THE OCULAR COMPLICATIONS OF BRIGHT'S DISEASE.*

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CINCINNATI.

GENERAL STATEMENT.

The eyes become involved as a result of the general systemic conditions arising in Bright's disease. These systemic conditions, briefly considered, are the result of faulty kidney excretion, leading to the retention of urea or other excrementitious substances in the blood. These retained products are either poisonous in themselves or secondarily lead to the formation of toxins, thus producing inflammatory and degenerative changes in the vessel walls and the tissues of the body; or they may interfere with the proper conversion of the elements of food into those favorable for assimilation by the cells of the body, thus producing anemia and hydremia. To this abnormal condition of the blood is attributed the general arteriofibrosis, which finds its expression in hypertrophy of the heart and general increased arterial tension, leading to exudation and hemorrhage or total occlusion of the arterioles, with subsequent death of the parts cut off.

In the eye we find the vascular system (the choroidal and retinal vessels) and its nervous expansion (the optic nerve and retina) most frequently involved, although its media, the aqueous, lens and vitreous, may be secondarily affected. Paralysis of ocular muscles may follow as a result of cerebral involvement. All of the conditions are observed clinically, as hemorrhages, inflammations and degenerations.

The hemorrhages are due to the combined influence of arterial degenerations and increased arterial pressure. The inflammations and degenerations follow in the wake of improper nutrition and presence of toxins.

As a rule, as in all constitutional affections, ocular involvement is bilateral, but in some cases only one eye is primarily affected, and active treatment may so control vascular and nutritive conditions as to prevent the second eye from becoming affected. Ocular involvement is met with at all times of life, and may occur during the progress of acute Bright's disease, the Bright's disease of pregnancy, chronic parenchymatous and chronic interstitial nephritis. The majority of cases have been attributed to the last-named.

No doubt, many cases of Bright's disease develop ocular symptoms which are never discovered, as neither choroidal nor retinal disease causes pain, and unless the functional disturbances are prominent they are allowed to pass without further investigation. On the other hand, the oculist is not infrequently consulted for failing eyesight and makes the first diagnosis of Bright's disease.

In the acute forms, however, as a result of the rapid accumulation of toxic products in the blood, we find the nervous tissue of the eye more especially involved, and these cases are distinguished by the severe and diffuse character of the pathologic changes, from which the tissues may or may not be able to recuperate.

In the subacute forms it has been held that the changes are due to an interference with the nutrition in consequence of alterations in the vascular system. These vascular changes are attributed to toxic influences, but here likewise I believe that the toxins or other re-

tained excrementitious substances play a more directly important rôle.

In these two forms of the disease we find either a diminished or normal quantity of urine and large quantities of albumin, and it has been inferred that the loss of albumin is responsible for improper nutrition. But this would not in any way explain the cases of chronic interstitial nephritis, in which the eye symptoms are said to occur most frequently, and in which we find large quantities of urine and only a trace or absence of albumin. So that in chronic Bright's disease we seek the cause of the ocular symptoms in the retention of excretory products in the fluids of the body which, acting as an irritant or a toxin, produce inflammatory and necrotic changes.

These excretory products, though constantly present in Bright's disease, in the beginning are not so in overwhelming quantities, and the tissues may be able to adjust themselves to changing conditions (a possible reason why all cases of Bright's disease do not develop retinal and choroidal changes), but in some a point is reached where the intoxication becomes excessive and the tissues react to the influence of irritants or poisons, and gradually succumb to permanent changes which are not easy to combat and offer a very unfavorable prognosis.

So that it seems rational to ascribe the ocular changes, especially choroidal and retinal, in all forms of Bright's disease to the same cause; namely, some constituent of the urine which is not excreted, but is retained in the blood, and acts as a chemical irritant or a toxin.

What the chemical nature of the products retained in the blood are, is as yet unsolved, but in all probability is connected with an improper elimination of urea. Since I have fixed my attention on this subject, I have found in a number of cases of Bright's disease, independent of the variety, a diminution of the urea quantity in the urine; that is, a retention of urea in the blood. In other words, the ocular symptoms are evidence of acute or chronic uremia. And we may place in the same category the cases of uremic amaurosis, in which the lesion or irritation is in the brain.

