The refraction-changes dependent upon glycosuria / by George M. Gould.

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THE REFRACTION-CHANGES DEPENDENT UPON GLYCOSURIA.*

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THE majority of oculists have long but vaguely known that glycosuria produces changes in the refraction of the healthy eye, but no one seems to have gathered the facts to a focus or gleaned the lessons derivable from their study. When a perfect illustration of the condition came into my practice I was therefore astonished to find that the few reports of cases observed in the past were mostly badly reported, a portion probably incorrectly reported, and a series of eight absolutely irreconcilable with another series of eight. Moreover, the theories as to the mechanism of the refraction-changes were as numerous as vague, and as irreconcilable as the reports of the cases themselves. As I tried harder and harder, in my first studies of these cases and theories, to understand them and resolve the mysteries, I found myself always deeper in doubt and amazement. But when I faced a final charting of the cases, the problem seemed to me to be suddenly resolved. The report of my case is as follows:

Gould's Case.† Dr. M., a busy physician carrying on a large practice, had for many years

*Read before the Ophthalmic Section of the College of Physicians, Philadelphia, April 16, 1907.

†Hitherto unpublished report.

been a patient of mine, and by frequently repeated examinations I had kept close watch of his refraction-errors, because the least eyestrain in so severe a worker and student meant a vast deal for him. I may add that this patient also took a great deal of personal interest in the eyestrain problem, and had learned by bitter experience, subjective and objective, that certain theories as to the ocular origin of much systemic disease were true. He was, therefore, admirably fitted to help me correlate the facts to be described. Another condition to be noted is that the patient is now 58 years of age and myopic, so that the chance of error from incorrectly diagnosticated accommodation is quite out of the count.

In December, 1902, Dr. M. had been wearing

R. – Sph. 2.62 – Cyl. 0.75 ax. 35° = 20/20+
L. – Sph. 2.62 – Cyl. 1.25 ax. 167° = 20/20+
B. E. – Sph. 0.87 and Cyls. for near work, in bifocals.

This error of refraction, although frequently tested had not materially changed for many years. On the 18th of the month my patient appeared with clearly marked symptoms of eyestrain. I found a sudden increase of myopia, measured by

R. – Sph. 3.25 – Cyl. 0.75 ax. 25° = 20/20+
L. – Sph. 3.25 – Cyl. 1.50 ax. 166° = 20/20+
B. E. – Sph. 1.12 and cylinders for near.

There was also a noteworthy limitation of the range of accommodation. This increase of myopia in a man of 53, in one apparently healthy, although explainable, perhaps, in other ways, aroused my suspicions. Rigid questioning brought out the confession of several symptoms, which made me urge careful urine analysis. Quickly came the answer: "The urine is loaded with sugar."

The strictest dieting was immediately carried out, and within a few days every trace of sugar was eliminated from the urine. But then, as promptly, there was again eyestrain. A reexamination of the

refraction showed that the errors had returned almost exactly to the point of seven days before.

In the next four years the total of the myopic corrections increased slowly and more in the last year, when Dr. M.'s health began to show some instability. I warned in vain. The sequel proved that he had simply grown more careless as to his food, eating sweets and starches with thoughtless indifference. In December, 1906, the errors were demonstrated to be

R. – Sph. 2.87 – Cyl. 0.87 ax. 25° = 20/20+
L. – Sph. 3.00 – Cyl. 1.50 ax. 172° = 20/20+
B. E. – Sph. 0.50 and cylinders for near.

By February 18, 1907, symptoms of ill-health began to grow manifest, even to the careless-of-self mind of the patient, and he resumed the long-neglected analyses of the urine. The first one showed again an extremely high percentage of sugar. He was also having eyestrain, ignored for the time being, in his interest in other things. Of course the rigid diet was again put into force, and in two or three days not a sign of glycosuria could be detected. Then the patient returned to me with greater eyestrain than ever, and again there was the astonishing reduction in myopia measured by

R. – Sph. 2.00 – Cyl. 0.62 ax. 20° = 20/20 +
L. – Sph. 1.87 – Cyl. 1.37 ax. 170° = 20/20+ } Distance
R. Cyl. alone
L. + Sph. 0.12 and cylinder } Near

These corrections brought perfect visual acuity for distance and near, absence of all eyestrain, and the ocular conditions, with unessential changes, will probably remain the same so long as there is no glycosuria.

