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GOUTY RETINITIS, CHORIO-RETINITIS, AND NEURO-RETINITIS.

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During the last few years my attention has been attracted by certain lesions occurring in the fundus in gouty patients, and the more I have studied these cases the greater has my interest in them grown. The pathological condition of the blood in gout does not always work in a way that readily attracts attention, and this is particularly true in regard to the eye. Most authors who refer to retinal complications occurring in the course of gout only mention a hemorrhagic retinitis as being the most frequent retinal complication. Thus Nettleship says: "Hemorrhagic retinitis is more commonly met with in gouty persons than in others. It may be uniocular or binocular. The children or descendants of gouty persons, without being themselves subject to gout, are liable in early life to this insidious form of eye disease." (*Diseases of the Eye*, 4th edition, p. 445.)

Other authors refer to the subject in a still more casual or general way. F. Gauté says that there is a gouty retinitis which strongly resembles certain types of syphilitic retinitis, and which may or may not be accompanied by opacities of the vitreous, retinal hemorrhages, migraine and zona ophthalmia. (*Thése de Paris*, 1881.) Lychon speaks of intraocular hemorrhages and neuro-retinitis as due to gout. (*Thése de Paris*, 1885.) Jonathan Hutchinson, in an article on primary intraocular hemorrhages, says that gouty persons are prone to hemorrhages, venous obstruction, and irregularities in circulation due to a too large arterial pressure and a relaxed condition of the vascular walls, and that vitreous opacities may in these cases be constantly present. According to him there are four possible sources of these phenomena, viz.: First, changes in the blood; second, increased arterial tension; third, disease of the walls of the blood vessels; fourth, hypertrophy of the heart. He considers that both low tension and high tension are alike compat-

ible with liability to rupture of the capillaries. A condition of loss of balance is easily induced, as the vessels are not well under vasomotor control. The risks of rupture of the capillaries will be increased if there be hypertrophy of the heart or weakness of the walls of the blood vessels. (*Transactions of the Ophthalmological Society of the United Kingdom*, Vol. I, p. 26.)

Gowers speaks more positively in regard to retinal lesions in gout. He believes that spontaneous inflammation of nerve trunks and plexuses on one side only, and recurring after the age of thirty or forty, is seldom due to any other cause than gout. This always means primary perineuritis. Still, inflammation of the sheath of the optic nerve behind the eye is less common in ordinary gout than inflammation of the retina. Gowers thinks the characteristics of these cases are the greater degree of disturbance of the vision than corresponds to the visible changes in the optic disc, the tendency to irregular defects in the field of vision, and the strong tendency to lesion of the other optic nerve by an independent, symmetrical morbid process. (*Medical Ophthalmoscopy*, 3d edition, 1890.)

Galezowski says that in this disease the changes may begin in the retina and extend to the choroid and give rise to a retino-choroiditis, characterized by an alteration in their vessels and deposits in and outside their walls. When the retina alone is involved there exist only vascular alterations. Atheroma (?) is frequent in the retinal vessels of gouty people, and usually attacks persons of advanced years—from seventy to seventy-five. The papilla is ordinarily not involved, but all around the macula and along the vessels are brilliant patches of exudation, generally along the arteries. The rest of the retina may remain intact. The lesion is generally unilateral. The subjective symptoms are the same as those of ordinary retinitis. Gouty retino-choroiditis occupies the central zone of the fundus, is circumscribed posteriorly, the exudation is usually extensive, with hemorrhages and sometimes pigmentary deposits. The disease develops slowly, usually involves both eyes, and never ends in total blindness, as the periphery of the retina is rarely affected. (*Annali de Ottalmologia*, XIX, p. 199.)

We know that the retinal hemorrhages met with in gouty

persons may involve one or both eyes, and are of sudden onset. Small flame-shaped hemorrhages are scattered all over the fundus, and if the extravasations are punctate they have serrated margins. The disc may be hazy, but there is no such regular, glistening, white deposit as is met with in so-called retinitis nephritica. The veins are usually engorged and tortuous, and more or less indistinct by an effusion of blood into their sheaths. It seems hardly justifiable to separate this group of cases sharply from other forms of retinal hemorrhage associated with renal disease and diabetes, since renal disease is so often a concomitant of gouty cases. The influence of the cardio-vascular system must be carefully considered in these patients. Hutchinson thinks that these hemorrhages are due rather to venous obstruction, such as thrombosis of the retinal vein, than to arterial disease, as more explanatory of the suddenness of the attacks.