All text-books are vague on this subject. No attention is drawn to the fact that the seriousness of the case is not to be gauged by the loss of albumin but by the degree of auto-intoxication from retained excretory products, and that as yet we can do this only by a study of the percentage of urea in the urine.

So far as I know, no one has yet attempted to show that in all cases of choroidal and retinal disease accompanying Bright's disease there is less urea excreted, nor has a study of the relationship between diminution of urea quantity in the urine and the simultaneous increase of the severity, or even the presence of ocular symptoms, been made. We do know, however, that where the skin can be made to do the work of the kidneys vision does improve, though it be only for a time, since the control of eye symptoms depends on our success in controlling the kidney disease.

If this view that all cases of ocular involvement are due to chronic uremia and show a diminution of urea in the urine should be proved correct by observations in larger numbers of cases, it would also explain the reason for the comparatively rapid fatal outcome of nearly all, if not all, these cases.

The question would naturally suggest itself, how long a time after changes have taken place in an eye can a favorable outcome be expected? I should take it that vascular changes and exudates offer a better prognosis

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than changes in the nervous elements, the impairment of which, if degenerative, is permanent.

The degenerative changes are usually so matted in exudates that when these are cleared away the results are often better than hoped for. Rods and cones cut off from their nutritive supply for three or four weeks can not be restored, nor will ganglion cells take on new life. A degenerated vitreous can not be restored, nor will lenticular changes and opacities be resorbed. So that the degree of vision ultimately to be retained depends entirely on the degree of insult to the globe before the patient comes under observation, the energy with which treatment is pushed, and, finally, the form of the disease, since better results may be expected from acute than chronic processes.

The ocular symptoms of the Bright's disease of pregnancy cause special concern. We are confronted here not only with the possibilities of a spontaneous abortion and the dangers to the mother incident to such an occurrence, but we are called on to decide whether it is justifiable to produce an abortion to save the eyesight of the mother, since it is well known that the kidney disease in such cases subsides quickly after labor is terminated. Ocular involvement in pregnancy indicates latent, but more serious possibilities, namely, puerperal convulsions, uremia and death.

In pregnancy, where albumin is found in the urine, the eyes should also be examined. The ocular lesions are but more delicate and sensitive reactions due to widespread intoxication, to which the whole nervous system is undoubtedly also subjected. An estimation of urea at this time becomes a matter of special importance, and where this falls below the normal the time for active interference has arrived. The estimate should be made with special attention to diet and quantity of fluid ingested for 24 hours.

Where the pathologic changes in the retina are not great, pregnancy far advanced and urea quantity nearly normal, one may temporize, but where widespread exudates and degenerative changes are taking place, urine loaded with albumin and urea quantity low, I believe labor should be induced not only to prevent permanent serious impairment of eyesight, but equally because the eye symptoms indicate the seriousness of the general condition and the possible fatal outcome of the case.

Whereas, in Bright's disease, both acute and of pregnancy, we may look forward to a certain degree of clearing up of the media and an absorption of exudates, so that we meet with partial, at times even complete, restoration of function, and the prognosis is favorable, in chronic forms the eye symptoms, as a rule, follow in the wake of nutritive disturbances of long standing, the degenerative changes are gradually induced and are premonitory symptoms of approaching dissolution. Various authorities place the time of life after retinitis sets in in the chronic form of Bright's disease at from three months to two years as the ultimate limit. I have seen death ensue within twenty-seven days after retinal degeneration was recognized. Only a small percentage of cases of Bright's disease (5 to 6 per cent.) develops ocular symptoms, but where this occurs it is always an ominous sign and a signal for active treatment.