Grimsdale's Case,[1] of a woman aged 45, who came October 7, 1897, wearing for some years, for near vision,

R. + Sph. 1.00 + Cyl. 0.75 ax. 160°
L. + Sph. 1.75

Fourteen days previously the woman had noticed that her refraction had suddenly changed, so that her reading glasses were no longer needed, and distant objects were not seen so clearly as formerly. Vision, natural, was 6/60 in each eye. Without mydriasis the errors were estimated as follows:

R. −Sph. 2.00 −Cyl. 0.50 ax. 180° = 20/20
L. −Sph. 2.00 = 20/20

With this correction the patient at once said, "That is how I used to see." No mydriatic was used, nor was the range of accommodation tested. The right lens had scattered central opacities, the left was clear. At this time there were 26 grains to the ounce of sugar in the urine. Treatment was now instituted, and in ten days the report of the oculist was

R. 6/18 −0.5 = 6/12, not improved by cylinder
L. 6/6 No Hm

"She now required + Sph. 2.50 to enable her to read 0.3 Sn. at one foot." "She was on strict diet, and the total amount of sugar had much diminished." "The right lens presented considerable irregular astigmatism." In a few weeks death occurred following diabetic coma.

De Schweinitz's Second Case was that of a girl 20 years old, with no organic lesions except congenital posterior capsular opacity in each eye. Under cycloplegia the errors were determined

R. +Sph. 0.25 −Cyl. 0.75 ax. 135° = 6/10
L. −Cyl. 0.62 ax. 15° = 6/7 ½

Six months later the patient returned, complaining of rapidly deteriorating vision following some prolonged illness. It was learned that diabetic symptoms came on shortly after the first visit, and that large quantities of sugar had continued in the urine ever since, despite treatment. No mydriatic was now used, one judges, but a myopia of 3.00 D. in the

right eye, and 2.00 D. in the left was demonstrated, with vision of 6/22 and 6/12 respectively. Systemic treatment was continued, and in three months the visual acuteness was holding its own. But edematous choroid and vitreous opacities had now become manifest. In another month the myopia of the right remained the same, but that of the left eye had become 5. The patient died not long after this. Dr. de Schweinitz concludes that a diffuse edematous affection of the choroid is the underlying pathological process in such cases.

Appenzeller's Case (Graefe-Saemisch) was of a patient 43 years old, who had 1.00 D. myopia while the glycosuria existed, but when under treatment the urine was normal emmetropia again returned.

Hirschberg's Case.[2]—Hirschberg gives a brief résumé of the case of a man 48 years old, who had for many years seen plainly with — Sph. 9″, *i.e.* — Sph. 4.62 D., but who now complained of dimness of vision. With his old lenses he could not read any longer. There was no paralysis of the accommodation. The proper correcting lenses were now found to be — 8″ Sph., — Cyl. 40″ ax. 180°. Diabetes had been present for 14 days, followed by some (doubtful) loss of weight. The eye-grounds were normal. The urine contained 6¼ per cent. of sugar.

Dujardin's Case[3] was of a woman 69 years of age, applying April, 1899. There was high glycosuria, and despite treatment the percentage of sugar remained between 70 and 80 grams per liter. The media were clear and the eyes healthy. She could no longer see well at a distance, although formerly she had had sharp acuteness. For reading she had required + Sph. 4. D., but could not now read with these lenses. The pupils would not widen under atropin. By retinoscopy about 5. D. of myopia was

made out. Homatropin was ordered as a collyrium and belladonna given internally on the theory that a spasmodic condition of the ciliary muscle existed. A month later conditions remained the same.

Risley's First Case.[4]—A woman of 49, having had glycosuria in the past, complained that, with the suppression of the glycosuria, and consequent improvement in general health, her vision had grown rapidly worse. She was wearing + Sph. 2.75. The dimmed vision had been observed only within a few days. Under mydriasis the errors were

R. +Sph. 5.00 +Cyl. 1.25 ax. 75° = 20/20
L. +Sph. 6.00 +Cyl. 0.75 ax. 105° = 20/20

In ten days glycosuria returned, and with it dimness of vision again, and the correction (nonmydriatic, one gathers) was

R. +Sph. 3.00 +Cyl. 1.25 ax. 75° = 20/20
L. +Sph. 3.50 +Cyl. 0.75 ax. 105° = 20/20

Fifteen days after this she chose (nonmydriatic, probably)

R. +Sph. 1.25 +Cyl. 1.25 ax. 75° = 20/20
L. +Sph 1.50 +Cyl. 0.75 ax. 105° = 20/20

Risley's Second Case.[4]—A man, 74, suffering from saccharin diabetes for at least six years, was the subject. In August, 1896, the errors were

