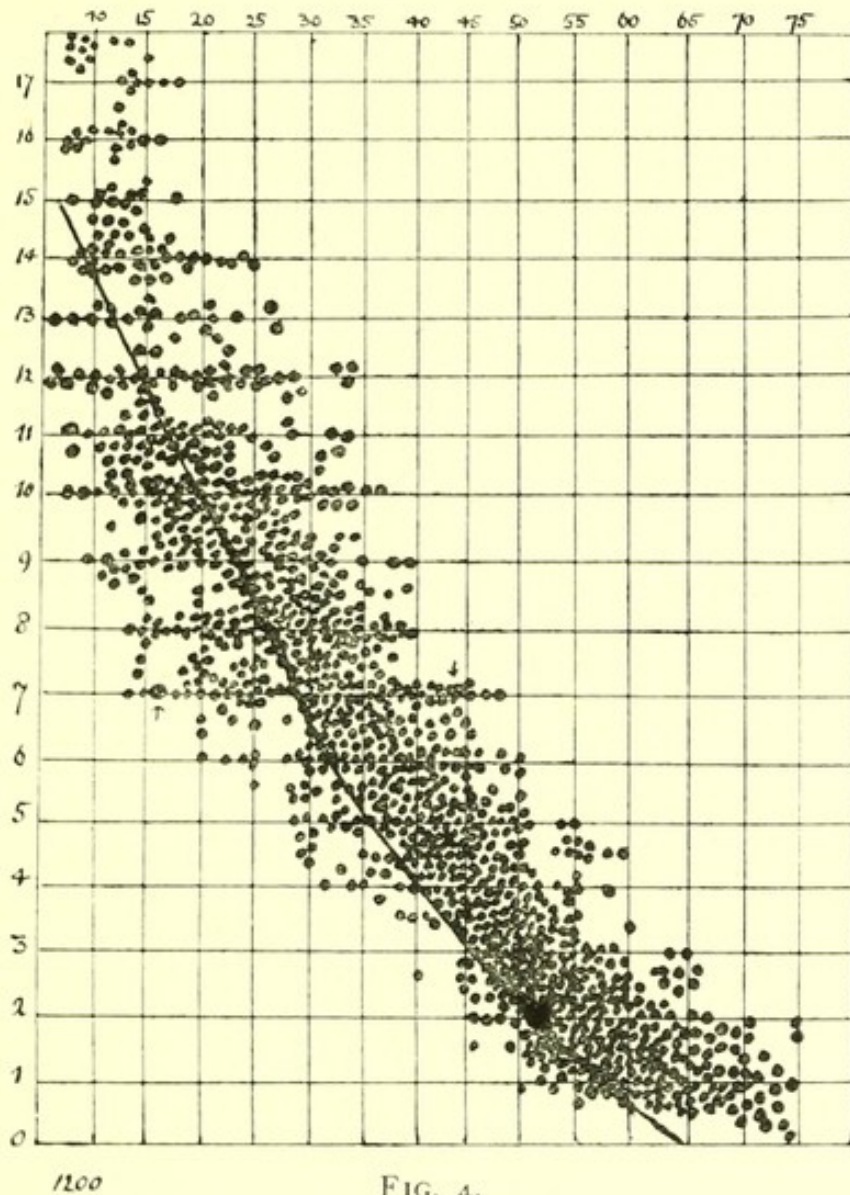


FIG. 4.

Out of the whole number, only *four* were emmetropic; hypermetropia, myopia, hypermetropic astigmatism, myopic astigmatism, and mixed astigmatism were represented by the rest.

Fig. 5 is another diagram, showing the near-point of 500 individuals, possessing different varieties of refraction corrected previous to the determining of P.

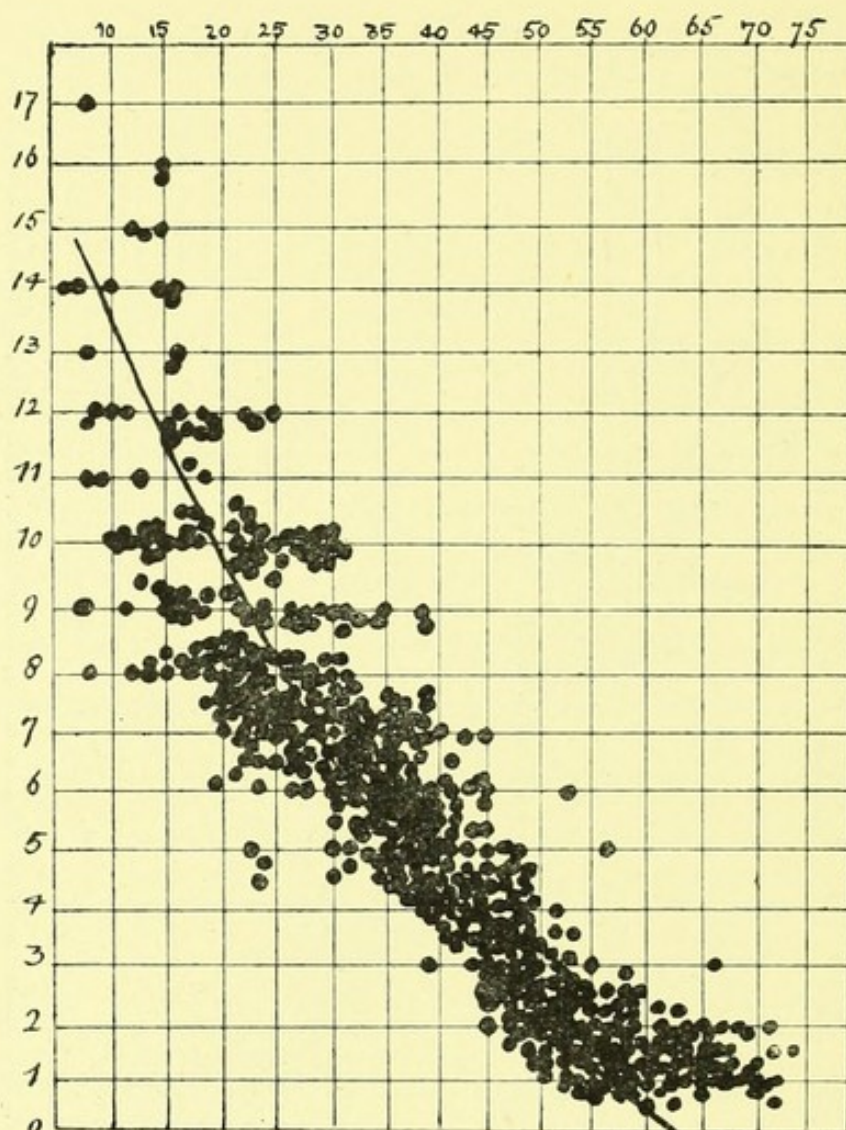


FIG. 5.

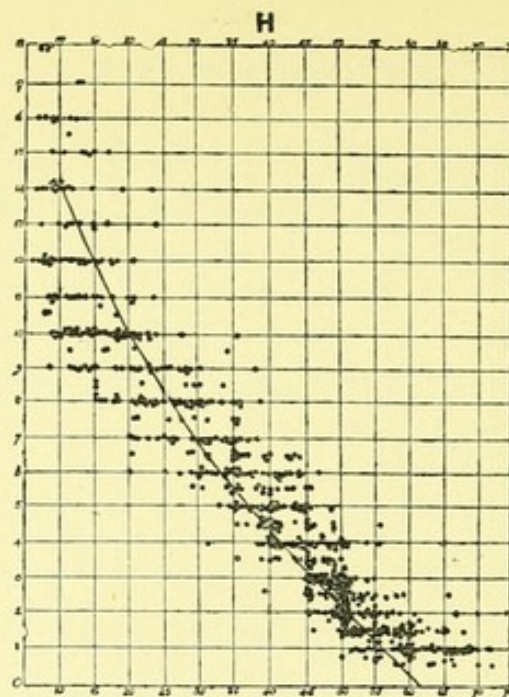
From diagram (Fig. 4) we obtain the following table :

Age.	Minimum.	Mean.	Maximum.
7-10	9	14	18
10-15	7	12	18
20	6	10	14
25	5.5	9	13.5
30	4.5	7.5	12
35	4	6.5	10
40	2.5	5.5	8.5
45	2	4	7
50	1	3	6
55	0.75	2	5
60	0.50	1.75	4
65	0.50	1.5	3
70	0.00	1	2

In order to discover whether there was any difference in the *curves of P* in hypermetropes and myopes, the diagrams on p. 24 were compiled :

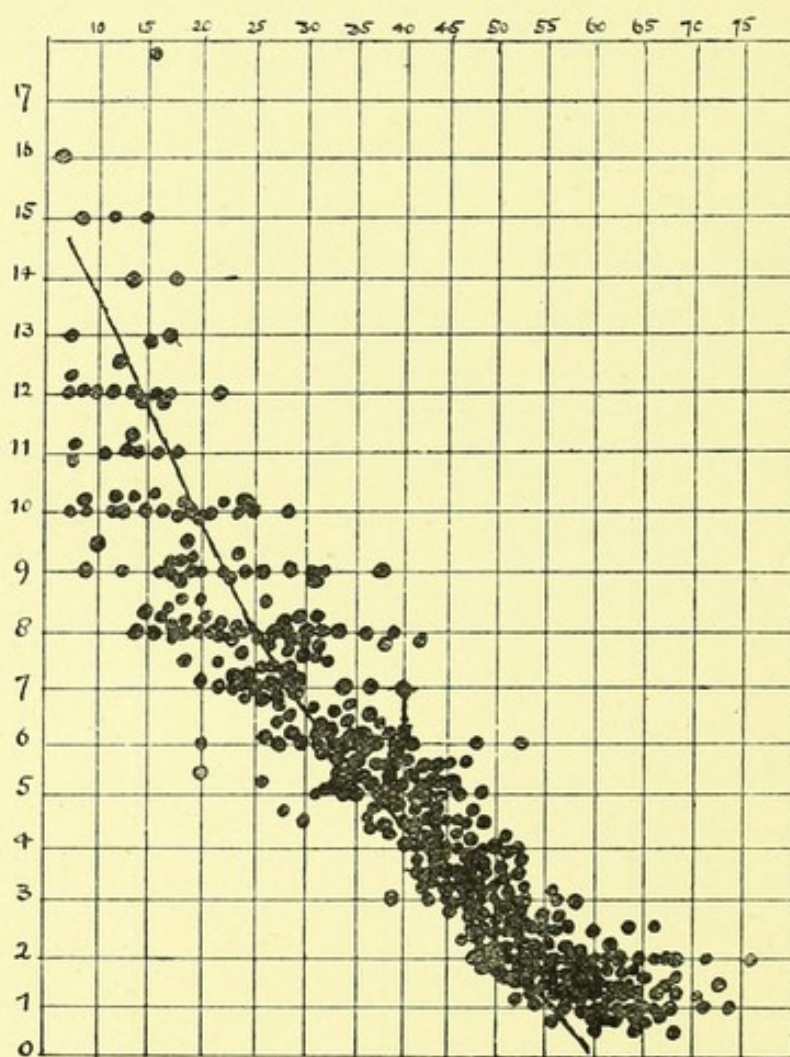
Fig. 6 shows the near-point of 700 corrected hypermetropes, and Fig. 7 another series of 400 cases, and from these diagrams we obtain the following table :

Age.	Minimum.	Mean.	Maximum.
7-10	9	14	18
10-15	9	12	17
20	6	10	14
25	6	9	13
30	5.5	7.5	10
35	4	6.5	9
40	2.5	5	7
45	2	4	6.5
50	.75	3	4.5
55	.75	2	4.5
60	.50	1.5	2.5
65	.50	1	2.5
70	.50	1	2



70

FIG. 6.



400

FIG. 7.

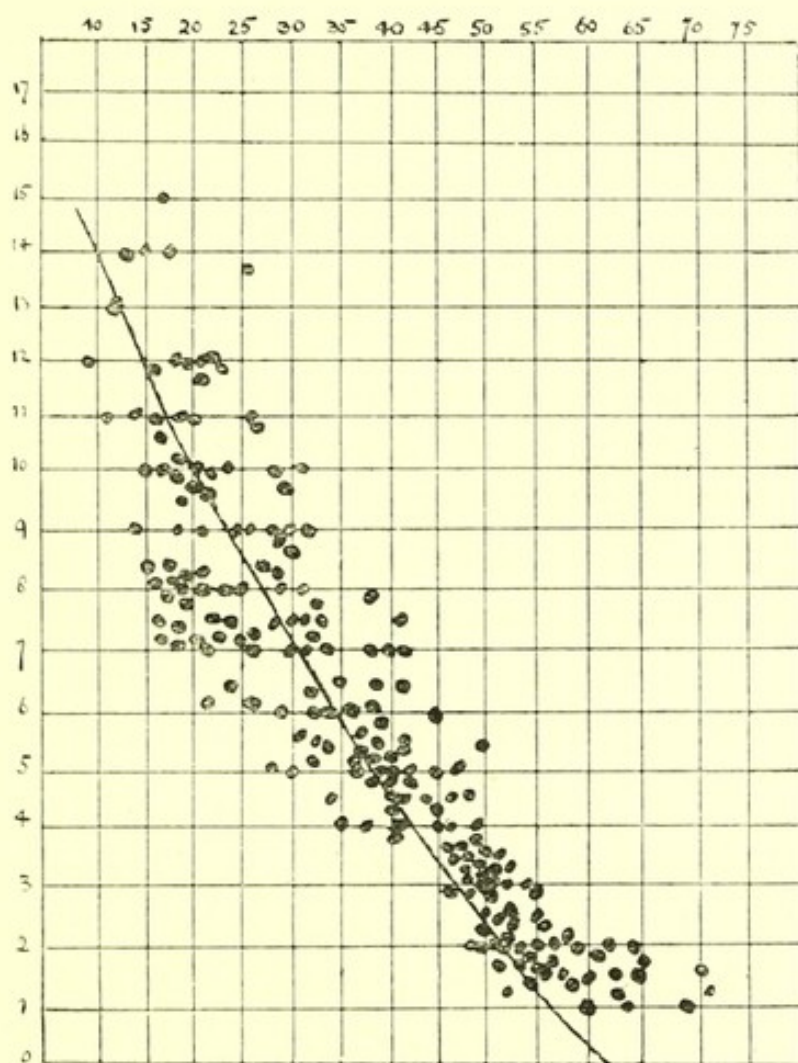
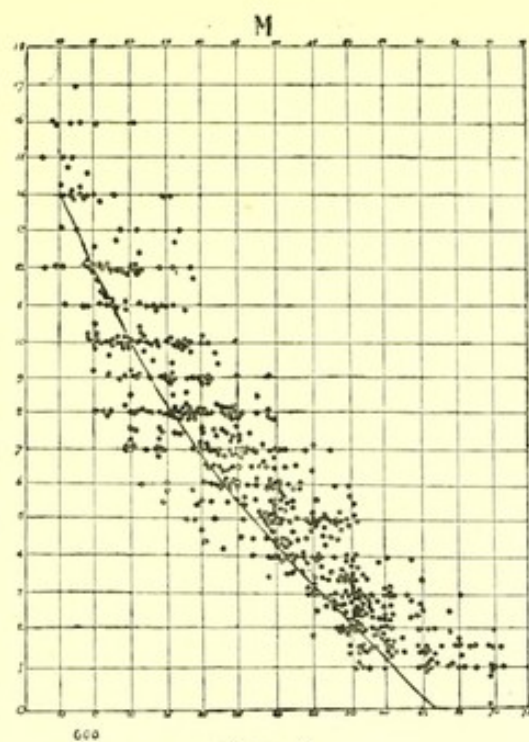


Fig. 8 shows the near-point of 600 *corrected* myopes, and Fig. 9 that of another series of 200 cases.