The effects of the poison of gout upon the vascular system are now generally recognized to be: First, a high blood pressure in the arteries; second, hypertrophy of the left ventricle; third, hard, incompressible arteries undergoing atheromatous change. From these result either apoplexy by rupture of a blood vessel or aneurism by dilatation of the vessel, or angina pectoris, or fatty degeneration of the heart.

Some years ago Dr. George Johnson found in cases of chronic renal disease a thickening of the muscular wall of small arteries and arterioles, which he attributed to increased contraction of the muscular coat due to the abnormal qualities of the contained circulating blood. This led to a damming of the blood in the arteries, a rise of blood pressure, increased action of the heart in systole, and hypertrophy of the left ventricle. Mahomed has since shown that high arterial tension is not a consequence but an antecedent of kidney disease. High blood pressure within the arteries is connected with spasm of the arterioles and hypertrophy of the thin muscular coat, and leads to hypertrophy of the left ventricle. The overdistension of the arteries causes a growth of connective tissue under the tunica intima of the arteries, which we call atheroma. Then follows hypertrophy with failing heart and arteries.

There seems to be no doubt that a gouty optic neuritis is occasionally met with, for Hutchinson and others have reported cases, and it is probable that the perineurium is first affected, leading to thickening and compression of the bundles of nerve fibres, the adjacent lymph spaces being filled with an exudation from the blood vessels. There is also but little doubt that a gouty phlebitis of the central retinal vein and its branches is occasionally met with. This leads to a roughening of the surface of the internal lining, which favors the occurrence of thrombosis. In these cases, owing to the gouty poison, there is naturally hyperinosis and tendency of the blood to clot. Duckworth thinks that there is probably a determination of acid urates to the part, which acts as the directly exciting cause.

Most of our knowledge of this condition of the arteries has been gained from Thoma's investigations. He concluded that arterio-sclerosis is caused by some general disturbances of nutrition, which are partly the result of infection, intoxication, and other pathological changes of varying kinds, and partly the accompaniment of senile changes. These general disturbances of nutrition cause not only disease of the arteries, but also of the veins and capillaries. In the arteries the middle coat is thinned, and there is a loss of elasticity by a diminution of the resistance which the wall of the vessel opposes to the stretching of the blood pressure. The vessel is stretched in all directions, it widens, and its lengthening leads to tortuosity. The widening of the lumen is followed by a connective tissue deposit in the intima, and this is followed in turn by a number of retrogressive changes, such as fatty degeneration, or calcareous degeneration, or hyaline degeneration. Similar changes occur in the veins. In the capillaries there is an increase in the porosity of the capillary wall and œdematous infiltration of the tissue. The important consequences of this so-called angio-sclerosis are varicose dilatation of the veins and aneurisms. In the beginning of the process there is a loss of elasticity of the vascular wall, shown in life by a soft, high pulse and pulsation and tortuosity of the arteries, notably of the retina. At this stage there is the greatest danger of rupture of the arteries. Later, the vessels, by reason of the deposit of connective tissue in the

intima, become firm and rigid. (*Archiv für patholog. Anatomie*, vols. 93, 95, 104, 105, 106, 111, 112, 113. *St. Petersburger Med. Woch.*, 1890. *Fortschritte der Med.*, 1884. *Deutsche Med. Woch.*, 1888. *Archiv für Ophthalmologie*, 1889.) In endeavoring to explain the results of these vascular changes we must consider three factors: First, the shortening of the median coat; second, compensatory endarteritis; third, atrophy of the muscular coat of the artery. A permanent slowing of the blood current in an artery is followed by a narrowing of its caliber, which is brought about in a regular way on the one hand by a contraction of the tunica media and on the other hand by a deposit of connective tissue in the tunica intima. Any retardation of the blood current in the arteries and veins which is not completely and at once neutralized by a corresponding contraction of the media leads to a hyperæmia of the vaso vasorum and to a new growth of connective tissue in the intima, which narrows the lumen of the affected vessel and consequently does away more or less completely with the normal rapidity of the current.