The treatment of the ocular conditions of Bright's disease has for its object the furtherance not only of the elimination of toxic substances from the system and the general improvement of the nutritive conditions, but purposes a rapid interchange of fluids, so as to bring about a rapid resorption of noxious fluids and exudates

in the eye. This is accomplished by stimulating or controlling the heart's action, by the drinking of large quantities of water, so as to flush the kidneys and dilute the poisons; to rid the system of the latter by active purgation, and, above all, by active diaphoresis, to cause the skin to do the work of the kidneys. Diaphoresis is of paramount importance. It is remarkable how quickly vision clears up on use of Turkish baths, where patients can get them, or by the use of a hot pack. (Pilocarpin injections are used, but are very depressing to the heart action.) The baths must be taken at least three times a week, and where the physical condition permits of it, every day. The hope of attaining good vision depends entirely on the rapidity with which we secure absorption of exudates, rid the system of poisonous substances retained, and on our success in improving the general nutrition.

RESUME OF CASES.

Of 16 cases of the severer forms of the ocular complications of Bright's disease under my care 5 were cases of optic neuritis, 3 of neuroretinitis, 2 of neuroretinitis hemorrhagica, 2 of neuroretinitis and choroiditis, 1 venous thrombosis, 2 uremic amaurosis and 1 keratitis, in all probability a general uveitis. Seven cases had previously had medical treatment; only 5 were referred to me; so that in 11 cases attention was drawn by the ocular conditions to the true character of the disease.

Two cases occurring during the progress of pregnancy might fairly be considered as acute Bright's disease, and both are living. In one case vision has been greatly impaired, the woman having aborted in the seventh month. The other, a case of general uveitis, went to full term and was delivered of twins, and sight was fully restored.

Four were cases of chronic parenchymatous nephritis subsequent to la grippe, typhoid and measles. Three of these died, and there is no doubt in my mind that the fourth case died, though he passed from my observation. All of these cases showed more or less edema of their lower limbs, and two of them showed enormous hypertrophy of the heart, the whole left side fairly quivering with each pulsation.

All of them had a large percentage of albumin in the urine, and in two of them the urea quantity was found below the normal, and in one, though the urea quantity was not estimated, the patient had had uremic convulsions before I saw him.

Ten were cases of chronic interstitial nephritis. Two of these were in all probability of syphilitic origin; 1 was due to tubercular kidneys; 1 to general atheroma and senility; in 6 no cause could be assigned. Two of the latter (8 and 12) had no history of ever having been ill, while three had been treating for Bright's disease for periods of from 2 to 6 years. All had a greater or less degree of hypertrophy of the left ventricle, and showed traces of albumin; 1 case had had uremic convulsions, and 4 of the others showed a diminished urea quantity.

Seven of my cases died, the shortest period being, in one case, five days, in another twenty-seven days after coming under observation, the longest period being one year and three months. In 8 cases the urea quantity was below the normal; 2 had had uremic convulsions before I saw them.

Four cases passed from observation and the subsequent history is unknown, and 3 cases are living, the two cases complicating pregnancy and the one of retinal venous thrombosis.

Two cases had had apoplectic seizures previous to the development of the ocular symptoms.

Two cases have come under observation within the past month and are still living.

CONCLUSIONS.

1. There are no ocular complications of Bright's disease from which, independently, a positive diagnosis of Bright's disease can be made. They should direct our attention to the probability of Bright's disease and lead to systemic examination.

2. The prevalent idea that one must find the characteristic picture of Bright's disease, with hemorrhages and areas of degeneration and white splashes in the region of the macula, is a mistaken one, since one may meet with any of the many manifestations of vascular or retinal inflammation due to the action of toxins.

3. Only a small percentage of cases of Bright's disease develops ocular complications, but if larger numbers of cases were systematically examined it is more than probable that a much larger percentage would be found to have ocular lesions, which have not caused functional disturbances, and consequently have been overlooked.

4. All the ocular complications of the disease are the result of altered nutrition, or toxemia; in other words, a chronic uremia.

5. Diseases of the eye coming on in the course of Bright's disease are of the most serious prognostic import, and indicate an early dissolution of the patient.

6. In the acute form conditions are of a more hopeful character, though a prolonged siege, with inability to stay ocular disease, can not fail to leave the patient, though he recover, with greatly impaired vision.