From these diagrams we obtain the following table :

Age.	Minimum.	Mean.	Maximum.
5-15	8	12	17
20	7	10	16
25	5.5	9	14
30	4.5	8	12
35	4.25	7	10
40	3.5	6	8
45	1.75	4.5	7
50	1.5	3.5	6
55	1	2.5	4
60	1	1.75	3.5
65	1	1.5	3
70	.25	1	2

More or less astigmatism was present in the large majority of all the cases whose near-point I have recorded, and was, of course, corrected before the examination.

Comparing all my diagrams, they will be seen to be very similar. The chief feature to notice is that in *myopia* the presbyopic point (22 cms.) is not reached on the average until forty-five, whereas in *hypermetropia* it is reached at forty-four. Now, this explains a remark made by Donders—viz., “Generally speaking, I have found that myopics require convex glasses at a still later period than the degree of M. should have led one to expect.”

The line marked in all the diagrams is Donders' mean near-point line.

Until the age of twenty-five, Donders' mean line would represent *my* mean near-point line ; but from that age onwards the majority of my cases are well above the line, so much so that Donders' *mean* near-point line almost becomes my *minimum* near-point line. This is seen not only in the mixed cases, but also in the others.

These investigations show that after the age of about thirty, individuals have more accommodative power than Donders indicated, and that whereas he found the average presbyopic point (22 cms.) was reached at forty, my tables put the age later, from forty-three to forty-five.

There is little doubt that errors occurred in Donders' examinations through many so-called emmetropes having latent hypermetropia, as he himself suggested.

Duane of America has been working at this subject at the same time that I have, and his results agree very much with mine (*Ophthalmoscope*, September, 1912).

Now, it may be said that the foregoing facts are quite interesting, but what good is it for us to trouble to find a patient's near-point, and especially to find it accurately, except in so far as it helps us to ascertain the assistance he requires for near work ? I think the following remarks will show how important the knowledge is to us and to him.

In looking at all these diagrams it will be noticed what wide differences there are in individuals of the same age as regards their accommodative power. This is even seen in Donders' original diagram, although here, of course, the differences are not so great, as the number examined was smaller.

The question may be asked whether these wide differences are opposed to Donders' statement that "at a given time of life the range of accommodation is an almost law-determined quantity." The answer is supplied by Donders himself, who says that the unfavourable exceptions are connected with definite defects, and *vice versa*.

Now, what is the definite defect of an otherwise normal individual of forty, who, instead of having 5.5 D of accommodative power, which is the average, has only 2.5? Obviously the answer is, *Premature old age!* This is exactly what one finds clinically.

It is common knowledge that the average man of fifty looks fifty, acts as if fifty, and *is fifty*; but many aged fifty look sixty-five, and to all intents and purposes *are sixty-five*, while others look only thirty-eight or forty, and act and live as if only that age.

In quite early youth the crystalline lens is practically a small bag of semifluid jelly, and accommodation takes place by its being squeezed by the action of the ciliary muscle in such a manner that its antero-posterior diameter is enlarged. So great is this "squeezability" (if I may use the term) in the

very young, that an accommodative power of 20 D can often be recorded. As age advances, a hardening process or sclerosis goes on in the lens as in all the other tissues of the body, and so its "squeezability" becomes less and less until a point is reached when the near-point of accommodation, which represents the fullest accommodative power, has so far receded that the normal eye requires assistance in the shape of a convex lens in order to see near objects distinctly.

This hardening of the lens may be delayed by the absence of and accelerated by the presence of certain poisons in the system, and *intestinal toxæmia* takes a very high place in the list.

On looking at my diagrams, it will be observed that although there is a distinct grouping of the cases by which we can obtain a curved line indicating the mean accommodative power at different ages, still, if we take any particular age, we notice a very wide range for that particular age. Take the line of age forty (Fig. 4), one individual has only an accommodative power 2.5 D, while another has 8.5 D! What is the difference between these two men?

In the man of forty with only 2.5 D accommodative power, the lens has hardened prematurely and become equal to the lens of a man of fifty-five—that is, he is suffering from premature senility—and in the great majority of cases in physical appearance, habits, and powers, he is fifty-five!

There are many causes which help towards this

premature senility, but the factor common to a very large majority of them is *intestinal stasis*.

On the other hand, those whose accommodative power is higher than normal look much younger than their years, and in every way are younger; and on going into their history, it will be found invariably that they have taken the greatest care to avoid the least symptom of intestinal stasis.

These facts in connection with the premature senility of the lens are another proof of the correctness of the old saying that "a man is as old as his arteries," the premature sclerosing of the vessels taking place at the same time as the process in the lens. Amongst the many examples I have bearing out the foregoing statements, that shown in Fig. 4 is perhaps the most remarkable. It shows the accommodative power (after correcting all errors) of two females—one fifteen and a half, and the other forty-four. They both have the same accommodative power—viz., 7 D. The girl was too young to show any outward sign of ageing; but she had a most unhealthy appearance, and confessed to habitual constipation, and asserted that the bowels were open barely twice a week. On the other hand, the woman of forty-four looked ten years younger, and had always paid the most scrupulous attention to the bowels, and for some time past had taken a daily small dose of petroleum oil.

I have notes of another patient aged forty-nine, with only 2 D of accommodative power, the amount that many men of sixty have. He had white hair,

no teeth, a flabby, dirty tongue and habitual constipation, which he did not trouble to correct.

To show how short a time is required to induce premature senility by intestinal stasis, I cite a man, who two years ago, aged then forty-eight, had 5 D accommodative power. In the interval he has suffered very badly with intestinal stasis, which he has very often not troubled to overcome. His accommodative power is now only just over 2 D. He has lost more than half his accommodative power in two years. There was no other cause ascertainable, except the intestinal one, to account for his ageing ten years in two years.

We must not forget that a very common cause of loss of accommodation is weakness of the ciliary muscle. This has not necessarily any connection with age, and is generally temporary. It is due to physical fatigue, diminished vital energy of the individual due to general disease, etc., and is very often the result of eyestrain. Correction of the error of refraction, rest from near work, and improvement in the general health restores the accommodative power, in most cases, to the normal.

A small error of refraction uncorrected not only tends to lower the power of the ciliary muscle by the constant drain on its energy, but also tends to hasten the sclerosing processes in the lens (see page 99).

All these facts go to show what increased power this knowledge gives us of benefiting our presbyopic patients. When we find a patient with marked

decrease in accommodative power, we can sound the note of warning and send him to a physician for a thorough overhauling and treatment. This, coupled with the correction of all refractive errors, will stay the progress of the senile processes.

TREATMENT OF PRESBYOPIA.

Up to Donders' time it was the general opinion that the wearing of convex glasses for presbyopia should be postponed as long as possible, but he says: "So soon as, by diminution of accommodation, in ordinary work the required accuracy of vision begins to fail, there is need of convex glasses" (p. 217), and the reason he gives for this is that fatigue of the accommodation is otherwise likely to ensue. Years have not altered the accuracy of this statement. It is as true to-day as when he said it, and, further, we can most emphatically assert that too strong convex glasses can do nothing like the harm that too weak ones may do.

Donders very truly said: "Essential injuries to sight, which are often, with so much exaggeration, predicted, I have never seen arise, even from an undue use of convex glasses" (p. 232).

At what age should an individual begin to use convex glasses? The answer is, There is no definite age!

When we note the wide differences in the accommodative power of different individuals of the same age, we realize that the time for beginning to use convex glasses for near work depends entirely on the individual and his work. A long-armed man who does very little near work may not require glasses until the age of fifty; whereas a seamstress with the same refraction and accommodative power and of the same age may have to take to glasses five years earlier.

Donders says: "It is true that eyes differ too much to make age alone the criterion in the choice of spectacles. But, on the other hand, the regular diminution of the range of accommodation . . . shows that in the case of emmetropic eyes the time of life may in general be taken as a guide" (p. 220).

The recognition of presbyopia is not difficult. When a person complains that he has to hold his book when reading farther away than he has been accustomed to; that this is more especially so by artificial light, that the figures 3, 5, and 8 become confused; and that *n* and *u* are difficult to distinguish; and at the same time asserts that his distant vision has not altered—we may be almost certain we have before us a presbyope.

When a person whose near-point has receded, say to 33 cms., attempts to read or work at that distance for any length of time, symptoms of eye-strain will be sure to supervene. It is a fact that we get from everyday experience, that the full power of a muscle can be exercised only for a very short time without fatigue. A person whose near-point is at 33 cms. is using the whole power of his ciliary muscle in order to focus an object at that distance on his retina, and fatigue of the muscle will very soon ensue. This fatigue causes the muscle to relax; it cannot contract to its full extent; vision is rendered hazy, and becomes distinct again only when the object has been removed farther from the eye. At the same time, the patient will probably complain that after reading for some little time,

headache comes on, and the eyes begin to water. These temporary symptoms of eyestrain will pass into chronic symptoms in time, and the red, irritable-looking, watery eyes of middle-aged people are often due to this cause. This latter is referred to by Donders as *accommodative asthenopia*.

A cycloplegic is rarely necessary (except in isolated cases where our results are unsatisfactory, or we wish to examine the lens for cataract), because ciliary spasm is very unlikely to be present, and there is no latent hypermetropia, it having all become manifest. We first ascertain the distant correction, and then the near-point of distinct vision, and the most comfortable distance for reading or working. Suppose distance vision is normal and the punctum proximum is 28 cms., his amplitude of accommodation is $\frac{100}{28} = 3.5$ D; to avoid fatigue he must not use the whole of this, but must keep about one-third in reserve. Let him have 1.5 D in reserve; this leaves him 2 D available accommodative power, and as he requires 3 D we supply the deficit by giving him + 1 D glasses.

The treatment, simple as it appears, is not always successful because of the great variation of the accommodative power in different individuals already referred to. Of course, when the accommodative power is low, a greater reserve has to be left. We generally find that an emmetrope of about forty-eight requires reading glasses of + 1, at fifty + 1.5, at fifty-three + 2, at fifty-six + 2.5, and at sixty + 3.

In the above example we supposed distant vision to be normal; but even when a small error was present, it was the custom until quite recently for presbyopes to be provided with reading or working glasses only. Now, however, through our increased knowledge of the bad effect of eyestrain, we recognize the importance of correcting all errors of refraction, even the smallest, especially at the period of life when presbyopia appears, if any symptoms of eyestrain are present, in which case glasses are required for distance as well as for near vision.

To avoid the trouble of changing the glasses, and especially to avoid the eyestrain which follows not changing them when a change is requisite, *bifocal* glasses (the top part for distance and the lower part for reading) are required.

Such glasses were known to Donders, and they were given when high hypermetropia or myopia was present, to presbyopic painters, etc., and were known as Pantascopic or Franklin glasses, or split bifocals. The line across separating the two glasses was extremely objectionable, and there was not much improvement upon this when the reading addition was balsamed on as a small oval disc (Fig. 10).

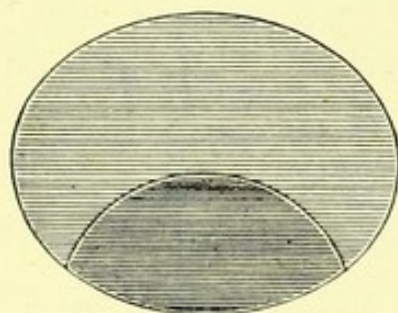


FIG. 10.

It was not until quite recently, when the improvement in the optician's art provided us with "in-

visible"* bifocals, that the more extended use of this form of treating presbyopia became possible, and it is to these invisible bifocals that we owe this reform.

The smallest error of refraction can be corrected in the upper part, and the lower part is the correction for near vision; and if the glasses are made to fit properly and are accurately centred, the wearer is practically unconscious of having *two* glasses! Not only this, but the constant necessity of putting up a glass whenever he requires to see any near object is dispensed with. The glass is there ready for him!

Of course, if the presbyope is practically emmetropic, lives an outdoor life, and has no symptoms pointing to eyestrain or waste of nerve power, the old treatment of simply giving near-work glasses can be adhered to; but if the smallest suspicion of eyestrain is present, these *invisible bifocals* should be prescribed.

