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GEORGE M. GOULD, M.D.

OPHTHALMOLOGIST TO THE PHILADELPHIA HOSPITAL; CHIEF OF THE OPHTHALMIC CLINIC OF THE GERMAN HOSPITAL, PHILADELPHIA

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THE etiology of the peculiar form of choroidal disease called central choroiditis seems, by common consent, to be a mystery. Hutchinson classifies all choroidites into twelve groups, of which the third and fourth alone concern us in the present inquiry. His third class is "Senile Central Choroiditis, or Tay's Choroiditis, always central, and met with only in those past middle age"; the fourth, or "Hereditary Choroiditis, various in form, often beginning in childhood, but sometimes not till middle life; several members of the same family are affected, and the changes are usually aggressive. Tay's choroiditis may sometimes occur as a family malady." I have selected from my records the clinical details of the following eight cases to illustrate my theory that central choroiditis is due to ametropia. As they are neither senile nor hereditary they do not fall into Hutchinson's classification.

CASE I.—N. J., a young man aged twenty-five, otherwise in excellent health, complained of growing inability to read or write, except for but a minute or two at a time, and by a strong light. He could not read a line at night or in a dark day. There was no ocular or cerebral pain, though the irritative and reflex symptoms usually accompanying eye-strain had in previous years been severe. From this and other symptoms I concluded that his present myopic refraction had but recently come on, and that in the beginning of his retinal failure he had been a hyperope. His acuteness of vision was but about $\frac{20}{200}$, and not improved by correction of the

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ametropia. The maculæ, especially above, were surrounded by nearly concentric strata-like clouds of dark-brown pigment patches, extending about 15° outward. Both eyes were about equally affected. The family and personal histories were excellent, hereditary disease and syphilis being almost certainly excluded. He was a civil engineer by profession, and had been a hard student. I advised him to give up his profession and take to farming or some similar occupation. He is now trying ranch-life in Colorado, and vision does not now, as formerly, seem to be deteriorating.

Case 2.—Dr. G. J., aged twenty-eight, complained only of slight asthenopic symptoms, and that the eyes soon tired with near work, especially by artificial light. A hasty ophthalmoscopic examination showed nothing more than compound hyperopic astigmatism, and this seemed to explain the symptoms and dictate the treatment. I was accordingly surprised to find that the most careful refraction brought out only a doubtful $\frac{20}{50}$ vision. A more careful ophthalmoscopic search solved the riddle, as I believe it would often do in many cases of "amblyopia." Both maculæ were clouded and surrounded by a tawny brownish stippling, differing in character entirely from the pigmentation of the rest of the fundus, and plainly pathological. The family and personal history was above reproach. His refractive error—mixed and hyperopic astigmatism of 1.00 D at unusual axes—was at once corrected and all possible avoidance of near work ordered. In addition to his professional studies, his specialty had long demanded much continuous ocular labor of an exacting nature.

Case 3.—Miss C., aged twenty-two, of healthy parents and history, complained that her eyes tired quickly with near work, and especially in the evening, and also of the usual symptoms of hyperopia. Normal vision—R $\frac{20}{40}$, L $\frac{20}{30}$. Under a mydriatic I found a simple hyperopia of 4.00 and 3.50 D respectively. With these lenses the vision with strong light was with either eye doubtful $\frac{20}{20}$, but with the least weakening of the light the acuity of the right eye quickly fell. The pathological stippling or pigmentation about the macula of this eye was so slight that unless one looked for it carefully, and knew about what to look for, it would easily be missed. The left is normal in this respect. The degenerative process was in this patient in its earliest stages, and it will be of interest to watch the future developments, whether the glasses prevent further deterioration, etc.

Case 4.—Mrs. K. B., aged nineteen, complained of headache and asthenopic symptoms, quickly made worse by near work. Normal vision, R $\frac{20}{30}$ (?), L $\frac{20}{50}$. The refraction, under a mydriatic, showed R + sph. 0.75 D, ⌒ + cyl. 0.50 D, ax. 75°; L + sph. 0.50 D, ⌒ + cyl. 0.75 D, ax. 105°. But with these lenses the acuity of the left eye is still only $\frac{20}{50}$; that of the right being $\frac{20}{20}$. While looking at the $\frac{20}{50}$ line, one letter of the line is not seen at all, whilst the letter at either side is readily detected. The pigmentary changes about the left macula were marked, the stippling had often become confluent, and formed coarse brown splotches in some places, and there was an unusual lack of symmetry or concentric character in the grouping around the macula. No such changes were recognizable in the other eye. Family and personal history showed no disease or diathesis.