R. +Sph. 1.50 +Cyl. 1.25 ax. 180° = 6/7 1/2
L. +Sph. 1.25 +Cyl. 1.25 ax. 180° = 6/10

The lenses were cataractous. In April, 1897, the vision in each eye remained the same, presumably, with the same errors of refraction. Fourteen days later dimness of vision was complained of, and the visual acuteness with his distance-glasses was with each eye 6/30. No sugar, or but a trace, was present, and the man now selected

R. +Sph. 2.50 +Cyl. 1.25 ax. 180° = 6/7 1/2
L. +Sph. 2.00 +Cyl. 1.25 ax. 180° = 6/10

Within three days of a month later the patient had

been compelled to return to his old glasses within a few days, and he now chose this correction, the first above given, with almost the same visual acuteness. Sugar was again found—4.20 per cent., and a specific gravity of the urine of 1,027.

*Carpenter's Case** was of a woman, 51 years old; consultation was for dimness in reading during last six months. There was no local ocular disease. Refraction (without a mydriatic) was diagnosed

R. −Sph. 0.25 −Cyl. 0.50 ax. 90° = 20/20
L. −Cyl. 0.50 ax. 90° = 20/20
+Sph. 1.75 added for reading. Reading glasses only ordered.

Six years later the patient returned, stating that her reading glasses had proved comfortable until within a few weeks. Her correction was now found to be

R. +Sph. 1.50 = 20/20
L. +Sph. 2.50 = 20/20
} Distance
With + Sph. 2.25 added for reading
} Bifocals ordered.

Four weeks before this last visit glycosuria had been diagnosed by her physician, and treatment begun, with great decrease of the percentage of sugar. Still no local disease of the eyes was found. In three weeks she returned to her original careless diet (rich food, sugar, etc.), and convex lenses made vision worse, but R. — Sph. 0.75, L. — Sph. 0.50 again brought the distant acuteness to normal.

De Schweinitz's First Case[5] was of a patient, a man of 47. The man had fairly healthy eyes, but the report does not state whether the first refraction was made under cycloplegia, or not. "Each eye was hyperopic, + 1.25 D. The complaint was of failing vision in presbyopia. One gathers that glasses were ordered for presbyopia only. Four years later the complaint was of dimness of vision for distance, "scarcely one-half" what it had been four years previously, *i.e.* normal, and — Sph. 0.50 was now re-

*Not published. MS. report kindly loaned by Dr. Carpenter of Philadelphia.

quired to give normal acuteness. Glycosuria was suspected and demonstrated. By June 14, 1895 (a month later) sugar had been extinguished. Although the static refraction does not seem to have been determined—a matter of regret—the inference is that the change of refraction from + Sph. 1.25 to — Sph. 0.50 was due to the glycosuria. As there was no decided lesion to be discovered by the ophthalmoscope, etc., the inference seems justified, and another case of displacement of the focus forward is to be added to the list.

Neuberger's First Case.[6]—A 50-year-old woman who had been emmetropic and able to read the finest print with + Sph. 2.00, came four months later, reduced in flesh and with a myopia of R. 1.5 D., L. 2.00 D. Smallest print could now be read with + Sph. 1.5 D, and L. + 1.00 D. Weakness of the accommodation was also present. The lenses were, and remained clear. The urine contained 3.5 per cent. of sugar. In about three weeks emmetropia returned and + Sph. 2.5 D. was required to read fine print. The myopia had thus disappeared, *although at this time the glycosuria had increased to 7 per cent.* Diabetic retinitis later appeared, but the lenses continued clear and the refraction emmetropic.

Neuberger's Second Case[6] was of a woman of 48, who two years before had a hyperopia of 0.75 (+ 1.5 for near), but now was myopic, R. 1., L. 2.5 D., and no glass for reading. The lenses were clear at first, but later became obscure. Six months later the myopia was R. 3.00, L. 8.00 D., with cataract progressing.

Alexander's Case.[7]—The patient was a man of 57 years of age, who had felt a decrease of visual acuteness for distance during the last 14 days. A myopia of 1.00 was diagnosed. For near + Sph. 0.75 was

sufficient when 1.75 was before demanded. The sugar was 5.7 per cent. In 8 days dieting brought the urine to normality, emmetropia returned, but this in 5 days changed to a hyperopia of 1.75 D., the urine continuing free from sugar. In epitomizing this history Groenouw* rightly says the original refraction was probably hyperopia, which later became manifest.