There are two forms of these bifocals: (1) The Kryptok, where a concavity is formed in the lower part of the distance glass, and the reading glass of a higher refractive index is fused into it; (2) the Luxe, where the whole glass is made from a solid piece of crown glass with the two lenses ground invisibly on its surface. The chief advantage of

* A form of "invisible" bifocal glasses was known to Donders as "*verres a double foyer*," but they had the great disadvantage of having the curved division between the glasses turned the wrong way (see p. 138).

the Luxe bifocal is that the centering of both portions is more under control, and there is no chromatic aberration so often present in the fused form.

Up to a few years ago Donders' treatment of presbyopia was generally followed, and the "revolution" in the treatment coincides with the appearance of these invisible bifocal glasses.

As the bifocals have to be used for writing, working, eating, etc., it is very important that the lower section should not be too strong. It is rarely necessary at any time of life to prescribe an addition of more than 3 D. Should the patient want a stronger glass for reading or sewing by artificial light, this should be ordered as a separate near-work glass.

The optician must take great care in fitting and centering the bifocals. The division between the glasses should never be higher than the margin of the lower lid, and full allowance should always be made for convergence. Thus, if the patient converge 3 mms., each reading portion should be decentred in 1.5 mms.

Donders very properly draws attention to the importance of ascertaining the particular work of the presbyope before selecting his glass, small fine work, such as engraving, requiring much stronger glasses than work at a distance, such as reading music.

Too strong a glass entails too near approach of the work to the eyes, and often bending the head,

or stooping; and too weak a glass causes difficulty in reading or sewing, and very often leads to eye-strain. In other words, it is desirable to keep a certain amount of accommodative power in reserve, but not too much.

PRESBYOPIA AND ANISOMETROPIA.

When presbyopia is present with anisometropia we should, whenever possible, prescribe bifocal glasses. The lower reading section must be found by the same kind of trial as we made for the distant correction. It does not follow that the same addition is given to each eye. For instance, one eye has a hypermetropia of 2, and the other of 4, and the patient is fifty-two; we should try +4 and +6 for reading; or, if this is not comfortable, +4.5 and +5.5, or +5 in both eyes.

When one eye is permanently excluded, and the remaining eye is ametropic and presbyopic, as in aphakia after cataract extraction, reversible spectacles are useful; the distant correction is on one side, and the reading on the other. When the patient is walking, the distant correction is in front of the "good" eye, and the reading glass in front of the useless eye, and when he wishes to read he reverses the spectacles, and so brings the reading glass in front of the good eye.

When the difference between the two eyes is marked, and simultaneous binocular vision exists, although the distance correction is accepted, discomfort may come with near work. This is due to the production of an artificial *hyperphoria*. Let us suppose the right eye to have a myopia of 1.5, and the left a hypermetropia of the same amount. When reading and looking, say, 6 mms. below the

optical centres, the patient is looking through the equivalent of a prism 1° base down in front of the right eye, and 1° base up in front of the left. (A lens of 1 D decentered 8.7 mms. produces a prismatic effect of 1° .) The difficulty can be overcome by cementing on the lower portion of each lens the necessary correcting prism. In the above case a prism 1° base up in front of the right eye and 1° base down in front of the left will correct the hyperphoria.

When a presbyopic correction is also required, the prismatic effect may be obtained by decentering, or by making the prism convex to the amount required.

STANDARD FOR VISUAL ACUITY

STANDARD FOR VISUAL ACUITY

ON page 32 Donders refers to Snellen's test types, which have been our standard for visual acuity for the last fifty years.

The smallest visual angle in which the standard eye can recognize an object is an angle of one minute, so that two points of light, such as two stars, separated by an angular interval of less than one minute, would appear on the retina as only one point.

Snellen conceived the excellent scientific idea of arranging his type on this plan. Each letter is made up of several parts, each of such a size that it subtends an angle of one minute vertically and horizontally, the whole letter subtending an angle of five minutes vertically and horizontally when read at the standard distance.

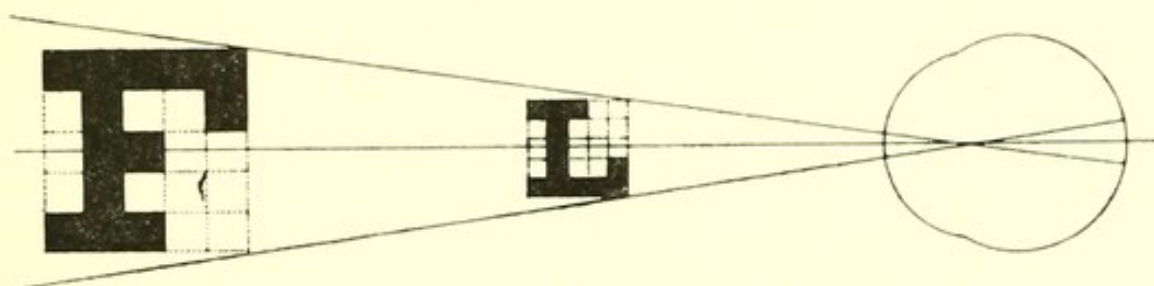


FIG. 11.

Thus, in Fig. 11 the F is made out of twenty-five squares, each subtending an angle of one

minute (the whole letter subtending an angle of five minutes) when read by the normal eye at 12 metres; and the L, which is constructed on the same plan, subtends the same angle when read by the normal eye at 6 metres.

The numbers over the different-sized letters in Snellen's types represent the distance in metres at which the standard eye can read them—in other words, at that distance they subtend an angle of five minutes. For instance, the largest type, $D=60$, can be read by the normal eye at 60 metres, and it subtends the same angle as the type $D=24$ read at 24 metres, and $D=6$ read at 6 metres. The acuteness of vision is represented by a fraction which has for its numerator the distance in metres at which the type is read, and for its denominator the distance at which it ought to be read, and this fraction represents the *value* of the vision.

For instance, if $D=6$ can be read at 6 metres, vision = $\frac{6}{6}$ —*i.e.*, *normal*; if only $D=18$ can be read at 6 metres, vision = $\frac{6}{18}$ —*i.e.*, one-third of normal; if $D=60$ can only be read at 2 metres, vision = $\frac{2}{60}$ —*i.e.*, one-thirtieth of normal.

Snellen added in numerous ways to our knowledge in ophthalmology, and his test types have been an incalculable boon to oculists. He is another great ophthalmologist of whom Holland has a right to be proud!

ERRORS OF REFRACTION

ERRORS OF REFRACTION

HYPERMETROPIA.

DONDERS' first communication in connection with H. was in 1858 (*Nederl. Tijdschrift voor Geneeskunde*). In the next year, 1859, at the Heidelberg Meeting, Helmholtz tried to bring in the term *hyperopia*, but Donders considered that *hypermetropia* was more in accordance with the words *ametropia* and *emmetropia*, and it was finally generally adopted, although of late years there has been a tendency, especially in America, to revert to the shorter word.

In "Ametropia en hare Gevolgen," 1860, Donders further developed his ideas on H., and finally, in the English treatise, produced an exhaustive and comprehensive essay on it in all its bearings.

When we realize that it was he who first showed that hypermetropia was the opposite of myopia, that it was the most constant cause of strabismus convergens, and that "asthenopia," as it was then called, was invariably associated with it, and that almost all his writings in connection with H. remain true to this day, we can fully appreciate the enormous debt we owe him. Let us briefly review what he taught us:

The hypermetropic eye is the undeveloped eye in which, with accommodation at rest, parallel rays come to a focus beyond the retina (Fig. 12, *H*), and only convergent rays focus on the retina ; but

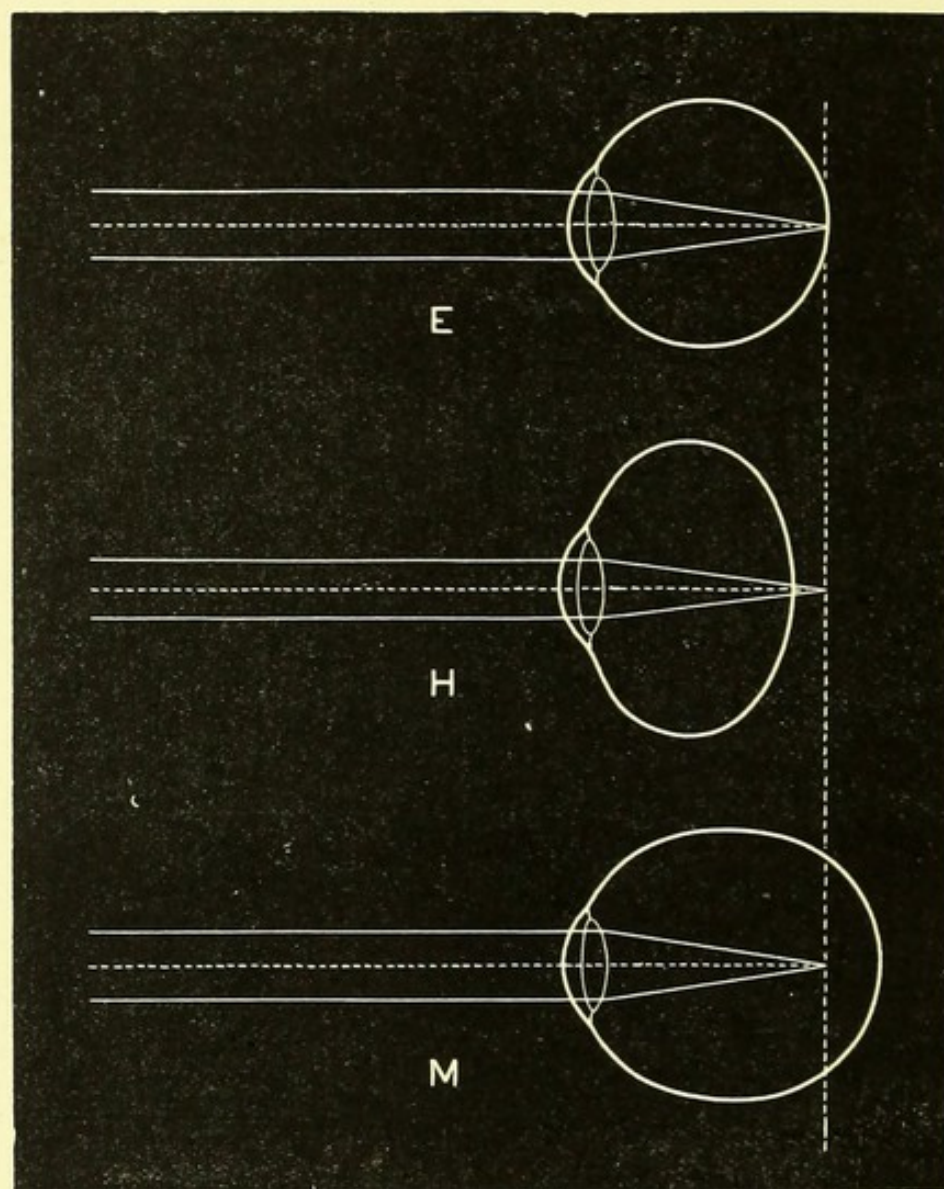


FIG. 12.—SHOWING PARALLEL RAYS FOCUSED ON THE RETINA IN EMMETROPIA (*E*), BEHIND THE RETINA IN HYPERMETROPIA (*H*), AND IN FRONT OF THE RETINA IN MYOPIA (*M*).

as in nature all rays are either parallel or divergent, it follows that the hypermetropic eye at *rest* sees everything indistinctly.

Rays coming from a point on the retina diverge, and, on passing through the dioptric system, emerge from the normal eye as parallel rays. In hypermetropia, although they are not so divergent as they were before refraction, they still diverge if the eye be at rest, and therefore never come to a focus in front of the eye, but, if prolonged backwards, they would meet at a point behind the eye—the *punctum remotum*. This punctum remotum of the hypermetrope is therefore not the actual focus of

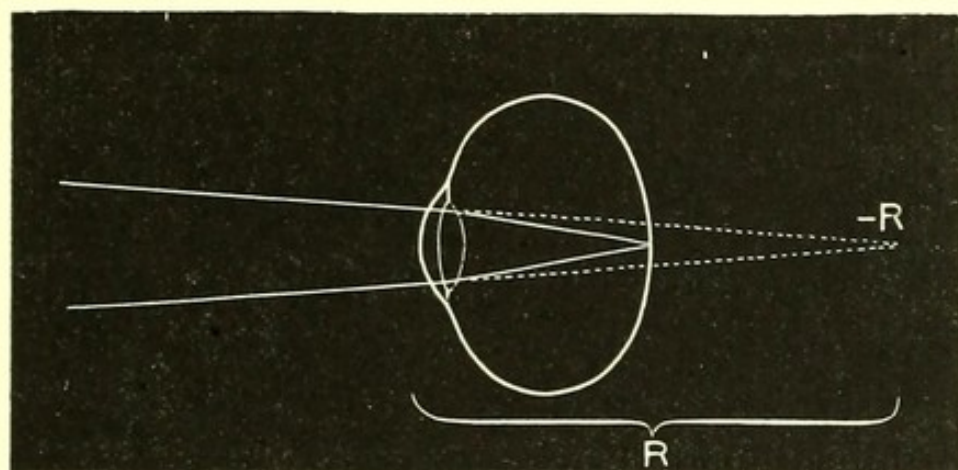


FIG. 13.

the distant rays, but the virtual focus, and is represented by the negative sign, $-R$ (Fig. 13).