Case 5.—Miss P. N., a seamstress, aged forty-three, complained of tired eyes after work. Normal vision, R $\frac{20}{20}$, L $\frac{20}{40}$. The refractive error of hyperopia, one dioptry, having been corrected, the right eye regained perfect acuity, but the left was improved only a very little. She had sewed every day for many years; had had good health all her life, and her parents had enjoyed good eyesight and health up to old age. The left macula region was typically pigmented, the right normal. There is also in the left papilla a pretty sharply delimited bundle of atrophic fibres, upon the temporal side, having a dead bluish-white appearance, and comprising perhaps one fifth of the total papilla space, and at a position corresponding to that of the gathering of the macula fibres.

Case 6.—Mrs. P. L., aged thirty-two, used formerly to be a great reader at night, and by a common oil lamp, after sewing much of the day. The eyes finally began to tire, until of late she can read but a little while at a time, except by daylight. At night the eyes tire, and the image fades after working or reading a few minutes. Vision, without a mydriatic: R $\frac{20}{50}$, L cannot count fingers; with a mydriatic; R $\frac{20}{30}$ + sph. 0.62 D, ⌒ + cyl. 0.25 D, ax. 90° = $\frac{20}{20}$; L + sph. 2.50 ⌒ + cyl. 1.00 D, ax. 90° = $\frac{10}{200}$. The ophthalmoscope reveals an advanced and extensive pathological pigmentation of the left macula region, extending in this case farther toward the equator of the eye than I had ever seen it. The stippling of the right was limited, and delicate in the extreme.

Case 7.—Wm. J., aged thirteen, has had headaches for several years, and made worse by reading. He is an ambitious, bright-

minded school-boy, and has studied hard both at school and at home at nights, until of late he at once gets sleepy when he tries to study by artificial light. The refraction and the ophthalmoscope explain both symptoms. With paralyzed accommodation I find both eyes alike in acuity, refraction, and a typical incipient ametropic chorido-retinitis. Refraction : + sph. 1.75 D, ⌒ + cyl. 3.00 D, ax. 90° = $\frac{20}{30}$.

Case 8.—Miss A. L., aged twenty-five, has for five years been wearing Sph. — 1.75 from a prominent advertising optician of this city. She has compound hyperopic astigmatism, and has suffered much from the artificial hyperopia and retinal injury directly produced by this "scientific" optician. The proper correction of her ametropia gives her a visual acuity of $\frac{20}{50}$ and $\frac{20}{30}$. Both maculæ show the typical pigmentary degenerations—the right the more pronounced.

The emphatic teachings of these and other cases that have come under my care are: (1) the absence of all systemic, toxic, or hereditary conditions that might be or that are commonly adduced in a vague sort of way to account for the ocular lesion; (2) the existence of an irritating ametropia that threw a blurred or abnormal image upon the macula; (3) the exaggeration of the eye-strain by long-continued and severe ocular labor at near-range.

As to senile central choroiditis, since I first recognized the connection of ametropia and the choroido-retinal lesion, I have had but few cases of central choroiditis in the old, but in these there also coexisted a considerable refractive error, and of course with presbyopia—also uncorrected. It is plain that presbyopia, if uncorrected or improperly corrected, even without other refractive error, would induce the condition that seems to me to play the chief etiological rôle—*i. e.*, an habitually imperfectly formed retinal image. Therefore, central choroiditis may be senile, the result of presbyopia, and still be due to precisely the same essential cause as in adults and the young.

