ANALYSIS OF THIRTY-SEVEN CASES OF UVEITIS.*

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The form of uveitis I propose to consider is choroido-cyclitis. The cases, with two exceptions, were seen in private practice. They divide themselves naturally into those with and without descemetitis. This was found in twenty of my thirty-seven cases: two of serous iritis and eighteen of choroiditis with descemetitis. The closest description I have found in text-books of this form of choroiditis is by A. Hill Griffith in Norris' and Oliver's "System." Under the heading "Anomalous Forms" he describes a "Choroiditis with Descemetitis." He had seen forty or fifty cases, chiefly in young women. His explanation of the descemetitis is: "The dots on Descemet's membrane are formed in the choroid, set free in the vitreous and carried by the nutrient currents of the eye to be deposited on the back of the cornea, which view necessitates the permeability of the suspensory ligament by solid particles."

As to causation, syphilis was never found. Anemia and tuberculous family history were sometimes present, and the cases treated accordingly; but in many the cause was undetermined. My own cases confirm this difficulty of fixing a cause. In giving as "causes" associated life epoch or functional defect, I mean only to suggest such inference as can be drawn from association in a number of cases. As descemetitis is the boundary between this and other forms of choroiditis observed, it may be well to state the method of looking for it. This was direct examination, aided by the 20-D. convex lens. Mydriasis, by furnishing an illuminated background, brings the spots into prominence. In a few cases, seen very early, the dots were not found, but appeared by the second or third day. Nor were they transient, lasting two or three weeks. General muddiness of vitreous, in distinction from a fairly clear chamber with large or fine opacities, hyperemic disc and plaque-like white or yellow deposits somewhere in the fundus were uniform. A striking feature was the small number showing retinal hemorrhages or marked implication of retinal vessels. In only three was retinal hemorrhage found. The exudate was choroidal. In two cases, examined several months after the attack, no trace of exudate was found. In such others as afforded late inspection there was atrophy. Subjectively, all presented the same initial symptom: sudden loss of visual acuity, varying from cloudy appearance of objects, with test vision of 20/40 or less, to positive peripheral or central scotoma, or seeing only moving hand. Maximum loss came quickly. Improvement was very gradual and slow, being noted as late as a year after the attack. In twelve of the twenty cases vision normal or as good as 20/30, was ultimately obtained. The paracentral atrophy observed in many of these cases was strikingly inconsistent with such vision. The scotoma was negative, and the eyes functionally satisfactory. Permanent vitreous opacities and central atrophy accounted for visual defect in others.

Omitting for the moment the two cases of serous iritis in girls of 15 and 16, there were eighteen cases of this form of choroiditis, seven male, eleven female. Five male and seven female presented primary, two male and four female relapsing attacks. Of the eighteen cases the site of acute exudate, or atrophy from former

attacks, was central or paracentral in ten, peripheral in eight. In relapsing cases the second or later exudate adjoined atrophic spots.

Etiology is best studied with sex and age. There was no reason to suspect syphilis in any of the cases. Nor could I get a history implying tuberculosis. Of the five primary male patients, one, age 23, developed a large peripheral exudate a week after trauma—blow from end of a ladder. In two, 22 and 27, no cause was found. The patients were reliable business men, apparently in perfect health. Neither in them or in any of these patients was there tendency to myopia, or other chronic degenerative changes. A fourth, 23, had suffered from chronic constipation for years; but this was the only trouble found. Seven years have passed since this attack, and he has had no relapse, or shown constitutional disease. The fifth, 25, seen in '94, with right central exudate, had a tapeworm at the time, and was of frail build. He was not tuberculous, and has not, I believe, developed it since. In June, 1901, while I was at the St. Paul meeting, this young man had a relapse in same eye, and was attended by my friend, Dr. Randolph. During the past winter he underwent operation for appendicitis. In two remaining male patients, who had relapsing attacks, two and eight years after primary, no cause for latter was found. The ages were 16 and 38. Thus, in these seven male cases in but one was a definitely recognized cause found—trauma. Associated gastro-intestinal disorder was an accompanying condition in two, and nothing was discoverable in the others.