7. An estimation of the urea quantity made from a total amount of the urine of 24 hours, under proper diet, is the barometer by which we are to be guided in determining the early or immediate fatal outcome of the case.

8. In the Bright's disease of pregnancy, with ocular involvement, a low urea quantity should be a positive sign for immediate interference, and should justify artificial abortion both to save the eyesight as well as the life of the mother.

9. In cases of Bright's disease disclosing choroidal and retinal changes I have found the urea quantity in the urine below the normal; and it seems more than probable that the urea or other excrementitious substances retained in the blood, due to faulty kidney excretion acting as chemical irritants or toxins, are directly responsible for the inflammatory or degenerative changes.

ILLUSTRATIVE CASES.*

OPTIC NEURITIS, CHRONIC INTERSTITIAL NEPHRITIS.

CASE 1.—Mrs. L. G., 57 years of age, came of her own accord, Dec. 22, 1902, owing to a sudden diminution in her vision. She has had two children; one died in its eighth year. The son is living and married. She has had some severe spells of illness; did not walk for six years, and states that she had peritonitis and has had an ischiorectal abscess. Is nervous and constipated, but has had few headaches. For the past two years her heart has troubled her considerably. A year and a half ago she had a stroke of paralysis of her entire right side, including an anesthesia of the right side of her face. Improvement began in the leg in a few days, but the arm and hand have never fully regained their strength. She had no aphasia at that time. From the time of her paralysis, she states that her vision was poor, and she found

great difficulty in reading and sewing, but did not consult an oculist at that time. Her right eye, she says, has since that time been of little use to her, but the left eye has only been failing during the past week.

Examination.—She appears fairly well nourished, but says her weight has fallen in the past two years from 160 to 126 pounds. Her eyelids are markedly swollen, but no other signs

Fig. 1.—Case 1. Optic neuritis, left eye.

of edema could be found in other portions of her body. The heart is enlarged; pulse strong and full, 100 per minute, and at times intermits and gets tumultuous. Heart extends one-half inch beyond the nipple line.

An examination of her fields of vision disclosed on the left eye loss of the entire nasal and lower segment of 150 degrees,

Fig. 2.—Case 2. Optic neuritis, right eye.

and the remainder of the field is contracted down to 30 or 40 degrees, and this is extremely hazy, but in the retained segment colors are faintly recognized.

On the right eye only the upper nasal quadrant, including the region of the macula, is retained, and this likewise is contracted. This loss of the nasal half of the left eye and the

* The illustrations are the work of the author, made from sketches taken by the direct method.

temporal half of the right eye would lead one to suspect a hemorrhage in the visual center on the left side at the time of her paralysis a year and a half ago, and this, I should take it, is entirely independent of her present illness, and this is partly borne out by the ophthalmoscopic examination, in which we find the left eye disclosing a pronounced neuritis, whereas, in the right eye, the disc is plainly visible and gives one more the impression of an optic atrophy. The consensual and direct reaction of the pupils is somewhat slow, though intact.

Ophthalmoscopic Examination, Left Eye.—Vitreous slightly hazy, through which, however, the entire fundus is visible. The disc is markedly swollen and enlarged to double its size, and appears as a deep hyperemic area which is gradually lost in the retina. It has not the characteristic fire-brand appearance, but very few enlarged capillaries being noticed on the surface. It has more the granular appearance. The arteries are normal in size, sharply outlined. The superior, however, can not be traced to their origin in the disc, but seem to emerge far out on the retina proper. The veins are double their normal size, extremely tortuous. A few striated hemorrhages are visible in the upper inner quadrant in the hyperemic area, close to the disc. Nowhere on the fundus can any signs of retinal degeneration or conglomeration of white exudates be observed.

Fig. 3.—Case 2. Optic neuritis, left eye.

Right Eye.—The fundus is veiled by vitreous opacities, but nevertheless the disc is distinctly visible and sharply outlined, with the suggestion of a bluish cast. There is just the slightest suggestion of beginning neuritis, notwithstanding the atrophy. The arteries are normal in size, whereas the veins are dilated, but show no tortuosities so markedly defined in the left eye. There is likewise here no suggestion of retinal degeneration.