Donders says: "The flat anterior surface of the sclerotic, the strong curvature of its meridians in the region of the equator, the shallow position of the iris, the relatively small pupil, the apparent diverging strabismus—all these give a peculiar physiognomy to the eye. But there is still more. In the form of the face, too, the existence of H. is not infrequently expressed. If I am not mis-

taken, the peculiarity which here prevails *depends chiefly on the shallowness of the orbit*. The margins of the sockets are flatter, less curved; the whole face is flattened, with little relief; there is little rounding in the cheeks, because the anterior surface of the face quickly passes into the lateral flatness. Often, too, the nose is but slightly prominent, and *the upper part of its dorsum is so little marked that it can scarcely give support to ordinary spectacles*. The eyelids are flat and broad; *the eyes are far from one another*. The same is true of the orbits—at least, of their outer margins, whose mutual distance is easily measured” (page 252). He goes on to say that we must not expect these anatomical peculiarities in every case, “but that a connection does exist between the refraction of the eye and the form of the face appears most distinctly from the asymmetry of the bones of the latter, including the frontal bone, which, almost without exception, accompanies a great difference of refraction in the two eyes. In general, we find in such cases the eye on the hypermetropic side placed farther from the root of the nose, and, together with the whole side of the face, sloping backward. It is as if the bones of the face on this side are, in general, less developed” (page 252).

The H. structure is hereditary.

Donders divided H. into Acquired H. and Original H.

(a) ACQUIRED H.

All eyes, whatever their refraction, begin to acquire H. when past middle life. The emmetropic eye becomes hypermetropic, the hypermetropic eye more so, and the myopic eye less myopic. If we look at his diagram (Fig. 1), we see r , the far-point line, remaining the same until the age fifty-three, when it gradually curves downward, and Figs. 14 and 19 show the same.

He says (page 206) : "In advanced life the lens appears to become flatter, on which account the radii of curvature of its surfaces and its focal distance are increased," and that this change in the lens is the cause of the acquired H.

(b) ORIGINAL H.

He divided H. into "manifest" and "latent."*

Manifest H. is expressed by the strongest convex glass the patient will accept.

* The division that usually obtains now is as follows :

Total hypermetropia (Ht.)	{	Latent H. (Hl.)	{	Only revealed under a cycloplegic.
		Manifest H. (Hm.)		<i>Absolute</i> (Hma.), which no amount of accommodation can correct, represented by the weakest convex glass. <i>Facultative</i> (Hmf.), when distant objects can be clearly seen with or without convex glasses, represented by the difference between the strongest and weakest convex glass.

Latent H. is the additional H. revealed by the action of a cycloplegic.

Manifest H. Donders divided into *absolute*, *relative*, and *facultative*.

The distinctive character of H. is that the position of focus ϕ'' is behind the retina in rest of

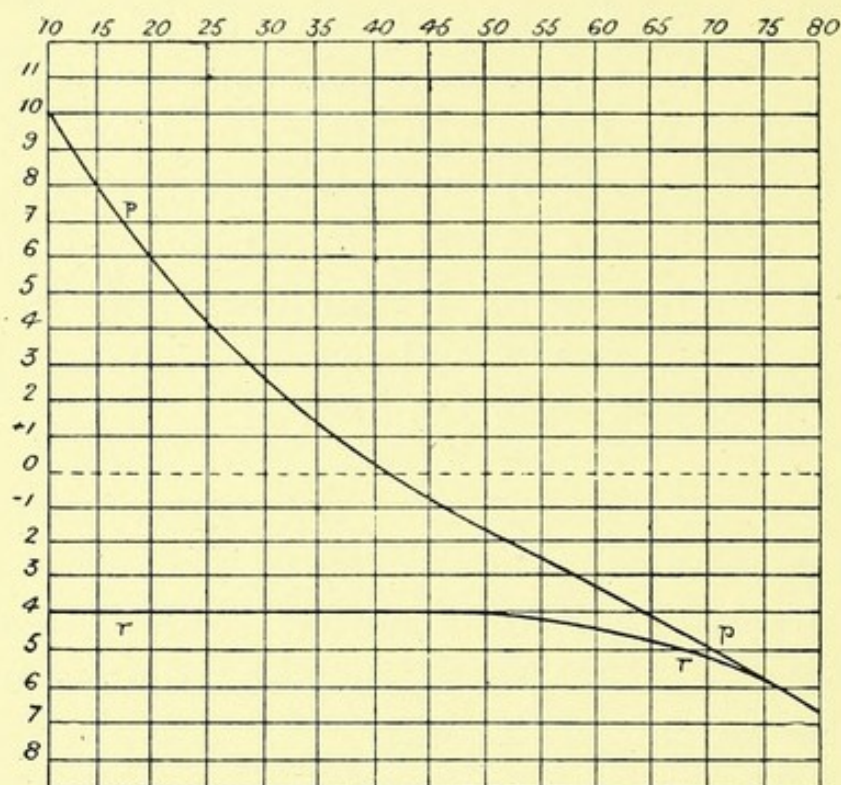


FIG. 14.—SHOWING THE RANGE OF ACCOMMODATION OF AN "UNCORRECTED" HYPERMETROPE OF 4 D AT DIFFERENT AGES.

In the above figure it will be seen that *P* crosses the zero line at age *forty*, which means that at that age the hypermetropia, which has been facultative, becomes *absolute*.

accommodation (page 242). If, moreover, with the most powerful tension, ϕ'' remains behind the retina, Hm. is *absolute*; if ϕ'' can reach the retina only with convergence of the visual lines, Hm. is *relative*; it is, on the contrary, *facultative* when

also with parallel visual lines ϕ'' can be brought on to the retina.

With absolute Hm., vision can never be acute; with relative Hm., only monocular vision can be so. With facultative Hm., vision may be acute, but is often accompanied by fatigue (asthenopia). With increase of years, facultative Hm. passes into relative, and finally into absolute, H.

All the foregoing facts, and Donders' subdivision of H., still obtain to-day, and no change has been made.

See under Presbyopia, Figs. 6 and 7 (page 24), which gives the near-point of 1,100 hypermetropes examined by me while wearing their correction.

STRABISMUS.

Donders pointed out that an *apparent* divergent strabismus is comparatively frequent in H. (page 248). The arrested development, especially of the external portion of the H. eye, is the cause of the macula being placed more external than in emmetropia or myopia; the result is a positive, and often large, angle gamma; consequently the corneal axes converge relatively to the lines of vision, and appear to diverge when the latter are parallel.

What Donders called "angle alpha" is now generally styled "angle gamma."

THE ANGLE GAMMA.

When the optic axis passes through the fovea, it coincides with the line of vision, or line of sight,

and also with the line of fixation; but the exception to this is the rule, and an angle is formed by the line of fixation MO , with the axis AA' . This angle is called the angle gamma (OMA , Fig. 15). (The angle OKA (Fig. 15) made by the *line of vision*, and the optic axis, may be considered identical with the angle OMA , and is sometimes called the angle gamma.)

The angle gamma is *positive*, as in Fig. 15, when the fovea is on the outer side of the optic axis, and it is generally positive in emmetropia and hypermetropia; and in

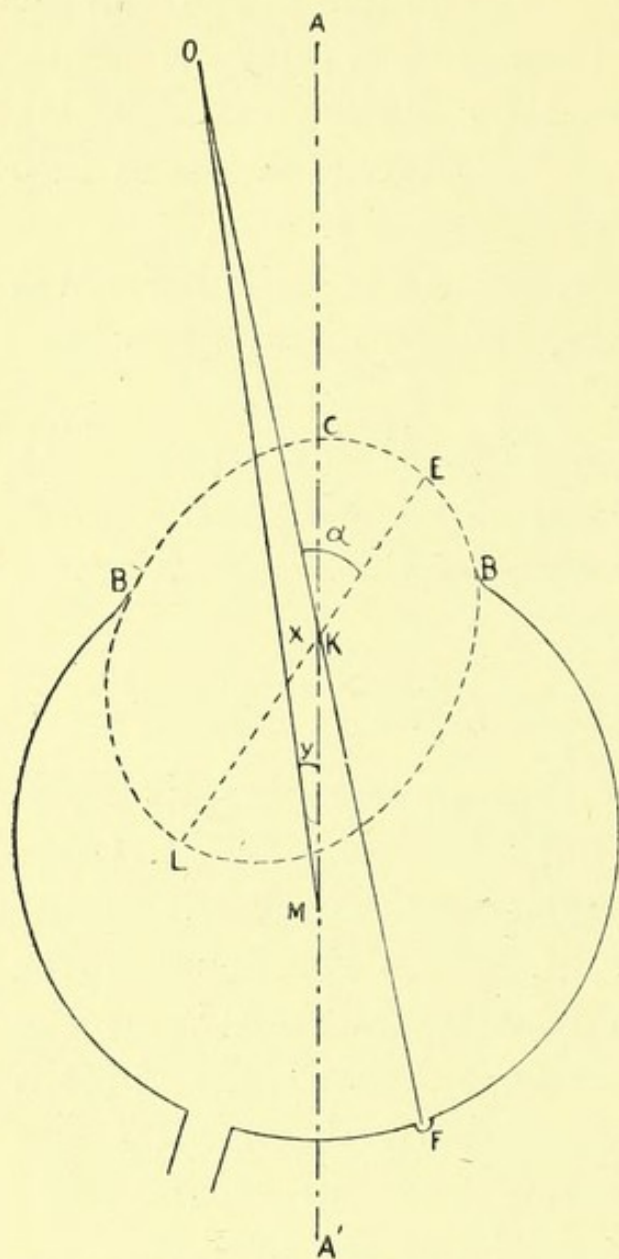


FIG. 15.—A SCHEMATIC FIGURE TO SHOW ANGLES α AND γ . (After Landolt.)

AA' , Optic axis; K , nodal point; M , centre of rotation; C , centre of cornea; BB , base of cornea; EL , major axis of the corneal ellipse; F , fovea centralis; O , point of fixation; KO , line of vision; MO , line of fixation; OXE , angle α ; OMA , angle γ .

some cases of hypermetropia the angle is so great (amounting even to 10°) that it gives the eyes an appearance of divergence (see Fig. 16)—an apparent divergent squint: the eyes, although looking at the point O , appear divergent in the direction $A O'$.

The angle gamma is *negative* when the fovea (F , Fig. 17) is on the inner side of the optic axis

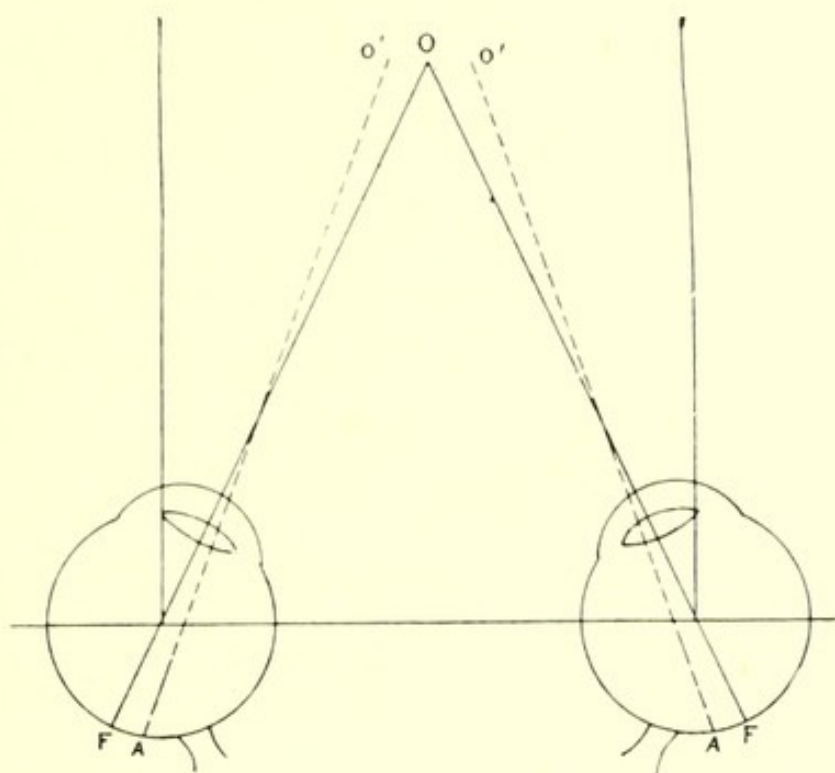


FIG. 16.—AN APPARENT DIVERGENT STRABISMUS DUE TO LARGE POSITIVE ANGLE GAMMA.

—that is, between the optic axis and the optic nerve. In some cases of myopia this is so marked as to give the eyes the appearance of convergence (see Fig. 17)—an *apparent convergent squint*; the eyes, although looking at the point O , appear to converge in the direction of $A O'$.

The angle alpha ($O X E$, Fig. 15) is the angle formed by the axis which passes through the most

curved part of the cornea (the summit) with the line of vision. It is spoken of as positive when, as in Fig. 15, the anterior portion of the corneal axis is situated on the outer side of the line of vision, and negative when it is on the inner side. Generally, the axis of the cornea very nearly co-

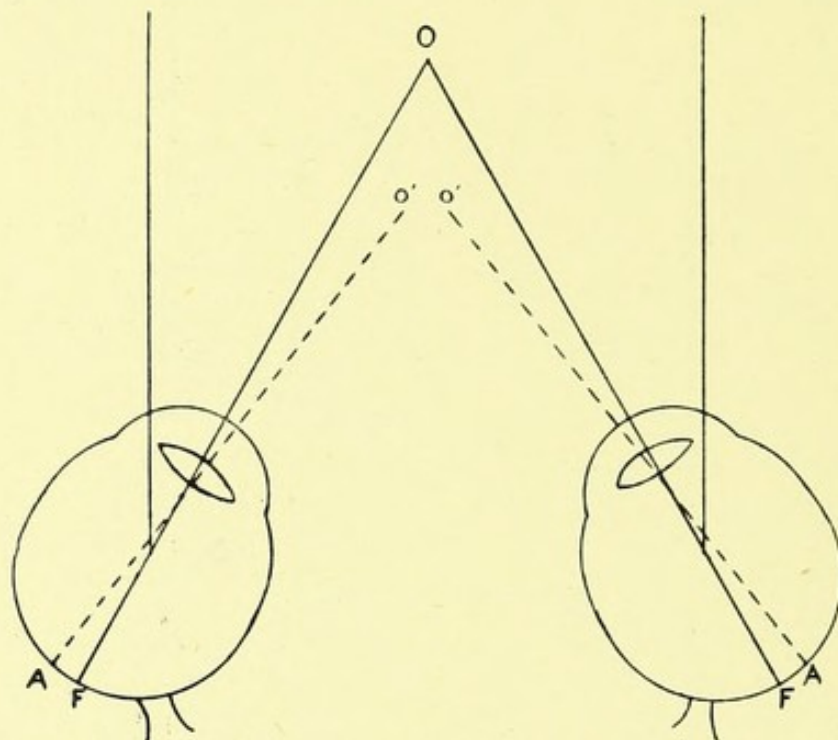


FIG. 17.—AN APPARENT CONVERGENT STRABISMUS DUE TO A NEGATIVE ANGLE GAMMA.

incides with the optic axis, so that for all practical purposes the angles gamma and alpha mean the same thing.