Concerning the existence of the lesion in the young, I have never seen a case of central choroiditis in children that showed any thing but the slightest stippling. I have no doubt I have passed by many such however. Our

school systems must produce them more frequently than supposed, but in such cases the difficulty of detecting such a lesion is greatly increased. At best the defect is difficult to examine, is overlooked because unsuspected, and is so easily classed as amblyopia. In cities where schools and high-pressure force the young and ametropic eye to an excess of work, early visual troubles bring the child into the oculist's hands, and his prescription stops the growth of the macular disease. That chronic eye-strain *produces* lessened acuity is a truism of ophthalmic practice ; an undiscovered pigment-change at the macula would and does *explain* it. But in this connection I cannot forbear availing myself of another's work, and though he is an unconscious and perhaps unwilling witness, his conclusions seem powerful testimonials in behalf of the theory above suggested. I allude to the article by Edgar A. Browne on " Central Choroiditis, with Slight Cerebral Symptoms in Children," in the *Royal London Ophthalmic Hospital Reports*, January, 1889. The argument there advanced is that these cases are connected with and due to slight attacks of meningitis or other cerebral disease, " just as similar symptoms are allied with optic neuritis." This seems a wholly gratuitous, if not forced, assumption, and a non-convincing analogy. Since Deutschman's investigations, we can understand how coarse cerebral disease may induce papillitis, but one can only wonder how a slight and passing meningeal hyperæmia could produce intimate and localized choroido-retinal degeneration. The symptoms of these cases, as given by Mr. Browne, are night-terrors, gastric derangements (vomiting, loss of appetite, etc.), constipation, choreic movements, etc. But surely every ophthalmic surgeon knows these are the commonest symptoms of simple uncomplicated eye-strain in the young. Apply proper spectacles to such patients, and the night-terrors, nausea, nervousness, anorexia, and headache disappear as if by magic. This is not hobby-riding, or mistaken enthusiasm, or *post hoc—propter hoc* logic, but undeniable fact. According to my theory and belief, the " slight attacks of meningitis," are the common reflex-neuroses of eye-strain, and the choroido-retinal lesion the result of an

abnormal stimulus,—both sets of facts the consequence of a single cause—ametropia.

Without any violence to the facts, I think the existence of this peculiar choroido-retinal lesion, whether in the young, the middle-aged, or the old, whether hereditary or not, may be correlated and ranged under the one etiological factor. The existence of any considerable number of cases, that have arisen without ametropia, would invalidate the theory. I have never seen a single instance. Neither have I, since the theory took shape in my own mind, ever seen a case of long-standing uncorrected hyperopia or astigmatism too great for the ciliary muscle to neutralize, that did not show the pathological pigmentation and the subnormal acuity,—subnormal at least by weak light in the early stages, little or not at all improvable by lenses, in the later stages. It may be said that entirely too few illustrations exist as a supposed result from a widely acting cause. Ametropia is immensely common, central choroiditis very rare. To this it may be replied that we have no idea how common the early stages of central choroidal disease may be. It may be doubted if oculists examine the macular region of all their patients with great accuracy, and the blanket-name for a mystery, amblyopia, doubtless covers a multitude of negligences. The location of the lesion also points to some causal relation with the act of habitual vision. It must also not be forgotten that the strain of civilization upon the naturally hyperopic eye is not yet on to the full. It remains to be seen whether that eye will remain so resistant in the future as in the short past in which it has been tested. Moreover, we must remember that change of occupation constantly sifts out those eyes that cannot withstand the strain of near work, and sets them easier tasks; that the growing practice of applying spectacles to the young checks the retinal deterioration; and lastly, that the varying resisting power of retinæ, like that of other tissues, produces exceptional and apparently unaccountable immunity in withstanding the attack. It is to be supposed that the brunt of such an attack is always borne by the ciliary muscle. The Müller ring-fibres are the response to the

excessive stimulus. With continuous near work and uncorrected ametropia this resource fails, and the retina is then subjected to an abnormal and pathogenic stimulus. Central choroiditis, with its resultant atrophy of the nerve-fibres and subnormal visual acuity, is the reaction and result of this unaccustomed and unnatural stimulation. It is but one illustration of the universal physiological law, that functional reaction to new stimuli develops modification of structure, and if the new stimulus is so abnormal that physiological reaction and adaptation cannot take place to resist and preserve, then the organ must suffer, and even atrophy. Were it not for the beneficent work of spectacles, it might reasonably be doubted if civilization would not, in part at least, bring itself to an ending in the results of severe eye-strain ; that is, in myopia, ocular and nervous disease, headache, chorea, etc., etc., and, more important still, in the mal-assimilation that results from reflex derangements of the digestive system, primarily caused by eye-strain.[1]

In order more clearly to bring out the characteristic features of my conception, let me briefly epitomize the different aspects of the disease :