Examination of the Urine.—Acid reaction, fair quantity of albumin; no sugar; urea quantity 0.35 normal. Passes enormous quantities of urine daily.

Treatment.—This patient was given bromid to quiet the heart, placed on a milk diet and given energetic sweat baths, and her vision in a few days commenced to clear up.

Subsequent History.—December 27. The hyperemia of the disc is not so great, inasmuch as on the left eye the vessels in the superior part can be followed now to their origin on the disc.

Oct. 2, 1903. The patient is still living.

OPTIC NEURITIS, CHRONIC PARENCHYMATOUS NEPHRITIS.

CASE 2.—S. S., 23 years of age, was referred to me for examination owing to some edema of his lower eyelids and for a very annoying scotoma scintillans. Suffered from typhoid

fever in his fourteenth year, and was refused life insurance in his nineteenth year owing to albumin in his urine.

Examination.—He has a slight hypertrophy of the heart; apex beat a little below and to the left of the nipple line; pulse rapid and bounding.

Examination of the Urine.—Approximately three pints are passed per day, loaded with albumin; urea half of the normal quantity. Chronic parenchymatous nephritis.

Ophthalmoscopic Examination, Left Eye.—A distinct optic neuritis; the edges of the disc are faded, many dilated capillaries radiating from the disc, and a large area of edema follows the vessels for the lower half of the retina in their course. A white, edematous area occupies the region of the macula; vision = 6/6.

Right Eye.—Vision = 6/9. The disc is likewise edematous, swollen, great hyperemia of the vessels and a similar edematous area surrounds the vessels of the lower half of the disc.

Subsequent History.—This patient was not seen again until June 12, 1901. He tells me that he has had uremic convulsions; was in coma at one time for 36 hours. Ophthalmoscopic examination of both eyes showed large atrophic areas, many of them punctate in form, a result of violent choroidal inflammation. These areas are especially confined to the regions immediately surrounding the discs and in the macular region

Fig. 4.—Case 3. Neuroretinitis, right eye.

and for a distance along the line of great vessels, but they do not extend into the periphery. Owing to his great weakness, ophthalmoscopic drawings could not be made. He died Sept. 10, 1901—just one year and three months from the first time he visited my office.

NEURORETINITIS, CHRONIC INTERSTITIAL NEPHRITIS.

CASE 3.—Mrs. B., a widow, aged 55 years, was referred to me October, 1896, for examination for glasses. She states that she has been under treatment for chronic Bright's disease for some time.

Examination.—Hypertrophy of the left ventricle, and an accentuated second sound. Patient is emaciated, weak and suffers from severe headaches, but has no edema.

Examination of the Urine.—Large quantities of albumin. Diagnosis, chronic interstitial nephritis.

Left Eye.—Vision equals 6/60, with +150 D.; no improvement. Examination of the fundus shows the typical picture of retinitis Brightica. Papilla is enormously swollen, giving the typical fire-brand appearance, +3 D. required to see it distinctly. All capillaries are widely dilated and tortuous, with many hemorrhagic foci scattered over the enlarged papilla. The veins are tortuous, overfilled, almost double their usual size, the arteries almost normal, but very tortuous.

In the region of the macula is a large atrophic area, surrounded by pigment, the result of choroidal atrophy. In the center of this area can be seen distinctly an exquisitely, sharply defined point, which reflects the light like a flame. Around this area of macular degeneration are many splashes of fatty degenerated retinal tissue. Also, the characteristic stippling on the region of the macula. Following the course

Fig. 5.—Case 3. Neuroretinitis, left eye.

of the vessels are also many splashes of retinal degeneration.

Right Eye.—Much the same condition prevails—the hemorrhagic areas on the papilla, if anything, greater. The swollen papilla seems to extend far out into the retina proper. The region of the macula presents the characteristic figure, and here, likewise, areas of retinal degeneration follow the lines

Fig. 6.—Case 4. Neuroretinitis, right eye.

of the vessels. Above the macula are several hemorrhagic foci.