Of the seven primary female cases one, 50, was rheumatic. She presented to a marked degree rapid opalescence of the lens during, and persisting for sometime after the attack. Peripheral striations found in a previous examination for refraction, were not affected. A second case, 39, followed traumatism—blow from a piece of wood, in two weeks. It is noteworthy that failure of accommodation seemed the earliest symptom. When I saw her after two months, there was 20/20 central vision, with peripheral scotoma, corresponding to exudate. A third, 36, was seized with central choroidal exudate while sewing. No cause was found. In a fourth, 37, sympathetic ophthalmia seemed the most likely explanation, though a severe attack of grippe, immediately before the eye trouble, may have had influence. The right eye had been lost eight years previous, after puerperal fever. The globe was shrunken, with opaque cornea, and obliterated anterior chamber. When I saw the patient this atrophied ball was injected, painful to touch, and had been so since the grippe. The left eye had normal central vision; but ciliary injection was present. Pupil was, if anything, hypersensitive. The next day descemetitis appeared, and two days later a faint peripheral choroidal exudate with scotoma in upper and inner field. Recovery after enucleation of right eye was complete in three months. The primary attack in the other seven female patients occurred in the second decade of life. Adding to these the two cases of serous iritis, there are thirteen cases in the female sex, in nine of whom uveitis made its first appearance during the early years of menstrual life, this function being, so far as known, normal in four, abnormal in five. Three of the four relapsing cases were under the care of gynecologists. It is impossible to say whether the pelvic trouble causes the eye lesion or if both are due to some hemic or other influence at present unknown.

To summarize: This form of choroiditis seems to differ from others, 1, in ophthalmoscopic appearances; 2, mode of onset and early symptoms; 3, etiology, so far

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as we are aware; 4, prognosis. Some of them get entirely well by absorption of exudate, but, especially if atrophy remains, there is great danger of relapse. This may continue indefinitely, but so long as the fovea is spared, useful vision remains. A woman of 42 gave history of six attacks, during each of which she was practically blind; yet, in spite of numerous atrophies she had

Fig. 1.—A. Paracentral scotoma corresponding to fundus lesion. Field limits normal.

normal central vision. It is also noteworthy that in but one of the eighteen cases were both eyes involved.

Of the seventeen cases without descemetitis syphilis was present in three, while a fourth showed typical foveal changes, though neither history nor other evidence pointed to systemic infection. In the three, periods of two, six and nine years had elapsed since the

Fig. 2.—B and C. Areas of color scotoma and dim white perception. S. Absolute transient scotoma. Field limits narrowed.

initial lesion; considerably beyond the usual time for secondary eye symptoms. Interesting queries are: how recent should infection be to justify causative relation, and, are the syphilitic foveal lesions sufficiently characteristic of this, in distinction to other forms, to justify diagnosis in absence of history or other manifestations? In these, in two cases of choroiditis disseminata and five

with subretinal extravasations, visual impairment was confined to the field area corresponding to fundal lesion. Scotomata existed in eight, macropsia in one, photopsia in two; symptoms characteristic of choroidal disease. Causes, so far as determined, were syphilis in three, unknown in the fourth with specific changes, and gout in both cases of the disseminated variety. Hemorrhages occurred in two old patients who had atrophies, one

Fig. 3.—E and D. Shape of color scotomata and dim white areas.

during convalescence from pneumonia, a fourth in connection with severe asthenopia, the fifth coincident with menstrual suppression.

I shall use the remaining six patients as a basis for inquiry into the diagnostic and prognostic significance of the symptoms of so-called "choroidal hyperemia," and vitreous opacities. In Griffith's excellent article, already mentioned, the indefiniteness of symptoms of

Fig. 4.—Narrowing of upper temporal field with decided "cut-in."

choroidal congestion is dwelt upon, reddened disc being practically the sole sign. This comes through cilio-retinal anastomosis. He says: "Even patches of localized inflammation may not be discoverable till they have broken through the hexagonal pigment layer of the retina." Concerning vitreous opacities, he says: "They are often absent through the entire course of the disease.