Groenouw's Case.[8]—A woman of 55, suffering from diabetes, suddenly acquired a myopia of 1. D., which in 3 weeks became emmetropia. The lenses were clear. This is given by Groenouw as a case of latent hyperopia becoming manifest.

Doyne's Case† was of a physician of 40 who consulted the oculist for failing sight. The man was suffering from acute diabetes. Three diopters of hyperopia were found, and this correction was ordered and the glasses were worn without discomfort. Practice was discontinued and a sea voyage undertaken, but upon his return he complained of dimness of vision for distance. The hyperopia had lessened and + Sph. 2.50 each eye again gave normal vision. Soon afterwards dimness of distant objects again recurred and the strength of the glasses had again to be reduced, "and later on, when the sugar disappeared, under homatropin freely used, only 0.75 of hypermetropia existed."

Horner's Case[9] was of a woman of 55. The hyperopia had rapidly increased; it was 1/14″ at the time of the visit, but with improvement in the general health it sank to 1/48″.

Cohn's Case (Graefe-Saemisch) was one of increase of the hyperopia in a 68-year-old woman, from 1.50 to 3.00 D.

*Reported in the discussion of Mr. Grimsdale's case.[1]

†Augenleiden bei Diabetes Mellitus, Groenouw, 1907, p. 46.

Landolt's Case.[10]—"We have observed, among others, a most interesting case of this kind. A lady had a fall and became diabetic as a consequence of this traumatism, although her general condition was not much affected by it. At the same time she commenced to no longer see well at a distance, and found the spectacles, that she had previously worn on account of her presbyopia, to be insufficient. The refraction could be determined, in this case, with the utmost accuracy, not only because the patient was very intelligent, but because she had reached an age at which the accommodation is almost *nil.* I made out a hyperopia of 0.5 D., which certainly had not previously existed. It increased and then diminished, according to the amount of sugar excreted, and finally disappeared entirely. So that, when this traumatic diabetes was cured, the patient could dispense with her distance-spectacles, and substitute, when reading, her former spectacles for the stronger glasses which she had been obliged to resort to. I have seen the patient for several years, and it has been extremely interesting to note that each little recurrence of diabetes has announcd itself at once by a diminution of refraction, to such a point that the curve of hyperopia was, so to say, parallel with that of the quantity of sugar eliminated with the urine."

Gallus' Case.[11]—A man aged 51, under treatment for diabetes for two weeks, noticed impairment of vision for distance. Sph. + 2.00 for reading had become too weak, although he "could see well through them into distance." Examination revealed R. + Sph. 1.50 + Cyl. 0.50 ax. 180° = (What?) L. + Sph. 2.00 + Cyl. 0.50 ax. 180° = (What?) With + 2. added he could read the smallest type. Under treatment glycosuria ceased, and in two months the hyperopia had subsided so that the patient needed

only cylinders and could read again with + Sph. 2.00 + Cy. 0.50.

Lichtenstein's Case[12] occurred in a man seventeen years old. The refraction is given as + Sph. 1.50, estimated without a mydriatic, but 12. D. + Sph. lenses were required to enable the patient to read from print at 9 cm. The paralysis of the accommodation is said to have been "complete." He had suffered from no diseases (except glycosuria) which would cause this paralysis. Despite treatment his glycosuria had increased in severity, and urinalysis now showed 4.5 per cent. of sugar present, and some six liters of urine were voided daily. The patient was sent to a colleague, and the next day the hyperopia was found to be 2.5 D., with 13 D. lenses required for near. Homatropin now showed the same 2.5 D. of hyperopia. Five days later it was 3.5 D., demonstrated by atropinization and skiascopy. For 14 days the conditions remained the same, but the young man felt better. Now 3.5 D. was required for distance, but only 11 D. for near. But this was soon reduced to 6 D., and in about two weeks from the first visit it had fallen to 5 D. The hyperopia gradually fell from 3 D. to 2.5 D., then to 2 D., and finally to 1.5 D., the point whence it started on March 8, 1906. Lichtenstein says the doubt as to the latency of hyperopia was excluded by his atropinization. L. explains that in this case two factors united to produce the result, paralysis of the accommodation, and transitory hyperopia. He explains this as due to loss of water by the contents of the globe and a resultant shortening of the anteroposterior axis.

Sourdille's Case.—In an emmetropic woman, 53 years old, S. observed a hyperopia of 2.00 D. occur, which again disappeared with the disappearance of sugar.