A real divergent strabismus is rare in H. It is usually associated with myopia (page 292).

CONVERGENT STRABISMUS.

Donders was the first to point out that H. was the chief cause of *strabismus convergens*, and he says: "I do not hesitate to declare that it is exceptional to find *strabismus convergens* without H." (page 293). He pointed to the fact that "incipient *strabismus* gives way when the H. is neutralized by a convex glass," and explains why this occurs.

A hypermetrope of 2, when he looks at a point 1 m. off, not only has to use +1 of accommodative power like the emmetrope, but also +2 in addition to correct his defect; thus he uses +3 accommodative power. Now, if he cannot accommodate in excess of his convergence, instead of converging to 1 metre angle, he must converge to 3 *m.a.*—in other words, he squints. Correct his defect by supplying him with +2 convex glass, and the excessive convergence becomes unnecessary, and the squint disappears.

A hypermetrope (uncorrected) cannot have binocular vision and distinct vision; he must choose. Distinct vision is more useful than binocular vision; hence, if it can be obtained by an effort of accommodation, binocular vision is sacrificed, and this is especially the case if one eye is more defective than the other. Opacities of the cornea, and anything that lowers the visual acuity of an eye, helps to bring about a convergent *strabismus* in a hypermetrope, and Donders gives tables, made from the

examination of a number of hypermetropes, to show that the large positive angle gamma is not indifferent in its bearing on the connection between H. and strabismus convergens (page 299). His opponents tried to prove that the above theories were wrong because strabismus was rarely observed in high degrees of H.; but Donders pointed out that this fact was exactly what one would expect, because in high H. clear vision cannot be obtained by any effort, or only by very great effort, and consequently binocular vision is not sacrificed.

Donders made a very true assertion when he said, "H. is a very widely spread anomaly"; and again, "Only in a comparatively small number of cases of H. is strabismus developed" (page 294); and he makes a great point of the fact that "the abhorrence of double images, or, rather, the instinctive adherence to binocular vision, preserves most hypermetropic individuals from strabismus" (page 295).

He had, many years previously, noted this in one of his earliest communications on the subject made in 1845 (*Nederlandsche Lancet* 2° Ser. D. iii., page 233).

In addition to these statements, which are all accepted even to-day, we can add that just as the myope may be able to converge in excess of his accommodation, so do a large number of hypermetropes learn to accommodate in excess of their convergence.

Donders suggested that among the causes of a

convergent strabismus the action of the internal recti might rank; that just as we have insufficiency of these muscles, so they might, conversely, preponderate; and this is most true, for what we now call *esophoria* is a preponderance of the action of the internal recti, which, at rest, causes the visual lines to converge instead of being parallel, and we know how easily this *esophoria* can develop into an *esotropia* or strabismus convergens.

The treatment of strabismus convergens to-day is in the main the same as Donders advised:

1. The correction of the error by the constant use of glasses.

2. The education of the amblyopic eye.

3. The readjustment of the muscle by operation.

It may be news to some to know that Donders recommended the instillation of atropine into the "good" eye of young children, in order to force the *amblyopic* eye to work, and we know how successful this treatment is in many cases.

The only additional treatment for strabismus convergens that exists now is in connection with the fusion sense.

When a child is born, the eyes move independently of each other, and often give the impression of a squint. As the child begins to take notice, it develops the power of fusion. The two images which fall on the maculæ are fused by the brain, and the centre is known as the "fusion centre," or centre of binocular vision.

Some ophthalmologists assume that it is the non-development of this fusion centre that is the cause of squint; but their statement is by no means generally accepted. Anyhow, there is no doubt that we can in quite young children (under six) do a great deal by *educating their fusion sense* by means of stereoscopic instruments.

ASTHENOPIA.

This, with a much wider signification than it held in Donders' day, is included in the present-day word for it—viz., *eyestrain*, and it will be dealt with under that heading (see page 95).

SPASM OF THE ACCOMMODATION (see page 13).

Spasm of accommodation, as a result of the excessive use of the ciliary muscle, is referred to by Donders as occurring in H. (page 622), and he says: "It is a peculiarity of H. that, with the act of vision, tension of accommodation is associated, and thus the H. is in part concealed" (page 239).

It often produces an apparent M., but the true condition is revealed by the use of atropine. It is this spasm that makes it so imperative never to treat errors of refraction in young people without using a cycloplegic.

TREATMENT OF HYPERMETROPIA.

The treatment of H. with glasses differs somewhat now from the rules Donders laid down. He

says: "When H. is still wholly facultative, when persons can even say to us, 'In ordinary life I have no inconvenience, and at a distance I see excellently,' we should not press spectacles on them to be worn constantly" (page 281). We now know so much more about the insidious effects of eyestrain, and that facultative H. is a most constant cause of eyestrain, and also that astigmatism is so constantly associated with H. that we advise the much more constant use of glasses than Donders did.

For the same reason while he advised correcting the whole of the manifest H. and one-fourth of the latent H. we advise correcting at least one-third of the latter.

It follows that in the treatment of H. the *exact* refraction of the eye must be ascertained. Now, Donders himself said: "Without producing paralysis of accommodation, we are never perfectly sure that we determine the refraction in the condition of rest" (p. 107); consequently a cycloplegic must always be used at an examination unless the patient has reached forty or forty-five, atropine, if possible, up to the age of thirty, and homatropine over thirty.

Donders asks whether there is any hope of a radical cure of H., and he further answers this by saying that if development has once taken place, he has never seen H. give way (p. 283), but it is most important to remember that *while development is proceeding*, H. tends to *decrease* towards emmetropia.

A child may be hypermetropic to the extent of

2 D, and as the growth and development of the different parts of the body proceed, the flatness of the eye may disappear, and by puberty the eye may have become emmetropic. For this reason it is necessary to re-examine the eyes of children at least once a year in order to make sure they are not wearing too strong a convex glass, which would induce an artificial myopia, which in turn might lead to real myopia. This tendency for the eye to grow normal is often interfered with by the presence of eyestrain; hence one of the benefits derived from glasses in young children is the increased chance of the eye improving and growing to the normal shape, and the possibility of discontinuing the use of spectacles.

MYOPIA

DONDERS was the first to point out that myopia was the opposite condition to hypermetropia. He emphatically states that typical M. depends on *elongation of the visual axis*, a conclusion which has never since been contradicted.

Myopia, or short-sight, is a condition of the eye in which the retina is situated behind the principal focus (Fig. 12, *M.*), and only divergent rays from a

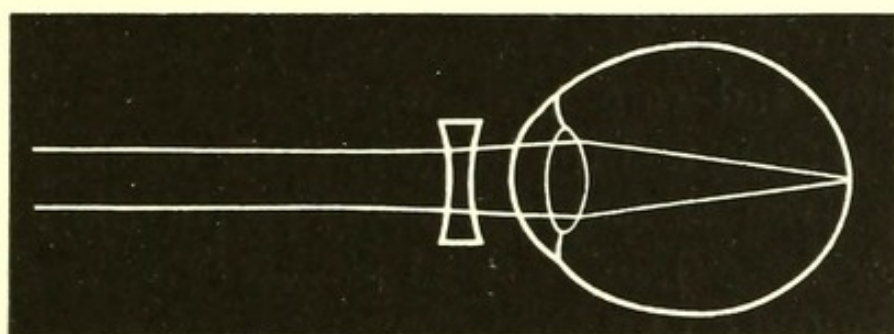


FIG. 18.

near-point, or parallel rays made divergent by a concave glass (Fig. 18), can come to a focus on the retina.

The retina of a myopic eye is the conjugate focus of an object situated at a short distance in front of the eye, or, in other words, the punctum remotum of a myope is always at a definite distance (less than 6 metres), the distance being measured by the

amount of myopia. Thus a myope of 1 D has his far-point 1 metre from the eye, or a myope of 2 D has his far-point $\frac{1}{2}$ metre, or 50 cms., and a myope of 5 D, 20 cms. from the eye.

A myopic eye sees distinctly distant objects (when accommodation is relaxed) with that concave glass whose focal length is equal to the distance of the far-point from the eye, and the converse is true; the measurement of myopia is that concave glass with which the myopic eye sees distinctly objects at a distance, and its focal length, is equal to the distance of the myope's far-point from the eye. If the accommodation be relaxed, the weakest concave glass is the measure of the myopia.

In reading Donders' writings on Myopia in his classical treatise, one is particularly struck with the accuracy and care with which he made all his investigations, and the masterly spirit which guided him. Even as long ago as 1845 (in the *Nederlandsche Lancet*), he sounded the note that ought to have guided all his followers—viz., *that myopia was the result of excessive convergence*. He pointed out that it was a defect of civilization, that the most civilized nations were the greatest sufferers, and that those employed in close work with both eyes were particularly marked out. He refers to the fact, which is so true, that those who do close work with one eye, such as watchmakers using a magnifying monocle, do not necessarily become myopic (page 344).

Donders says: "In the higher degrees of myopia

the difficulty of maintaining binocular vision does not proceed from tension of accommodation, but rather from difficulty of convergence" (page 120). In other words, the exophoria may be latent for some time, but sooner or later it may easily give place to exotropia or divergent strabismus.

He says that the range of accommodation is almost equal in the myope to the normal eye, and gives a diagram of a case of fairly stationary myopia of -1.75 D on page 346.

In my diagram (Fig. 8) which gives the near-point p of 600 individuals in which the myopia has been *previously corrected*, it will be seen that the range of accommodation is a little larger, and that the presbyopic point is reached about three years later (see Presbyopia, p. 26).

I reproduce below a diagram, showing the mean-line of the near-point in an *uncorrected* myope of 3 D arranged on Donders' plan. It will be seen here that the p line does not reach the 22 cms. spot (the Presbyopic Point) until the age of fifty-five.

Such a person very often does not recognize that he is short-sighted, and is very proud of being able to read without glasses at an age when others have been wearing reading-glasses for many years (see Donders' reference to this on page 213).

Donder's classical description of the fundus changes in high myopia cannot but evoke the highest admiration; there is little of importance to add after fifty years!

Here again he sounds the note that rings out so

clearly: "The undue use of the eyes (for near work) promotes the occurrence of staphyloma posticum" (page 386), and he points out that excessive convergence causes increased pressure on the eye, that this pressure causes the bulging at the posterior pole on account of the sclera being abnormally thin here, and the parts not supported by the muscles.

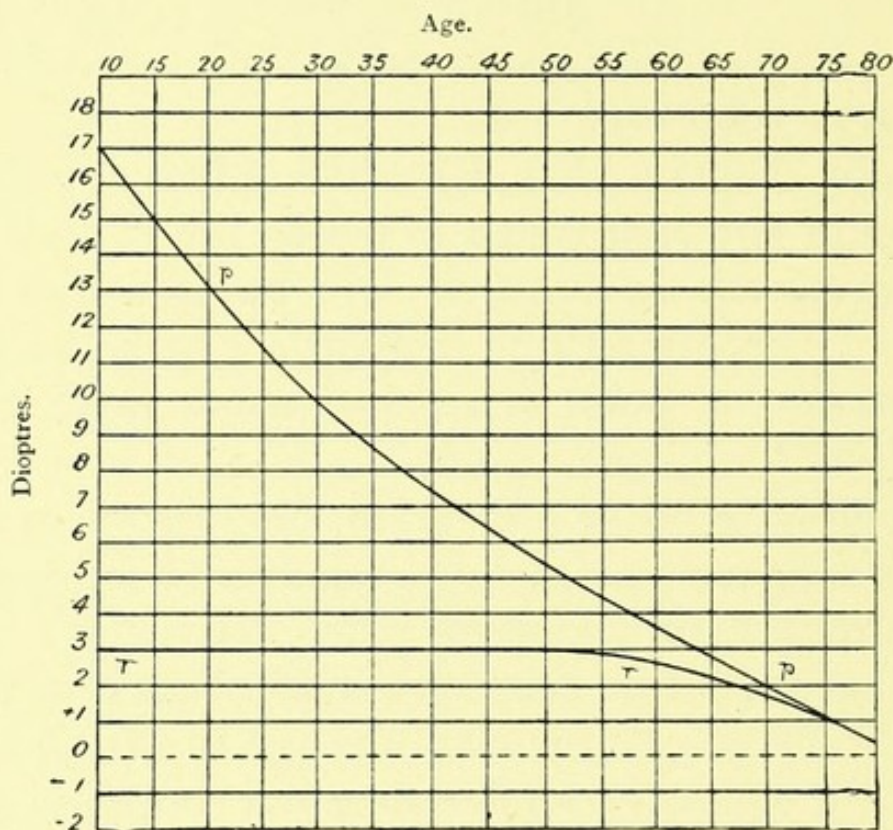


FIG. 19.

A myope requires more convergence of the visual lines because vision takes place closer to the eyes, and, as Donders has shown, precisely in myopia is this for two reasons more difficult, first on account of impeded movements, due to the altered shape of the eyeball, which becomes ellipsoidal in form, and which has to move in a cavity of similar shape ;

and, secondly, on account of the altered direction of the visual lines, the angle gamma (angle formed by the visual and optic axis) being smaller than in emmetropia or hypermetropia (see page 57). If a myope cannot dissociate his accommodation and convergence, he has the same difficulties as a hypermetrope; he can either see distinctly but sacrifices binocular vision to remove the diplopia, or he can use his accommodation when he does not require it, and see distinctly.