Their presence depends on the coincident implication of the retina rather than upon the choroiditis itself, and they are found only where the retinitis is more marked than usual, and the inner layers are affected, as shown by opacity of the retina and blurring of its vessels." He regards "the retinitis as a separate process, from the same cause which produces the choroidal trouble, and not as an extension of the latter." I can-

ocular appearances in an eye in which he had a positive scotoma in the upper temporal field. Central vision was unaffected. Symptoms had followed unusual eye work, and recovery came after a few months' rest. I have had under observation for ten years or so a lady in the fifties who has persistently complained of muscæ in the right eye. Central and peripheral vision have been normal. Several times have I noted this reddening of the

Figure 5.

not resist the conviction that the statement concerning vitreous opacities appearing only in connection with opacity of the retina and blurring of its vessels will not tally with general experience. I am sure I have seen intensely red disc and muscæ without involvement of retinal vessels. I have such a case now under observation: a lady 32 years old, with simple myopic astigma-

Figure 6.

tism, normal central and peripheral vision. Red discs and floating bodies were apparent on first examination a year ago. Refraction correction seems to have relieved the choroidal hyperemia, if such was the meaning of the red disc; yet fine vitreous opacities are still observable, and the eyes are not capable of prolonged work. A boy of 19, student at Johns Hopkins University, whom I examined six years ago, had the same intra-

Figure 7.

Figs. 5, 6, 7 show progressive narrowing of right temporal field; letters Y, X, W, color scotomata and dim white areas always preceding loss of portion of field.

disc, and fine vitreous opacities when reviewing her glasses. In March she came with a choroido-retinal exudate. Some influence, apparently rheumatic, has kept the eye sensitive and finally produced ophthalmoscopic choroido-retinitis.

Fig. 8.—Only a little temporal field left. Nasal field contracting. Space V, color scotoma, as in other figures, encroaching well into nasal field.

In the following three cases changes followed each other in such rapid succession as to leave no doubt that the red disc and vitreous opacities meant serious trouble. A lady, 31 years of age, was seen in December, 1900, for defective central vision in the left eye. She had consulted another oculist a month previous, who had found choroiditis, and given an unfavorable prog-

nosis. Vision was 20/70, with a paracentral scotoma (Fig. 1). Red disc, floating vitreous opacities and perifoveal pigment heaps were present. In eighteen months fundal appearances have not materially changed. In March, 1902, she thought this eye was getting worse. Examination showed a peripheral positive scotoma and two field areas in which colors were lost and white was dim (Fig. 2). On May 27 the shape of the dim areas had changed, and scotoma disappeared (Fig. 3). In the right eye, which was examined and found normal in December, 1900, visual changes have progressed. In February, 1901, she complained of pain, which yielded to atropia. Vision was 20/15, field normal, but accommodation had fallen off. She needed +2 D. to read 1 Jaeg. I found a few small muscae in the anterior vitreous, and reddened disc. A month later a small choroido-retinal exudate was found in lower nasal fundus, only after careful search, and field examination had demonstrated narrowing up and out (Fig. 4). From this there have developed in the temporal field dim areas as described above, soon becoming scotomatous and gradually approaching the fovea (Figs. 5, 6 and 7). Central vision was affected only after nine months, when it was 20/30, with complete loss of temporal field. The dim area has now crossed into the nasal field, and central vision is reduced to 20/70 (Fig. 8). Only hyperemic discs, floating bodies in a clear vitreous and insignificant peripheral changes have been found. It seems to me that atrophy of the retinal cells is the only explanation dependent upon a pre-existing choroiditis, possibly, or an independent retinal affection from the same cause which produced the signs of choroidal hyperemia, as held by Griffith. In this case no cause was found, unless it was an offensive post-nasal catarrh. It is possibly worthy of remark that the father of this lady once had what was called "hyalitis," from which he recovered. He himself, a very intelligent man, while understanding the causes of such things, could throw no light on the etiology in his own or his daughter's case.