Subsequent History.—No improvement was observed from treatment and the patient died in June, 1897.

NEURORETINITIS, CHRONIC PARENCHYMATOUS NEPHRITIS.

CASE 4.—William N., 33 years of age, was referred to me Sept. 10, 1901, for an examination of his eyes, which began to

fail a week previously; he is a clothing cutter, married and has always led an abstemious life. He had typhoid fever a year previous to his present illness. He has had many dizzy spells, headaches and sickness of his stomach. He had a uremic attack a week previous to his visit, since which time his vision has begun to fail.

Examination.—He has an enormously hypertrophied heart; pulse 120, full and tension high. His whole left side quivers with each pulsation. He has all the signs of arteriosclerosis on his temples.

Examination of Urine.—He passes 7.5 pints of urine daily, which is loaded with albumin. Diagnosis, chronic parenchymatous nephritis.

Present Treatment.—Nitroglycerin to reduce violent heart action, and sweat baths by means of the hot-air apparatus; salts.

Left Eye.—Vision=6/60. The entire retina is so edematous that conditions in the temporal half can scarcely be observed. There is also some cloudiness in the vitreous, close to the retina, and considerable exudate behind the retina, so that the retina has a grayish appearance, with a possible slight detachment. In the region of the macula are some white glistening points and everywhere are white striated patches,

Fig. 7.—Case 4. Neuroretinitis, left eye.

more particularly in the upper half of the retina, where several large hemorrhagic exudates exist. The disc itself is so enormously swollen that the vessels can be only followed for a very short distance, and disappear in exudates, appearing again at long distance removed. There is enormous dilatation of all the capillaries of the disc and hemorrhagic areas scattered over the entire retina.

Right Eye.—Vision=4/60. The disc is edematous; enormously swollen; veins and arteries widely dilated and tortuous; disappear on the surface of the disc, and appear again at some distance from it. Many flaring hemorrhages exist over the entire fundus, and white areas of choroidal exudate, as well as degenerative and fatty changes in the retina, are marked everywhere. The characteristic star figure in the region of the macula is present.

Subsequent History.—Ocular conditions were not materially improved by the general treatment, and the patient died Nov. 20, 1901, having suffered from general edema of the lungs, glossitis, neuralgia, and finally dying of an enormous hemorrhage of the stomach, possibly a uremic ulcer of the stomach.

NEURORETINITIS, CHRONIC INTERSTITIAL NEPHRITIS.

CASE 5.—April 5, 1902, I was called to examine the eyes of Mrs. F. L., who had been under treatment for chronic interstitial nephritis for the past six years. She had had repeated

uremic attacks; was much emaciated and very weak. Her pulse was rapid and feeble; she was unable to read anything, and objects at a short distance were covered by a cloud.

Ophthalmoscopic Examination, Left Eye.—The vitreous was full of floating opacities, through which the disc is seen swollen, exceedingly hyperemic; its edges indistinct, and radiating from the disc in every direction are many flame-like

Fig. 8.—Case 5. Neuroretinitis, right eye.

hemorrhages. The disc is surrounded by many white patches, some of them flame-like; others confluent, and in the region of the macula is a large white area, surrounded by many glistening points. The *right eye* presents practically the same conditions.

Treatment and Subsequent History.—The patient was put through an energetic course of sweat baths, and vision seemed

Fig. 9.—Case 5. Neuroretinitis, left eye.

to improve, but death ensued June 21, 1902, the patient having been in uremic coma for four days.

NEURORETINITIS HEMORRHAGICA, CHRONIC INTERSTITIAL NEPHRITIS.

CASE 6.—Mrs. O. H., 64 years of age, came in March, 1902, to have her eyes examined for glasses, stating that her vision had been poor for the past four weeks; she is a large, stout

woman, weighing 185 pounds, with no history of ever having been ill, except the occurrences of headaches and dizzy spells.

Examination.—The heart is enlarged, apex beat two inches beyond the nipple line, pulse 120, full and strong.