The relative amplitude of accommodation and convergence vary considerably, not only according to the refractive error, but also in different individuals with the same error. There is a tendency for the accommodation to adapt itself to the altered state of refraction; hence most myopes can converge in excess of their accommodation; and when the myopia increases, the excess of convergence over accommodation also increases.

Donders calls attention to a fact which daily experience confirms—that when the work is approached very near the eye, but convergence is not used, as in the case of watchmakers, who habitually use a strong convex glass in *one* eye, there is no special tendency to myopia (page 344).

The excessive convergence and the excess of convergence over accommodation lead to strain of the internal recti, and then to “insufficiency,” which we now call “exophoria,” and this in turn leads to exotropia, or strabismus divergens.

Donders points out that just as H. leads to

“accommodative asthenopia,” often ending in converging strabismus, so M. leads to “muscular asthenopia,” often ending in diverging strabismus (page 415). He asserts that “in at least 90 per cent. of the cases of relative diverging strabismus” M. is present (page 410).

TREATMENT.

As previously stated, Donders gave the key to the treatment of Myopia when he asserted that it was the result of excessive convergence, and one naturally expects to find that he advocates the correction of the error. He says: "*When the myopia is slight in reference to the range of accommodation, and the eye is otherwise healthy, in this case neutralizing glasses may be worn as spectacles, and may be used even in reading and writing. I think it even desirable that this should be done. When persons with moderate degrees of myopia have in youth accustomed themselves to the use of neutralizing spectacles, the eyes are in all respects similar to emmetropic eyes, and the myopia is, under such circumstances, remarkably little progressive. I am acquainted with numerous examples of this even among those of my friends who have passed their lives in study. Glasses of - 4 D adopted at seventeen years of age are often still sufficient at forty-five years, both for seeing acutely at a distance and for ordinary close work. Not until the age at which emmetropes need convex spectacles, and often even some years later, do the neutralizing spectacles become rather too strong for close work, and it is desirable to procure somewhat weaker ones, which, with the narrower pupil peculiar to that time of life, are now nearly sufficient for distance also*" (page 421). He further says that when the M. is high we always have to begin with

weaker glasses, and gradually work up to the proper correction.

“The effect of wearing glasses is, in fact, that the relative range of accommodation is displaced, becoming gradually the same as the position proper to emmetropic eyes, and therefore the binocular farthest point approaches the eye while the absolute farthest point *r* by no means does so. The myopia thus neutralized is less progressive, because both too strong convergence and a stooping position are avoided” (page 422).

Since his day some ophthalmologists have advocated the practice of allowing those suffering from low M. to do near work without glasses, and when the M. was high, have given weaker glasses for near work. The consequence has been that as the convergence was still being used in excess, the Myopia tended to progress. If Donders' teaching had been followed, this error would never have been made, an error which has kept progressive M. and malignant M. dreaded for so many years.

Following on Donders' teaching, and making use of our increased knowledge, which indicates the importance of correcting low errors of astigmatism, we have made great advances in recent years. The full correction of the error (with the smallest minus cylinder), except in cases of very high M., leads not only to the arrest of the progress of the M., but in some cases to its distinct diminution.

In a paper read in 1904 at the British Medical

Association meeting at Oxford, I cited 532 myopes that had been treated by *full correction*.

The myopia ranged from .75 to 20, and the average period of observation was four and a half years. The following table shows the result :

532	{ 469 remained stationary, and of these, in 162, the visual acuity improved.			
	{ 63 progressed.	{ Average age, 15.	{ M. from -1 to -11.	Increase limited to 1 D, 47.
				" " 2 D, 13.
				" " 4 D, 3.

If we exclude the 47 whose increase was limited to 1 D, we have 16 left—*i.e.*, only 3 per cent. progressing.

The modern treatment of M. may be thus briefly stated : In early life the treatment of myopia is mostly preventive. It is rare to come across a child under the age of six with actual myopia. Apparent myopia may show itself (*a*) from spasm of the ciliary muscle, distant vision being subnormal and improved by concave glasses, and near work being approached very close to the eyes ; (*b*) in a young patient with high hypermetropia, in which case distant vision is poor and near work is held very close to the eyes in order to acquire large retinal images ; (*c*) in children who have acquired the habit of holding their work near the eyes, either through faulty illumination or on account of reduced visual acuity produced by some disease of the eyes, such as corneal nebulae.

In all these cases the true error is revealed by a cycloplegic ; consequently no attempt should ever

be made to treat a young myope without previously paralyzing the accommodation, and *atropine* should, if possible, always be used.

The preventive treatment is more especially indicated in all children whose parents are myopic, for they have probably inherited an "anatomical predisposition" to myopia.

Bearing in mind that excessive convergence is the most potent cause of myopia, the most rigid attention should be paid to ophthalmic hygiene. The schoolroom should be lofty and large, and have high windows on one wall. The seats and desks should be arranged in rows, so that the students sit with the windows on their left. When practicable, each scholar should have an adjustable seat and desk; but where this cannot be arranged, as most children of the same age are of the same height when sitting, and in the same class, the height of the desk from the seat should increase gradually with the classes, the highest class having the highest desk.*

The school-work that needs close application of the eyes should be continued only for a short period at a time, the period alternating with other work which does not require the use of the eyes, such as mental arithmetic, demonstrations, or play.

Schoolmasters should teach more—that is, they should explain and impart knowledge by demon-

* The desk should have a slight slope, and its height should be so regulated that the scholar can sit with head upright and the eyes about 33 centimetres from the work.

strations and simple lectures, and reduce as much as possible the time spent in "home preparation," which is usually work done by bad light, and when the student is physically and mentally tired.

Even in the nursery the greatest care should be taken with the children's sight. They should have large toys, and among these there should always be a large box of plain wooden bricks. Picture-books should be discouraged, and close work that entails undue convergence, such as sewing, threading beads, etc., should be forbidden. The nursery governess can teach them their letters and small words, and even simple arithmetic, by means of the wooden bricks.

No child with a tendency to myopia or with a myopic family history should be allowed to learn to write or to draw until at least seven years old.

The child's bed should not be allowed to face the window ; preferably it should be back to the light.

Having ascertained the concave glass that corrects the myopia of each eye under atropine, we may, in quite young patients, order such glasses for constant use ; in those over puberty it is wise to delay prescribing until the effects of the atropine have passed off, not only because an increase of .5 may very distinctly improve vision, but because it is important to try the glasses binocularly when the eye is in the normal state. It is too often forgotten that the eyes are not single optical instruments, and we often find that a *weaker* pair of

concave glasses give as good vision as a stronger glass used monocularly.

The only certain method of arresting the progress of myopia is to establish a normal state in which the ciliary muscle is strengthened by being forced to work, the excess of convergence over accommodation stopped, and excessive convergence made impossible ; and this can only be achieved by insisting upon the *constant* use of the glasses, and *refusing to give weaker ones for near work*. The patient can see his near work so much better *without glasses* that we may have some trouble at first in enforcing this treatment.

Of course the precaution must be taken of removing the glasses when rough games are being played.

In adults the treatment of myopia should be carried out in the same manner, substituting *homatropine* for atropine in those who cannot afford the time from work that the latter entails.

If the myopia be somewhat high, say 6 D or over, and has never been fully corrected, we may have to give glasses for near work, 1.5 or 2 D weaker ; but the patient should be strongly advised only to wear these on special occasions when fine work is being done, or by artificial light.

The older the patient and the higher the myopia, the more difficult will it be for him to use the distance glasses for near work, because his accommodation has been so long idle that the ciliary muscle is considerably atrophied.

If the patient be a student or engaged in literary or other work which entails close application for many hours a day, and if he be free to regulate his work, he should be advised to work for shorter periods and to take longer intervals of rest, and be especially careful to have his work always in a good light.

In patients of thirty years of age and up to forty homatropine should be used when practicable. Over forty, no cycloplegic is required. If the patient has never had the full correction, he will at this age be unable to read with his distance glasses, and weaker ones must be given, preferably in the form of bifocals. All the more will this be the case when he arrives at forty or forty-five—the emmetrope's presbyopic period. No rigid rule should be observed, but each case should be treated according to its requirements.

After carefully testing the patient, we should find his working near-point, and keep more accommodation in reserve than would be required in the emmetrope, because the ciliary muscle is weaker (see Presbyopia, page 35).

Some adults with a small amount of myopia obstinately refuse to wear the constant correction; ladies will wear lorgnettes at the theatre, etc., men will wear a monocle. If no astigmatism be present, this may be allowed so long as no increase in the myopia takes place, but only on the condition that the patient is re-examined at frequent intervals.

HIGH MYOPIA.

The treatment of high myopia is somewhat different. When the young adult has never worn the full correction, it will be useless to prescribe it, even for distance, at first. We should reduce the glass as little as possible, and test binocularly. For instance, the myopia may be 20 in both eyes, but — 18 before each eye is the strongest glass the patient will tolerate. These we order for constant use, and perhaps we find later that the full correction will be accepted. In older patients not only have we to be satisfied with a reduction in the distance glasses, but we must often take off as much as 4 D for near work.

When recent fundus changes are present in young patients, the eyes should be kept under atropine for a long period, the correcting glasses should be well tinted, and a country open-air life should be strongly recommended, with complete cessation of all close work while the changes are active; older patients should be warned against stooping or straining, and should be strongly advised to do little, if any near work.* In all these cases so much depends on the general health that it is wise for the surgeon to place them under the care of a physician, who, among other things, can advise as to aperients, the means of reducing high blood-pressure when present, etc., and by this care we may avert retinal hæmorrhage, which is so liable to occur.

* See Donders, page 435.

When any fear exists as to the possibility of detachment of the retina ensuing, the patient should be especially warned against riding on horseback, jumping, or doing any act which may jar the body.

THE TREATMENT OF HIGH MYOPIA BY DISCISION AND REMOVAL OF THE LENS.

Some ophthalmologists advocate that when the patient is not older than twenty-five, the myopia very high, vision very poor and not improved by glasses beyond $\frac{6}{24}$, and when the fundus changes are not very marked, and especially when the myopia is progressive, the lens may be removed by discision.

If lamellar cataract or other congenital opacities of the lens are present, such treatment appears to be quite permissible ; but there are many who shrink from advising the operation if the lenses are clear, and it is interesting to note that Donders quite agreed with this, as he wrote that he considered that anyone advising it would exhibit culpable rashness !

ASTIGMATISM

THE whole subject had been very carefully and thoroughly written upon by Sturm, Fick, Helmholtz, and others when Donders published in 1862 "Astigmatisme en cylindrische Glazen," and in 1863, in co-operation with Middelburg "De Zitplaats van het Astigmatisme" and added knowledge to the subject which was of supreme importance.

The result of his investigations and measurements led him to state that the seat of regular astigmatism is chiefly in the cornea, and that the direction of the principal meridian for the whole dioptric system, as well as for the cornea in particular, is of that nature that the meridian of maximum curvature usually approaches the vertical, and that of minimum curvature to the horizontal (page 490). This coincides with the astigmatism "*selon la règle*" and "*contre la règle*," or the "direct" and "inverse" astigmatism of to-day.

Donders further made this important statement: "That with a high degree of asymmetry of the cornea, asymmetry of the crystalline lens exists, acting in such a direction that the astigmatism for the whole eye is nearly always less than that proceeding from the cornea."

This was further developed by Dobrowolsky,* who found that the astigmatism of the lens was either static or dynamic that static astigmatism generally added itself to that of the cornea and thus increased the astigmatism of the whole eye; and that dynamic astigmatism was as a rule the opposite of that of cornea, was produced by an unequal contraction of ciliary muscle, and corrected, and so concealed the astigmatism of the eye. Dobrowolsky published his results in 1868, and we can fairly give Donders the credit of foreshadowing them.

Since then this unequal contraction of the ciliary muscle producing the corrective astigmatism of the lens has been denied by some, but the clinical proofs are overwhelming.

Take a typical case: A patient complains of headache accentuated by near work. Examination reveals no refractive error. The ciliary muscle is paralyzed, and astigmatism is discovered. This is corrected by cylinders, the glasses are ordered to be worn always, and in a short time the headache disappears. Again, very often when the effect of the cycloplegic has passed off, the patient refuses the cylinder that improved his vision under atropine. He tells you that it makes his vision worse. In spite of this you prescribe it and—this is a very important point—you insist on the glasses being worn always. He returns in a month or two,

* "Ueber verschiedene Veränderungen des Astigmatismus unter dem Einfluss der Accommodation" (*Arch. für Ophth.*, xlx. iii., p. 51, 1868).

assuring you that his headache has entirely disappeared, that he has become accustomed to the glasses, but that he cannot now see as well without them as he could before using them.

What has happened? At first, when the effect of the atropine has passed off, the ciliary muscle returns to its old habit of unequal contraction, and consequently the correcting glasses, instead of helping, make matters worse; but by constantly wearing them the necessity for this unequal contraction disappears, the muscle resumes the normal condition, and allows the glasses to do the work. Vision is apparently worse without the glasses, because the muscle has forgotten its old habit; but, of course, like all bad habits, it can be easily re-acquired. The patient has lost nothing but his headache. What stronger proofs could there be that this unequal contraction does occur!

We know now that this corrective astigmatism is very small, perhaps not more than .5 D; consequently it can only neutralize a low degree of astigmatism of the cornea, and it is in these cases that eyestrain so commonly occurs.

So here again we see that Donders laid the foundation which has been built upon by workers following him, aided by improved methods of examination and perfected instruments, such as the ophthalmometer of Javal and Kagenaar, and finally in our hands as the perfect instrument of Meyrowitz.