The two other patients were men, 37 and 40, both in robust health, free, so far as could be determined, from syphilis or tuberculosis, one a farmer, the other a country merchant. Both consulted me for the same symptoms—asthenopia in reading and muscae. Both eyes were involved in one, the muscae being seen only by the left eye in the other. Central and peripheral vision was normal in the first case in both eyes. The discs were red, vitreous, cloudy and contained many opacities, but no fundal exudate or hemorrhage was found. In a few months both eyes had detached retina and secondary cataracts formed later. Removal of these has given ability to walk alone. The other man of 40 presented similar red discs with vitreous opacities. The left eye presented a small peripheral retinal extravasation. Central vision was 20/20 R., 20/70 L. E. The right eye has developed only defective accommodation found at first. With emmetropic refraction he needs at 40 +1. for near. The left eye is lost, save in lower periphery of field, by detached retina and vitreous clouds. Here, then, are six cases, all free from syphilis, tuberculosis, central lesion or suspicion of toxic disturbance. All showed the same initial fundal lesion, red disc, the accepted sign of choroidal hyperemia and vitreous opacities. In three there was abnormally low accommodation. One had no lessening of visual acuity; one a scotoma which disappears with rest, a third developed, after years, an exudate. Three patients lost useful sight by retinal atrophy or retinal detachment.

To conclude, the 37 cases indicate: 1. That men-

struation, in its establishment, or later, if abnormal, intestinal disorders, acute infections and nasopharyngeal disease should be reckoned among possible causes of plastic choroiditis. Whether or not these conditions are themselves direct causes or lower resisting powers and so enable other causes to act, is uncertain. Nor can it be definitely said that some of these coincident conditions may not themselves be manifestations of recognized causes of uveitis; rheumatism, anemia and the like. 2. These cases of obscure etiology show greater tendency to relapse than forms whose cause is better known. They almost invariably show descemetitis. 3. The fundal changes of so-called "choroidal hyperemia," especially if accompanied by defective accommodation and vitreous opacities, demand guarded prognosis and repeated examination for dim field areas.

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(The symposium on uveitis will be continued by the papers of Drs. Friedenwald, Hansell, Wilder, Woodruff and Marple and the discussion.)

REMARKS ON THE METHODS OF OPERATIONS IN VOGUE FOR CYSTOCELE WITH AND WITHOUT PROLAPSE OF THE UTERUS.*

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When I again take the liberty of bringing the subject of operative treatment of cystocele and prolapse of the uterus, already threshed over in many gynecological societies, before this scientific body, it is not my intention to add to the large supply of methods in vogue a new procedure, substantiated perhaps by two or three operations, but to point out a few thoughts which I believe to be altogether timely.

There has been and still seems to be a great difference of opinion in regard to the question whether retroversion or retroversio-flexion of the uterus, so often connected with cystocele, is the antecedent and primary cause of the cystocele and prolapse or whether, as H. W. Freund¹ expresses himself in his article, "Ueber Moderne Prolapse-operationen," "The descent of the anterior vaginal wall is usually primary, as shown by the extended observation of puerperal cases." There is, in my opinion, hardly a doubt that both opinions find their analogies and that four possibilities of origin of prolapse of the anterior vaginal wall and uterus have to be taken into consideration:

First.—The descensus of the anterior vaginal wall is the primary event in the evolution of prolapse. This class is chiefly produced by sub-involution of the anterior vaginal wall during frequent previous births or attenuation during the climacterium, and we may find sometimes a large cystocele while the uterus shows probably elongation of the cervical part but lies in normal anteversio-flexion.

Second.—A retroflexion, or in most of the cases a retroversion is the causative agent of the prolapse and cystocele. I am absolutely of the opinion that in most of all the cases the mechanism of prolapse occurs in this manner.

Third.—In very few cases an excessive hypertrophy of the vaginal portion of the cervix may be the introducing medium of prolapse. This hypertrophy, which is often-

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