Examination of the Urine.—Sp. gr. 1008, much albumin, no sugar, no casts. Chronic interstitial nephritis.

Ophthalmoscopic Examination, Left Eye.—Vision is 2/60;

Fig. 10.—Case 6. Neuroretinitis hemorrhagica, right eye.

can read no print. The disc is hyperemic; swollen; edges indistinct and hazy. Extending outward from the disc, taking in the area of the entire macular region, is a large hemorrhagic area, in some places so dense as to appear almost black. There is also an area of degeneration appearing white and glistening, exactly in the center of the macular region.

Fig. 11.—Case 6. Neuroretinitis hemorrhagica, left eye.

Right Eye.—Vision equal to 6/36; disc is swollen; edges washed out, and extending downward and outward is a flame-like area of dilated capillaries, in which the inferior temporal artery is lost. The macula is not involved.

Subsequent History.—The seriousness of her condition was disclosed to her daughter, but they refused to believe my statements, and the case was lost sight of.

NEURORETINITIS HEMORRHAGICA, CHRONIC PARENCHYMATOUS NEPHRITIS.

CASE 7.—Miss E. F., aged 18 years, came of her own accord Aug. 22, 1899, owing to gradual diminution of vision. She states that two years previously she had the measles, subsequent to which she developed dropsy. Her father died of Bright's disease and two sisters died of tuberculosis. She has four sisters and two brothers living. She has had no other

Fig. 12.—Case 7. Neuroretinitis hemorrhagica, right eye.

disease of childhood, but since having the measles she has never been well. At present she is very pale, anemic, listless; at times can not follow conversation; has constant pain in head, vomiting spells every day, especially on arising in the morning; her feet are swollen. She has never had a complete convulsion, though at times cramps occur in her hands and legs. She has had severe diarrhea at times.

Fig. 13.—Case 7. Neuroretinitis hemorrhagica, left eye.

Examination.—Pulse rate is 128, full and strong. Her heart is enlarged beyond the nipple line and an accentuated second sound heard all over her left side.

Examination of Urine.—Her urine is loaded with albumin and the urea quantity is half normal. Diagnosis, chronic parenchymatous nephritis.

Left Eye.—Only light sense. Ophthalmoscopically the disc appears about three times its normal size, pure white, from which gradually radiate dilated capillaries. The veins are more than triple their normal size, the superior disappearing in exudates, the inferior, with great tortuosity, extending downward. The arteries are entirely lost in the exudate and swelling. The entire periphery of the retina is covered with radiating hemorrhagic areas, sometimes fine, sometimes in large blotches. The region of the macula displays a wonderful fatty degeneration, a formation of cholesterin crystals, extending downward, forming a large, almost triangular area.

Right Eye.—Vision $\frac{4}{60}$. The papilla is more normal in size, but swollen. From its edges radiate dilated capillaries, which give the fire-brand appearance, and which are gradually lost on the surface of the retina. The arteries and veins are dilated and tortuous, and to the nasal side and upward are many fine radiating hemorrhagic areas, whereas, below are many confluent, white plaques. The region of the macula disclosed many white spots, and just above the macula is a large hemorrhagic exudate.

Subsequent History.—This young lady took to her bed the day I examined her, and died September 18, just 27 days after my examination.

Fig. 14.—Case 8. Retinal venous thrombosis.

RETINAL VENOUS THROMBOSIS, CHRONIC INTERSTITIAL NEPHRITIS.

CASE 8.—Mr. E. B., aged 64, consulted me Jan. 31, 1898. He stated that on awakening from a sound sleep on the morning of January 30 he observed that vision of his right eye was completely blurred. He denies all venereal disease; in fact, he has no recollection of ever having had any disease.

Examination.—There was no external sign of disease. His pulse is strong and incompressible; heart enlarged.

Examination of the Urine.—Sp. gr. 1022; alkaline reaction, with but a trace of albumin; no casts.