Donders' classification of the varieties of regular astigmatism (page 482) has never been improved upon, and is as follows:

Variety of Astigmatism.	Refraction of the Principal Meridians.	Position of the Principal Focus.
1. HYPEROPIC ASTIGMATISM :		
(a) Simple ...	{ Emmetropic. Hyperopic.	On the retina. Behind the retina.
(b) Compound	Both hyperopic.	Both behind the retina, one being nearer than the other.
2. MYOPIC ASTIGMATISM :		
(a) Simple ...	{ Emmetropic. Myopic.	On the retina. In front of the retina.
(b) Compound	Both myopic.	Both in front of the retina, one nearer than the other.
3. MIXED ASTIGMATISM :		
	{ Hyperopic. Myopic.	Behind the retina. In front of the retina.

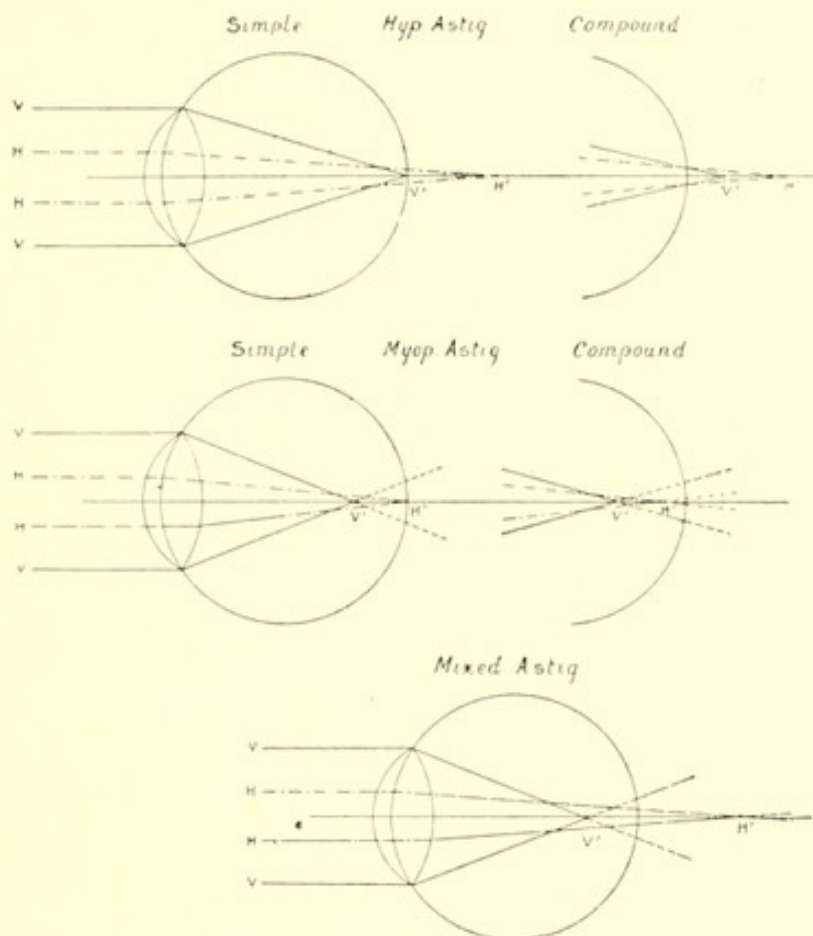


FIG. 20.

V, Rays passing through the vertical meridian ; *H*, rays passing through the horizontal meridian.

All the examples in the above (Fig. 20) show *direct* astigmatism (selon la règle); when the horizontal meridian is most convex it is called *inverse* astigmatism (contre la règle).

Donders found that the majority of astigmatic patients suffered from the hypermetropic form (page 520).

The refraction of 2,136 astigmatic eyes taken consecutively from my private case-books showed that 1,411 were hypermetropic, 597 myopic, and 128 mixed.

As "direct" astigmatism is the commonest form, it follows that the horizontal meridian is defective in simple *H. Ast.*, and the most defective in compound *H. Ast.* in the majority of cases. Donders cites what we may almost call an historical case of bad eyestrain in a patient with simple *H. Ast.* (direct).

I quote fully: "A clerk, aged twenty-six, recorded the following: 'My occupation is that of a clerk. The first effort to work was the most painful. Thereupon dazzling soon followed, obliging me to shut my eyes, and to keep them closed for some time. After that my work went on somewhat better, but I found it impossible to work all the forenoon; I was constantly obliged to leave off. At the end my eyes were painful, and I felt best when I walked for a considerable time in the open air, out of the sun. In the evening, by gaslight, my work went on pretty well at first, but soon red dazzling came on. I was then obliged every time

to leave off, and with fatigued and painful eyes I returned home.' 2.25 cyl. were given, *axis vertical*, and he reports: 'On using the spectacles I found even on the first day, an incredible improvement' (his acuteness of vision was, in fact, brought from $\frac{2}{7}$ to $\frac{3}{4}$). 'Next day I experienced no painful affection, and I found it easy to work uninterruptedly the whole morning. I saw everything infinitely sharper. In the evening I experienced not the slightest inconvenience from the light. In the open air, too, when I walk without the spectacles, I am free from pain. Spectacles which I had before tried (ordinary spherical glasses) had been of no use to me' " (page 521).

Donders thinks this bears out his statement, viz., "that in the pure form of asthenopia, hypermetropia is scarcely ever wanting" (page 261).

Donders appears to be the first to call attention to the acquired astigmatism after operation on the eye, such as iridectomy and cataract extraction, although he does not mention that the acquired astigmatism is generally the "inverse" form (*contre la règle*).

Donders remarks that a slight inclination of the crystalline lens is sometimes the cause of regular astigmatism, and cites himself as an example (page 468).

TREATMENT.

In the treatment of astigmatism considerable alterations have taken place since Donders' day.

He says: "I have never met with an eye whose focal distance in the different meridians was absolutely the same" (page 174). In other words, he believes in the almost universal existence of astigmatism; but he says: "So long as astigmatism does not essentially diminish the acuteness of vision, we call it normal" (page 456), and he does not advise the correction of "normal astigmatism."

The pendulum has swung now in the opposite direction; astigmatism that is high enough to lower the visual acuity will take care of itself; the patient calls out for glasses and himself seeks the oculist; but the small amount of astigmatism that is concealed and is only revealed under a cycloplegic is probably only suspected by the physician.

All those engaged in refraction work should observe two golden rules:

First Rule: Always suspect the presence of astigmatism.

Second Rule: Never be satisfied that astigmatism is eliminated unless the examination has been made under a cycloplegic in all under forty or forty-five years of age.

There is no refractive error in which cycloplegics are of such paramount importance as in astigmatism of a small amount. The ciliary muscle has formed a bad habit (of which the patient is often quite unconscious), and only gives up this habit when forced to do so by being paralyzed.

Cylindrical lenses correct regular astigmatism of the cornea, and when the error is small, do the work that the ciliary muscle has been doing at so great a cost to the nervous system. When the error is large, certainly when it is over .75 D, the ciliary muscle cannot correct the defect, and consequently makes no attempt to do so; but the greatest care must be exercised in giving the exact cylinder that corrects the defect, because, if a small portion is left uncorrected, the ciliary muscle can do the rest of the work, and strain results. For instance, by retinoscopy we find an eye with the horizontal meridian showing +4 and the vertical +2. We find that a cyclinder +2 axis vertical, and a sphere +1 give $\frac{6}{8}$, and when the effects of the cycloplegic have passed off, we give, say, cylinder +2 axis vertical. Had we been a little more careful, we should have found that the best result was obtained by a cyclinder +2.25 axis vertical, and this .25 which we have omitted to correct, is corrected by the lens, and we introduce eyestrain, which did not exist before.

Another important rule to observe in the treatment of astigmatism is that it should be *fully* corrected, and when the patient has recovered from the cycloplegic, neither the power nor the axis of the cylinder should be altered.

It is hardly necessary to add that these changes in the treatment of astigmatism are due to our increased knowledge of eyestrain and its evil effects, and to our increased facilities for diagnosing and estimating small errors.

ANISOMETROPIA

ANISOMETROPIA is a condition in which the refraction of the two eyes is different. A difference in refraction in the two eyes is more often met with than absolute equality, and in astigmatism it is very common to find a difference of .25 or .50 between them.

In Donders' time it was the practice only to recognize anisometropia when, to produce the maximum of visual acuity in the two eyes, different glasses were required; but the increased knowledge we have now of the effects of eyestrain has taught us that the smallest amount must not be neglected, and, further, that the smaller the amount of anisometropia, the more important is it to correct it.

Every possible combination may exist:

1. One eye may be emmetropic, and the other ametropic.

2. Both eyes may be ametropic:

- (a) The same variety of ametropia, but unequal in degree.

- (b) Different varieties of ametropia, one eye myopic, and the other hypermetropic (this variety is sometimes called "antimetropia").

When one eye is astigmatic and the other hyper-

metropic, or myopic, the astigmatic eye has generally the same form of ametropia as the non-astigmatic eye, and very often one of its meridians has the same amount of ametropia.

Donders refers to this "harmony of refraction on both sides" (page 558), and he also draws particular attention to the fact that "asymmetry of the eyes is usually combined with asymmetry of the other parts" (page 558). He says further: "I can only in general maintain that at the side where the strongest refraction, or rather the longest visual axis occurs, the orbit (and with it the eye) is situated closer to the median line, while its surrounding edges are placed more forward" (page 558).

With his clear classifying method he divided this condition into acquired and congenital.

Acquired asymmetry of the eyes is the result of operation or the loss of accommodation in one eye.

Congenital asymmetry he divided into—

I. SIMULTANEOUS BINOCULAR VISION.

For binocular vision to exist in anisometropia, the difference in refraction between the two eyes—that is, the degree of anisometropia—must be small, although cases have been recorded where it has amounted to 6 D. Under these circumstances, although the magnitude and acuteness of the images in the two eyes are unequal, they overlap and help each other.

If each ciliary muscle could act independently,

the anisometrope could very often correct each eye by a separate and independent accommodation in each eye ; but it is generally believed that the same effort of accommodation is made on both sides, with the result that only one image is sharp. At the same time, it should be noted that when the anisometropia is of low degree, some patients may have the power of producing asymmetrical accommodation to a limited extent. If we place a convex glass of $+ .50$ in front of one eye and a concave glass of $- .50$ in front of the other, and thus produce an anisometropia of 1 D and attempt to read with the glasses, a very distinct feeling of strain is experienced ; but whether this strain is brought about by actual asymmetrical accommodation, or only by the attempt to produce it, it is difficult to say.

Donders believed that with "even a slight difference in refraction *we are not able to adjust by accommodation*" (page 560).

The treatment in these cases varies considerably. In quite young patients, unless the difference between the eyes is very great, the full correction of each eye should be ordered in accordance with the plans stated in the previous pages. In older patients we must try the binocular effect before prescribing glasses. The full correction in each eye is especially indicated when marked symptoms of eyestrain exist, and in these cases, although the treatment may be complained of at first, it is wise to insist upon it, and in many cases

the discomfort produced at first by the glasses disappears in a few weeks, and eventually the symptoms of eyestrain disappear. On the other hand, patients who have lived many years without having their anisometropia corrected have become so accustomed to the difference between the eyes that the removal of this difference not only confers no benefit, but proves irksome, and they may complain of dazzling, head-swimming, and headache.

2. THE EYES ARE USED ALTERNATELY.

One eye is emmetropic or slightly hypermetropic, and is used for distance, and the other myopic, and used for near work. If eyestrain be not present, the patient may prefer to have no glasses. Although he has lost binocular vision, he has gained other advantages ; in some cases he can entirely dispense with muscular effort, his ciliary muscle and internal recti being rarely used. If eyestrain be present, we must prescribe glasses after ascertaining the binocular combination that suits best. We sometimes find that if, for instance, both eyes are hypermetropic, the patient prefers the same glass in both eyes corresponding to that required by the most ametropic ; this latter eye will then be used for distance and the other for near work. In the same manner, if both eyes are myopic, we give each the correcting glass of the weaker.

3. ONE EYE IS PERMANENTLY EXCLUDED FROM VISION.

When the difference between the eyes is great, the more defective eye is little used, and tends to become amblyopic, if it is not so already. In such cases we must give each eye its proper correction, and instruct the patient to practise the amblyopic eye by totally excluding vision with the good eye by covering it with a patch for a certain time every day.

In "amblyopia exanopsia," occurring in young patients with convergent strabismus, this treatment, patiently persevered in, is often most satisfactory.

The defective eye may never take its proper place in binocular vision, but in some cases it may become very useful, especially if any damage or disease should effect the good eye ; and, moreover, the cosmetic effect which sometimes occurs is considerable, for if treated in time, the strabismus, which so often appears in these cases, may be prevented.

EYESTRAIN

EYESTRAIN

EYESTRAIN, as we now understand it, has a much wider meaning than it had fifty years, or even twenty years, ago. It includes the "asthenopia" of Donders and the "muscular asthenopia" of v. Graefe.

The word "asthenopia" denotes weak sight, which rarely exists in such cases, and even if we take its broader meaning, "tired sight," we find it equally inappropriate, because too limited. It is therefore best to restrict this term to retinal fatigue (*retinal asthenopia*), with which we are not here concerned.

Up to the time of Donders, asthenopia was believed to be due to an affection of the retina or choroid (Taylor and Lawrence), fatigue of the nerves (Scarpa), or the commencement of amblyopia (Sichel), and Mackenzie took such a gloomy view of it that he advised patients suffering from asthenopia to give up their trade or profession and do country work, go to sea, or emigrate! At last, in 1858, Donders, in the "*Nederlandsch Tijdschrift voor Geneesk.*," Jaarg., 1858 (p. 473), announced his discovery that asthenopia was due to an anomaly of refraction (p. 274). It is true that he considered

the hypermetropic structure of the eye was the paramount cause, and we now know that, although H. is constantly associated with it, astigmatism is the most common cause. Still, that does not detract from the supreme value of his discovery. Donders led the way where others were able to follow.

Let us for one moment think of the thousands who have by this discovery been saved from constant headaches, inability to do their proper share of work in the world, a life of misery and ill-health, and even premature death, by the correction of a small error of refraction, and let us never forget that Donders brought this about.