NOMENCLATURE. — Though the lesion, like so many others covered with the indicative suffix—*itis*, is evidently extremely chronic, and in no true sense inflammatory, it is plain that the habitual terminal, *itis*, must be allowed. Again, every thing indicates that the lesion is retinal, and not choroidal. At the best we may qualify it as a choroido-retinitis. Since it is known that the pigmentary layer, both functionally and evolutionally, belongs to the retina, and not to the choroid, the error of calling these and certain other fundus changes, choroidal, becomes all the more apparent. But, under protest, the habit must be allowed to obtain. According to the theory, these macula-changes may be due to any one, or to any combination, of four factors—uncorrected hyperopia, astigmatism, presbyopia, and insufficiency. Either of these conditions, not corrected by the excessive

[1] See, for example, my article in the January, 1890 number of the *American Journal of Medical Sciences*.

action and neutralizing power of the ciliary muscle, nor by spectacles, supplies the retina wlth an abnormal stimulus. We cannot say the choroido-retinitis is due to eye-strain, since so long as the eye-strain is sufficient the dioptric mechanism supplies a proper image. It is when the task is too great or too continuous, and the ciliary muscle strikes work, throwing the labor upon the retina and the cerebral mechanism, that the retinal degeneration begins. Hence, the suggestion that the usual reflex consequences of pure eye-strain are greater prior to the retinal deterioration than after it has begun. The dulling of retinal sensibility also reacts to the same end, by not demanding of the ciliary muscle a clear-cut image. It is only by a circumlocution that we can express the diseased condition; we may call it a macular choroido-retinitis, due to abnormal focalization of the image or light-stimulus,—or, briefly, ametropic choroido-retinitis.

DEFINITION.—Chronic changes in the macula region, chiefly in the retina, but possibly implicating the choroid, occurring in the young or old, but generally in young adults, caused by long-continued ametropia (abnormalism of the image or light-stimulus), evidenced objectively by pigmentary changes, and subjectively by subnormal visual acuity, retinal asthenopia, and lessened reaction to weak light.

ETIOLOGY.—These changes are primarily produced by a long-continued, non-physiological stimulus, that slowly produces a functional abnormalism in the molecular and organic reactions of the retina, and that finally ends in organic tissue-change. The location and limitation of the lesion points to a connection with the exercise of habitual vision, and its association with chronic uncorrected ametropia affords clinical proof of the assumption. It is only natural that structures so highly and complexly differentiated and of such amazing delicacy of adjustment should be influenced pathologically by an abnormal stimulus. It has long been a source of wonder that the retina was little subject to idiopathic degenerative and inflammatory changes. I question if this supposed immunity is so great,

or if it will remain so great. I believe this lesion is far more frequent than supposed, and that it will become yet more so. The habitual and evolutional function of the retina is its peculiar response to a marvellously delicate and finely adjusted, properly focussed, image. Its task is to transform light-waves into nerve-force, the change of etherial into neural vibrations. In this work the retinal pigment cells are a *sine qua non.* Their existence precedes even the most primitive type of eye in the lowest animal. How they act is a mystery, but without them vision is impossible. It could hardly be otherwise than that a severe, long-continued, and abnormal stimulus, to which, prior to the invention of printing, the retina was never accustomed, and that is now acting with almost malignant exaggeration, should finally produce some such result as this macular choroido-retinitis.

PATHOLOGY.—Histological examination of the changed tissues is greatly to be desired. So far as I know no such studies by accurate and competent hands have been made. The fact that we know nothing of the intimate nature of the physiological retinal function precludes exact knowledge of pathological modifications. It may, indeed, transpire that the abnormal changes of the retina are secondary to changes in the cortical or trophic optical centres. The nerve messages from the macula must necessarily be inaccurate and functionally imperfect, and the labor of transforming them into normal sensations by the central cerebral cells must be greater, and may be reasonably supposed to produce abnormalism there, which might react upon the retinal end-organs and induce secondary functional and organic changes in them. Whether this be so or not, and whether such changes are trophic, inhibitive, or failures to adjust, etc., must for the present remain undecided. They are certainly morbid, and their objective manifestation consists in pigmentary changes; the subjective, in loss of visual acuity and power. These things, therefore appear to me indisputable: The retinal function is necessarily bound up with that of the pigment cells; long-continued ametropia, not neutralized by spectacles or by the ciliary muscle, produces patho-

logical changes in the pigmentation of the macular region, and a loss of visual acuity and power in proportion to the extent of the pigmentary abnormalism. The physiological production of light or color out of ether-waves requires the intermediation of peculiar colored organs in the retina. A chronically abnormal condition of the ether waves or chromatogenic stimulus would almost necessarily produce pathological conditions of the colored organs. Abnormalism of stimulus everywhere results in pathological reaction.