Ophthalmoscopic Examination.—Vision of the right eye was but $\frac{6}{24}$, and of the left eye a complete blur. The nasal and lower halves of the disc are indistinct and covered with fine hemorrhages. The two inferior veins are dilated to nearly four times their normal size, and following them downward in their course you find them strewn with hemorrhagic exudate; especially the inferior temporal vein shows two points at which the vein is entirely hidden by a grayish white mass, presumably the dilated portion of the vein, which I hold to contain thrombus. The veins in the lower half of the fundus are tortuous, and the entire lower half of the retina is strewn with hemorrhagic splashes. Curiously the entire upper half of the fundus, except a portion of the nasal, is free from hem-

orrhage; its veins and arteries are normal; whereas, in the lower half of the fundus, the arteries are much smaller.

Subsequent History.—This patient remained under observation for more than a year, but whereas the hemorrhagic exudate absorbed, his condition remained practically the same, and the upper half of the field was entirely wanting. He is still living.

[The discussion on the papers of Drs. Stricker and Suker will follow the paper of the latter next week.]

THE DEVELOPMENT OF HARD TISSUE IN THE PULP OF HUMAN TEETH.*

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Of the functions performed by the various organs of the body there are few, if any, more interesting or have the power of producing more varied results than that of the tooth pulp. In health it is the benefactor of the surrounding hard tissue; in disease, the medium whereby we are warned of the mischief that is going on. Among the varied functions of this organ may be placed the power of developing "secondary dentin."

Nature in many cases endeavors to overcome the evil produced by the destroying microbe, by forming a layer of fresh tissue between the soft, sensitive pulp and the dentin that is being broken down by decay; the same protective measure also occurs in those cases where erosion has commenced its destructive work, as well as in many other instances where disintegration of the hard tissue has been produced, by the various known causes. In all these examples the newly formed hard tissue is known as secondary dentin, the development of which we desire to consider in this paper.

On the examination of teeth, we find we may have new tissue more or less continuously developed from the pulp, until the tooth is lost either by disease or as a result of old age. In other cases the new tissue may be developed, without any apparent cause, and the development may be either continuous or spasmodic until the whole of the pulp chamber becomes filled with this new tissue. Again, we may find it in the pulp itself more or less surrounded by pulp tissue in the form of pulp nodules, and as Prof. Jos. Archovy has designated certain growths "Odontoma internum liberum."

In any and all of these cases the new tissue is spoken of as secondary dentin. It will thus be seen that there is a variety of tissue grouped under the head of secondary dentin, ranging from the comparatively structureless tissue as seen in the pulp nodule to that of the complicated character known as osteodentin. (Figs. 1 and 2.)

It may enable us to better understand the development of this tissue if we divided it into classes. We shall, therefore, consider first, normal secondary dentin; second, dentin of repair; third, pulp nodules, and lastly, a form of secondary dentin found in teeth where pyorrhea alveolaris has been pronounced.

NORMAL SECONDARY DENTIN.

In the first class we shall include all tissue produced apparently without any cause, the whole of the pulp and surrounding tissue being healthy at the time of the deposition of the tissue. A good example of such may be frequently found in those teeth perfectly free from diseases, such as decay or erosion. Nor have the teeth

been worn down by attrition; yet on examination they are found to have their pulp chambers partially or entirely filled with new tissue. This tissue may be very varied in its microscopic appearance, from that of an almost structureless character to the more complicated containing lacunæ-like spaces, and frequently an innumerable number of tubuli. The tissue is usually found in the twelve anterior teeth, but perhaps more

Fig. 1.—A pulp nodule, showing structureless character of secondary dentin.

frequently in the lower incisors and canines. There is, as a rule, no inconvenience produced by its development, and its presence is only detected on examining the tooth after extraction.

It would appear to be Nature's way of reducing the size of the pulp chamber, and is much more pronounced in those who have passed middle age. It is rarely

Fig. 2.—a. Osteodentin.

found in pulp canals; the deposition commences usually at or near the apex of the pulp chamber, gradually working downward toward the point where the chamber is united to the pulp canal. Even when the tubuli are in large numbers they rarely assimilate with those tubuli previously formed; hence it is not at all difficult to define the line of demarcation between the two tissues. This line is usually very pronounced, owing to the fact

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