Lundsgaard's Case.[13]—The patient was a woman, who in 1892 was found to have + Sph. 0.50; "the ophthalmoscope showed emmetropia." In the summer of 1905 there was great thirst and polyuria, but according to the general physician no sugar or albumin existed in the urine. In 1906 traces of sugar appeared, and in a month the percentage was 7¾. Dieting reduced the sugar somewhat, but sudden dimness of vision called attention to the eyes when a hyperopia of 2.50 and 2.00 was found. The media were clear. Accommodation accorded with the age. The percentage of sugar was now reduced but not extinguished, and the hyperopia fell; finally the sugar was eliminated entirely and on the 19th of November, 1906, the hyperopia returned to the figure of 1902.

These twenty-two cases are not all those which, by more rigid search or less rigid rules of exclusion, might possibly be included as data.* If we arrange them in three series we find the first, composed of those in which myopia is increased by glycosuria (or decreased by its extinction) is made up of six—those of Gould, Grimsdale, de Schweinitz's Second, Appenzeller, Hirschberg, Dujardin.

The second series, those in which hyperopia is decreased by glycosuria (or increased by a return to normality) is made up of eight—those of Risley's Two, Carpenter, de Schweinitz's First, Neuberger's Two, Alexander, and Groenouw.

The third series, those in which hyperopia is reported as increased by glycosuria, is composed of eight—Doyne, Horner, Cohn, Landolt, Gallus, Lichtenstein, Sourdille, Lundsgaard.

Principles Governing the Determination of the Refractive Conditions.—It is of first importance that

*Kako's case of developing + astigmatism, *e.g.* is omitted for evident reasons.

in the report of a case the precedent static refraction must be the basis of any comparison. In all persons under 60 years of age, except occasionally in myopia, the accommodation, unless paralyzed, would make a possible error rendering all comparisons inaccurate, and if under 50 years almost wholly untrustworthy. The reports of glycosuric refraction change in hyperopes under 50, unless the diagnostic tests have each been made under cycloplegia, are of little value, except that the physician believed and reported his belief that the presence or absence of sugar in a general way indicated certain refraction changes. If the presbyope is highly myopic that gives an added element of accuracy. Still greater precision is to be predicated in cases in which previous accurate refractions have been made frequently and over a long period of years. The astigmatic error, once correctly determined, may be discarded in speaking subsequently of the cases because this changes but slightly in cases of paralyzed accommodation, so that the statement of the relative myopia and hyperopia is practically all that is necessary. In no case so far reported has there been any exact mathematical relationship stated between the varying amounts of the sugar and the degrees of the resultant refraction-change. This valuable aid must therefore be left out of the count. In the reports of future cases it should be made out and reported upon. The absence of local ocular disease that would obscure the nature of the refraction changes must also be insured. When glycosuria has set up extensive choroidoretinitis, pronounced cataract, etc., a new factor is added which may be the cause of so much doubt that the report is made suspicious. The tests, to be of the best value, must be in cases with acute glycosuria, in which the eyes are so far healthy, the media sufficiently clear, the

funduses so nearly normal, the acuteness of vision so good, etc., that the measure of refraction is not in doubt. The shorter the period of time between the tests of the glycosuric and the nonglycosuric refraction the more valuable will be the data obtained, and the more convincing the deductions made from them. Lastly, the oculist must be a refractionist, believing in the value of accurate refraction tests, habituated to make them, and seriously aware of the evil results of bad and slipshod refraction to eye and general system. There is little or no possibility of securing a trustworthy estimate of the refraction in all Europe, where the refraction is pronounced alike in both eyes, astigmatism ignored, etc., and whenever a report comes to us wherein the refraction has been estimated with the ophthalmoscope, and bearing ludicrous internal evidences of bungling, and contradicting the reports of careful and keen-witted refractionists, there may be no delay in speedily setting it aside as worthless, or of little scientific value.

Exclusion of reports according to the foregoing principles becomes easier when we notice that there is no reported case of myopia in which glycosuria has not had the effect of increasing the myopia—that is, of bringing the focus of the dioptric system forward. All such cases are logical, one may say, as naturally the effect of glycosuria must be to affect the focus in that way. Whatever be the mechanism intermediating the change it is impossible to comprehend how glycosuria can displace the focus posteriorly. Now, as the myopia of an eye is far more easily and accurately measured than the hyperopia, and without a cycloplegic, it is not surprising that all observers, good or bad, unite, in their reports, that glycosuria, if it changes the refraction at all, increases the preexisting myopia.