OPHTHALMOSCOPIC PICTURE.—I have found that the peculiar brownish stippling of the macular region may precede any great loss of visual acuity if the eye has its refractive error corrected and the object be illuminated with a strong light. At this time the pathological change may be easily overlooked. It is difficult to observe the macula when the person is young and the visual acuity perfect. The differential diagnosis between physiological and pathological pigmentation of the macula, is the most difficult task in ophthalmoscopy, and requires the most exquisite judgment and delicacy both of physical and mental technique. When at a later stage the tiny dots of brownish discoloration have become confluent and more pronounced, their discovery becomes more easy. Examined attentively and by certain lights and lenses of the ophthalmoscope there may be seen bright or dull white dots or sinuous irregular white streakings, very small and delicate, interspersed among the brownish stippling and layers. Rarely these seem more noticeable than the pigment anomaly. In almost all cases the pigmentary changes show some sort of symmetrical or concentric relations with the macula, and do not usually extend beyond a radius of ten or twenty degrees. I think I have noticed that the more pronounced changes are above and to the outer side of the macula,—corresponding to the position of the most continuous stimulation by the retinal image. The fovea itself shows more decided pigmentation. In advanced stages the circumjacent pigmentation assumes a stratified appearance like clouds about the setting sun, only the color, a tawny dark brown, is darker than in earlier

stages, but is never black like that in other types of true choroidal inflammations. With an increase of the morbid changes there is a synchronous atrophy of optic-nerve fibres supplying this region. The remainder of the fibres and the peripheral field of vision remain normal in extent and function.

SYMPTOMATOLOGY.—The usual reflex symptoms of eye-strain will be found inversely proportional to the amount of permanent loss of visual acuity, or what amounts to the same thing to the amount of retinal damage. The greater the amblyopia the less the headache, gastric disturbance, etc. The chief and constant complaint is of inability to continue near work except for short periods (which is exactly in ratio of the macular injury and pigmentation), and by means of a strong light. The image cannot be held, fades out, the eyes tire, lachrymation and other symptoms of irritation follow if the suffering retina is forced to continue its work. The loss of acuity is not generally remediable by lenses, though the correction of ametropia is possible, the patient clearly distinguishing between better or worse, while not being able to decipher a smaller letter. The loss of acuity is exactly in proportion to the amount of pigmentation about the macula as shown by the ophthalmoscope. Both eyes may be affected, and in different degrees, or but one alone, and the eye that has had the most imperfect image is the one usually showing subjectively and objectively the greatest abnormalism.

PROGNOSIS.—Though my experience is too limited to be dogmatic, I believe that at most any stage of the disease the visual acuity then remaining may be permanently retained upon the condition that the treatment be rigidly followed. I doubt if any recovery of the lost visual power is possible.

TREATMENT.—The treatment is simple, and consists in the most perfect correction of the ametropia possible. Eye-strain must be permanently relieved. In addition to this, in cases when the acuity, we will say, has fallen as low as $\frac{20}{40}$, there should be permanent renunciation of all near work, at least except by aid of a strong white, steady light, —that is, by clear daylight. Whatever near work be

allowed must also be done by bits, as it were,—a short period of near work followed by a long period of rest.

SUMMARY.—Chronic uncorrected ametropia may result in permanent lesion of the macula region, usually accompanied by pigmentary changes, with deterioration, probably permanent, of visual power and acuity. "Central choroiditis" is explainable in this way. Simple myopia is the least apt to produce the lesion, and myopic astigmatism, simple and compound, comes next in order. Simple hyperopic astigmatism is next in the hierarchy, followed by simple hyperopia, whilst compound hyperopic astigmatism, especially if complicated with insufficiency, occupies the highest place as an etiological factor.

Apart therefore from the grave systemic and cerebral reflex disorders (dyspepsia, mal-assimilation, headache, chorea, etc., etc.) produced by "eye-strain," there is great risk to the ocular mechanism itself—to its most vital and essential part—in leaving ametropia chronically uncorrected.