Donders' "asthenopia" was distinctly more limited than our present "eyestrain." I quote his words: "The eye has a perfectly normal appearance, its movements are undisturbed, the convergence of the visual lines presents no difficulty, the power of vision is usually acute, and, nevertheless, in reading, writing, and other close work, especially by artificial light or in a gloomy place, the objects, after a short time, become indistinct and confused, and a feeling of fatigue and tension comes on, in, and especially above, the eyes, necessitating a suspension of work. The person so affected now often involuntarily closes his eyes and rubs the hand over the forehead and eyelids. After some moments' rest, he once more sees distinctly, but the same phenomena are again developed more rapidly than before. The longer the rest has

lasted, the longer can he now continue his work. It is remarkable that pain in the eyes themselves, even after continued exertion, is of rare occurrence" (page 259). He considered that besides H., diminished accommodative power after exhausting disease, insufficiency of the internal recti (Graefe's "muscular asthenopia"), and, finally, astigmatism, might lead to asthenopia. Compare this with our present definition of eyestrain. Eyestrain may be defined as a symptom, or group of symptoms, produced by the correction or attempt at correction by the ciliary muscle of an error of refraction, or as a want of balance between the external muscles of the eye.

There is nothing in this definition which does not conform with Donders'; we have extended the meaning, that is all.

Methods for examining the eye and estimating refraction are far superior to what they were fifty years ago. Retinoscopy and ophthalmometers have enabled us to estimate the smallest errors of refraction, and we know that it is these small errors that are the most dangerous. Where gross errors exist, either in the refraction or in the muscular equilibrium, the patient cannot correct, and consequently makes no attempt to correct the defect, and eyestrain is not produced. The smaller the error the more likely is eyestrain to be present, and also, unfortunately for the patient, the more likely is it to be overlooked.

I have already referred (under Astigmatism, page 80), to the discovery made by Donders, that

—I quote his words—“with a high degree of asymmetry of the cornea asymmetry of the crystalline lens exists, acting in such a direction that the astigmatism for the whole eye is nearly always less than that proceeding from the cornea” (page 492). In other words, he found that the lens tended to correct some of the astigmatism. We now know that in low errors of astigmatism (certainly not over .75 D) of the cornea, an opposite astigmatism of the lens can be produced by the unequal contraction of the ciliary muscle, and that this is a most prolific cause of eyestrain.

The symptoms of eyestrain may be grouped under three headings:

1. Manifestations on the eye and lids.
2. Peripheral irritation.
3. Nerve exhaustion.

1. MANIFESTATIONS OF EYESTRAIN ON THE EYE AND LIDS.

Eyestrain means an increased demand for work on the part of the ciliary or external muscles, which determines an increased flow of blood to these parts; if this is constant, congestion is liable to follow, and with it pain.

With the parts thus rendered specially receptive to infective germs, there is little difficulty in understanding that inflammation should ensue, and thus we find that blepharitis, corneal ulcers, phlyctenules, iritis, cyclitis, and glaucoma may have eyestrain as

one of the predisposing causes. There is also little doubt that cataract may be started by the irregular contraction of the ciliary muscle in correcting low errors of astigmatism, and that the correction of these errors, with the consequent disappearance of the eyestrain, will stay the progress of the disease.

There seems little doubt that eyestrain tends to hasten the sclerosis of the crystalline lens (which naturally takes place with advanced years), resulting in diminished accommodative power, *i.e.*, premature senility (see page 31).

2. PERIPHERAL IRRITATION.

- (a) With pain.
- (b) Without pain.

(a) *Peripheral Irritation with Pain.*—In the same manner that a decayed molar may produce neuralgia by peripheral irritation, so eyestrain may produce headaches or any other form of peripheral irritation. Headache is by far the commonest symptom. So common is it, indeed, that no physician should attempt to treat a patient for constantly recurring headaches unless the existence of eyestrain has been eliminated by proper examination under a cycloplegic.

The position and character of the headache form no guide to the cause, for ocular headache may be of any possible variety. It may be unilateral as a typical hemicrania, and may be indistinguishable from a true megrim attack.

There is no rule as to its position ; this varies with the individual. In some it is superficial, akin to neuralgia ; in others, deep-seated.

The commonest form of eyestrain headache is a pain over one or both brows, often termed "brow ague."

Again, the time of the headache varies ; it may be a permanent headache, or periodic, and it may appear to have no direct association with excessive use of the eyes.

Early morning headache is a very common form of ocular headache, and this astonishes most patients, as they imagine that the night's rest should have removed the possibility of this.

To explain the periodicity of the headaches, we have to remember that the cause of the headache may be multiple. There may be two, or even three, factors present. A patient who suffers from a periodical headache may have eyestrain, a gouty or other diathetic tendency, and at times, added to these, some special nerve depressant, such as worry or trouble.

Now, each of these factors, or possibly any two, may not suffice to cause a headache ; all three must be present, and only at such times as all three are present is the headache there. If the headache be removed by correcting the error, the other causes, save under exceptional conditions, will never succeed in producing the pain.

It is important to remember that with this headache there is often associated nausea and even

vomiting. The so-called *bilious headache* of the "old school" is generally an ocular headache.

The intimate relation of the nerve-supply of the ocular muscles with the fifth nerve, and the association of the latter with the sympathetic and pneumogastric, explain the method of origin of the symptoms.

The ciliary muscle and the external eye muscles, when strained, demand an increased supply of blood, which in time leads to congestion. This in its turn will not be limited to the strained part, but will spread to other parts of the eye, causing the watery, red eye, already alluded to. The pain of "fatigue" is probably due to this congestion, and accounts for the tender eyeballs so common in migraine.

Liveing says that the nerve-storms in migraine have their point of departure or principal focus in the optic thalami, and that the normal course is from above downwards to the nuclei of the vagi, and from before backwards in the sensory tract, thus explaining the peculiar visual phenomena, such as *teichopsia*, and other symptoms of ophthalmic migraine.

Of course there are cases of true migraine, when the "point of departure" is from above downwards, but a very large percentage of cases labelled "migraine" are purely *ocular* in origin; and if the error of refraction or the want of muscular equilibrium is corrected, the symptoms disappear.

The connection of the fifth nerve with the sympathetic enables us to understand how the periph-

eral irritation of eyestrain can pass to the dura mater, pia mater, and sensory layer of the brain cortex.

Gower says: "If the sensory cells of the cortex (in which the cranial and intracranial sensitive structures are represented) are the most readily influenced of all the sensory cells, we can understand that headache should result from vascular repletion."* Everyone knows from experience that a headache increases sometimes to an alarming severity when the head is lowered—*i.e.*, when more blood flows to the head.

As we have already seen, although in eyestrain the peripheral irritation, if it takes the above form, is always, or almost always, present, it may not manifest itself as a headache unless there is a general increase of blood-pressure. If the blood-pressure be lowered by general treatment, the headache will disappear. But this does not mean that the headache was not ocular in origin. It simply means that one of the factors causing the pain has been removed, and the other, or others, are not sufficient to cause it.

It is therefore a good rule never to attempt to treat a headache as a migraine without previously eliminating eyestrain.

(b) *Peripheral Irritation unaccompanied by Pain.*—The chief types of this form are epileptic attacks and choreiform movements of the facial muscles.

It is important to remember that it is not the

* "Nervous System," vol. ii., p. 795.

error of refraction that causes the peripheral irritation, but the unconscious correction of the error by the patient. When the defect is great, no attempt is made to correct it, as the ciliary muscle can only correct low degrees of astigmatism ; hence there is no eyestrain.

It is only a cycloplegic that will reveal this unconscious correction ; hence no eyes can be reported as normal unless carefully examined under atropine or homatropine.

The removal of eyestrain does not, in the strict sense of the word, cure the epilepsy any more than it cures a headache ; but by removing the eyestrain we remove one of the causes, and frequently the only cause, that determines an attack. An epileptic attack and some forms of headache only differ in degree ; they are "nerve-storms."

The foregoing remarks apply equally to those so-called choreiform movements, tics, and habit-spasms which are so distressing to children and young adults, which take the form of spasmodic involuntary twitching of the facial muscles.

In all such cases it is most important to eliminate eyestrain. Such cases are purely functional, and it is not too much to say that a pair of suitable glasses will immediately alleviate, and not infrequently cause a complete cessation of, the symptoms.

Vertigo as a symptom of peripheral irritation may exist, but its commonest manifestation is in connection with diplopia.

Nausea and vomiting have already been men-

tioned in connection with ocular headache ; they may exist without headache, and apart from diplopia and vertigo, and may be due to eyestrain. Their presence is explained by the intimate association of the fifth nerve with the vagus.

3. NERVE-POWER WASTE—NERVE EXHAUSTION— NEURASTHENIA—BRAIN-FAG.

This is a manifestation of eyestrain that is as common as it is subtle. Subtle it must be, as every possible cause has been cited as the origin of the various groups of symptoms which are exhibited, except the eyes. It has not been sufficiently recognized that in a large majority of those cases called "neurasthenia" the real trouble is a constant "nerve-power" leakage or waste of nervous energy, and, in a large number of cases, eyestrain is the cause.

This nerve-power waste may exist in a person of robust nervous temperament without much, if any, harm ; but in one whose nervous conditions are unstable it must in time show itself.

A person with a low degree of astigmatism, or with anisometropia, or want of balance of the ocular muscles, during all his waking hours is sending down impulses to the eye to correct the defect ; and when he starts on near work, he starts with a big deficit, and further strain results. This must mean great waste of nervous energy.

Writers on neurasthenia agree that the ages when

the disease is most liable to show itself are between twenty and forty-nine, and these are exactly the ages when this form of eyestrain is most manifest.

Again, those most affected are almost invariably people who are engaged in constant near work, such as engine-fitters, post-office and bank clerks, teachers and journalists.

Insomnia is a very prominent symptom of eyestrain, and so a vicious circle is started, eyestrain producing, among other troubles, insomnia, and insomnia in its turn aggravating the patient's condition because the all-important restorative is wanting. The extremely depressing effect of insomnia is a matter of common knowledge, and the depression caused by it has led hundreds to suicide.

The physician who is called upon to treat a so-called "functional nerve disorder," and fails to eliminate the element of eyestrain, fails in his duty both to himself and to his patient, for there is no functional trouble that may not be due to eyestrain.

The depression attending nerve waste may lead to the alcoholic habit, and the irritability so often present in those suffering from functional nerve disorders often induces the sufferer to resort to sedatives, such as morphia.

If the nerve waste is arrested, the depression or irritability is removed, and the drug habit is more easily overcome.

Once we allow that this nerve waste may exist, we recognize that there seems to be no limit to the

various ailments which may, perhaps gradually, and often imperceptibly, follow.

Dyspepsia and constipation have both been put down to eyestrain, and I think not unreasonably.* The dyspepsia may be, as we have seen, a direct reflex irritation from the strained eyes through the pneumogastric, and it may also be the result of nerve waste; for if this nerve waste causes depression, and the due amount of nerve energy for digestion is not available, then the various digestive processes are interfered with, not only in the stomach, but throughout the digestive tract.

It has been suggested that eyestrain is present in patients suspected of being in the so-called pretuberculous stage, and that by removing the eyestrain the advent of tubercle may be prevented.

This is quite consistent with the above theories and with the experience of many.

The patient suffering from nerve waste has a low resisting power to all disease germs, and is in what one might call a "pre-germ stage," so that he is more liable to any infection than the normal person.

There can therefore be little doubt that prevention of eyestrain will take a very prominent place in the preventive medicine of the future.

The chief reason why the eyes are so seldom suspected of being the cause of neurasthenia, brain-fag, and the different varieties of functional nervous

* "Aberrant Dyspepsias," by Leonard Williams (*The Hospital*, December 12, 1908).

troubles, is that the majority of patients have either good sight, or they are already wearing glasses which only partially correct their refractive errors.

Our present treatment of eyestrain essentially consists in correcting even the smallest error and prescribing the correction to be worn constantly. When this is done, not only do the symptoms disappear, but the eye tends to become normal, as, when small, the error tends to disappear, so that eyestrain becomes *cured*. We thus see that a very distinct advance has been made since Donders asserted "a radical cure of 'asthenopia' was extinguished for ever" (page 274).

Eyestrain is a disease of the higher civilization. The plough-boy and the dairymaid do not suffer from it. We see cases of it at our hospitals; but the vast majority of those who suffer are from the highly developed, highly strung, well-to-do, and professional classes, and the boon conferred on these by proper treatment is incalculable.

In the whole range of medicine there is no disease we can cure more easily or with greater certainty, for we have almost brought the treatment down to an exact science, which exists in no other department of medicine.

How often a patient coming to us with a history of headache and malaise, with all desire for living gone, has, after treatment, returned in a couple of months, declaring that the joy of life had returned, and that he or she felt a new person!

In one of the cases Donders cites he ends by

saying: "He left me as one saved from destruction" (page 286).

Those of us who have been working at this subject during the last twenty years must recognize the enormous debt we owe to Donders, and if that great physiologist had done nothing else but point out this way for us, he deserves the finest monument ever erected to the memory of man !

EPILOGUE

EPILOGUE

I CANNOT conclude without asking those who have perused the foregoing pages to pause and consider the greatness of this master mind. Not a single one of Donders' essential teachings has been proved wrong; they have all stood the test of half a century! Of how many men born into this world can this be said?

Where our present teachings differ is only in the development along lines laid down by him—a development which he might have expected to be even greater than it is.

And, further, Donders has left a lesson to future generations of care, accuracy, and truth in the carrying out and recording of investigations, never exaggerating, and whenever in doubt boldly stating the fact; and the confidence in him thus engendered has undoubtedly not only helped workers who have followed, but it has given them a grand ideal to live up to.

Where shall be his monument? *Circumspice!*

Throughout the civilized world all those relieved by the correction of errors of refraction are living monuments to his undying fame, and generations yet unborn will live to bless his name.