Confirming this result we find that the reports of eight cases of change in hyperopia also say that the effect of glycosuria is to advance the focus exactly as happens in myopia. And in this series occur the names of such trustworthy refractionists as Risley, Carpenter, de Schweinitz, etc.

It is simply inconceivable that the mere accidents of the location of the retina in the path of the refracted cone of light-rays should have the reverse effect in displacing the focus in hyperopia, from what it does in myopia. Therefore when eight reporters find such an illogical if not impossible reversal of the natural consequences as testified to by fourteen, it behooves us to doubt the accuracy of the oculists' tests and reports rather than to indulge a belief in the inherently improbable and impossible. Let us briefly glance at the cases critically of those reporting increase of hyperopia:

In Doyle's case, the patient was 40, no mydriatic was used at first and as the only true basis of comparison, and the squint of the corner of the mouth becomes visible when it is said that three diopters of hyperopia were found! The case was pretty plainly one of latent hyperopia, astigmatism, etc., becoming manifest "later on," because of the frequent effect of glycosuria on the accommodation, or because of advancing presbyopia. It should therefore be set aside. No wonder that Mr. Doyle had no explanation to offer.

Graefe and Saemisch explain Horner's case and that of Cohn as due to latent hyperopia. Schmidt-Rimpler, and Groenouw also explain similar cases in this way.

In Landolt's case there is too much vagueness in the report and too little increase of the hyperopia —only 0.50 D.—to make us heed the claim of "utmost accuracy," made in behalf of the improbable.

The patient of Gallus was 51, the acuteness of vision was not noted, no mydriatic was used, etc. The change was probably in the accommodation, not in the (untested) static refraction.

Of Lichtenstein's case one doesn't know what to say, except that the inherent absurdity of 2.5 D. hyperopia with 13. D. required for near vision is so great as to make us smile with incredulity. Then it must not be forgotten that atropin in Dujardin's case did not even widen the pupil. Until competent refractionists report other cases similar to this one it must remain as a single and anomalous empiric fact in which other factors than glycosuria were present. This is the only case reported of one so young as 17 years, a fact to be remembered.

Sourdille's case was in a patient 53 years of age, and no mydriatic was used. *Exit!*

In Lundsgaard's case, "the ophthalmoscope showed emmetropia," and with that admission "the defence may rest its case." (In speaking of his own case Schmidt-Rimpler (*Die Erkrankungen des Auges, etc.*, Wien, 1905), says, "the upright image demonstrated a hyperopia of 0.5 D.," so that case may also go to the Jury forthwith.)

We may therefore feel no compunctions in excluding on the ground of erroneous diagnosis due to failure to estimate correctly the static refraction almost all of the cases so far reported in which it is claimed that glycosuria removes the focus of the dioptric system to a position posterior to that occupied in the previous nonglycosuric period.

It would require an entire paper and a long one, to enumerate and critically judge of the theories advanced to explain the modus operandi of the effect of glycosuria upon the refraction. I doubt if the most capable mind could do much toward clarifying the obscure subject. I had made a list of these

numerous theories, but I do not think it would be edifying to read it. The arguments point toward a consensus of opinion favoring increased density of the ocular fluids, functional disturbances and paralysis of the lens and ciliary muscle, etc., rather than to changes in the corneal curves, changes in volume of the contents of the globe, or displacement of the macular region of the retina.

As to the significance of the phenomenon, everybody has emphasized the importance of the recognition of the refraction and accommodation changes as warnings of the existence of the systemic disease. In the days of life insurance examinations, of routine urinalyses by the general practitioner in almost all cases of ill-health, of the striking evidences to the patient of thirst, polyuria, etc., the warnings seem somewhat antiquarian, at least not of the first importance. The wideawake American oculist would prefer to doubly emphasize the overlooked truths: (1) That the eyestrain preceding the glycosuric refraction-change may have been a prime factor in producing the functional dietetic and nutritional disease called glycosuria; most significant is the fact that the great majority of the cases occur during the presbyopic period; (2) that the secondary refraction-change serves as a perfect illustration of the increase of the diseases due to overstrain by the proverbial vicious circle, increasing the irritation and nutritional abnormalism by the secondarily induced refractional changes caused by the glycosuria; (3) the necessity of preventing quick-following ocular disease, both functional and organic, by heeding the accurate warnings given by the refraction changes consequent upon early and curable glycosuria. But whatever the point of view, and whatever the injuries done or threatened, the accurate diagnosis of the static refraction

overtops and conditions every measure of prevention and every step of progress either in science or in therapeutics.

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