With this there is associated later a similar formation in the media and adventitia. The first recognized departure from the normal condition of a vessel consists in the stretching of the media, which is the cause of the diffuse primary arterio-sclerosis characterized by a dilatation and tortuosity of the arteries, by an eccentric hypertrophy of the media, and by a diffuse, compensatory fibrous thickening of the intima. All of these changes I have repeatedly seen develop in the retinæ of gouty patients while under treatment. We know from the investigations of Loring that arterio-sclerosis in the eye can be followed for years and the gradual obliteration of the vessel traced. Evidences of its presence are the tortuosity of the arteries, pulsation in their blood current, opacities in their walls, often a diminished lumen, hyaline degeneration of some of the vessels on the papilla, aneurism of the central artery, and varicose retinal veins. More rarely there occur complete obliteration of an artery and thickening of the walls of the veins with partial obliteration of their lumen. Changes like these in the central retinal artery and its branches are probably associated with similar changes in the ophthalmic artery and internal carotid.

More recently observers have begun to recognize corresponding changes in the veins, and to regard phlebo-sclerosis as a systemic disease analogous to diffuse arterio-sclerosis. Some years ago Sack described a condition which he called a chronic fibrous endo-phlebitis, and stated that it not infrequently appeared as a local lesion in consequence of local stasis in the venous system. Huchard in 1889 and Spillmann in 1890 both used the term phlebo-sclerosis to describe this process. According to the latter, phlebo-sclerosis in gouty persons may be both circumscribed and diffuse, the latter being met with in the small veins. The same pathological changes occur in the vaso vasorum, with an abnormal development of nuclei around the small capillaries. It is shown by his investigations that the venous lesions develop progressively and simultaneously with the arterio-sclerosis, without, however, ever ending in the profound alterations which change the arteries into rigid tubes. (*Gaz. hebd. de Méd. et de Chir.*, Oct. 11, 1890.)

Pokrovsky has made still more extensive investigations upon thirty cases. Microscopically the veins showed no marked changes, though they were sometimes found studded with circumscribed, irregular elevated white patches, both soft and hard. He concluded from his investigations that chronic inflammation of the arteries, so called, arterio-sclerosis, or chronic endarteritis deformans, is accompanied by analogous, though less intense, morbid changes in the veins. The most frequent process is a compensatory, diffuse, fibroid endo-phlebitis, analogous to Thoma's compensatory diffuse endarteritis. The endo-phlebitis commences with the appearance in the *intima* of the veins of many young cells, which are subsequently transformed into fibrous connective tissue. The young cells are supplied mainly by proliferation of the elements of the intima. Besides this diffuse inflammation there is a circumscribed nodose thickening of the venous walls, which is usually associated with retrogressive metamorphosis, and is analogous to the nodose arterio-sclerosis. As regards the regressive changes, the most frequent is hyaline degeneration, met with in all the coats of the veins. Fatty degeneration rarely occurs. In cases of intense endo-phlebitis the *media* frequently undergoes an inter-

stitial inflammatory process, with the formation of numerous blood vessels, profuse cellular infiltration, and proliferation of interstitial connective tissue. The adventitia is usually affected in a slighter degree. (*London Medical Record*, August 20, 1890.)

Raehlmann's paper on the changes in the retinal vessels in general angio-sclerosis, also calls attention to the changes in the veins. In about half the cases examined there were corresponding changes in both arteries and veins in the retina. The veins were relatively widened, always had a whitish edge or margin, and in some cases appeared changed into white cords, and the blood column seemed entirely interrupted. In some places varicose dilatations were distinctly visible. (*Zeitschrift für Klin. Med., XVI*, 1889.) His investigations have shown that while extensive sclerosis of the vascular wall is usually accompanied by a hyaline degeneration of the tissue elements, showing like white lines along the blood column, it may sometimes be invisible with the ophthalmoscope. Raehlmann has noted the following changes in the retinal arteries: 1st. Distortion and narrowing, sometimes so marked as to simulate the condition met with in atrophy of the optic nerve, but usually accompanied by a normal field for form and color and normal activity of vision. 2d. White lines along the arteries, due sometimes to cloudiness alone, and sometimes to that and the thickening together. This consists microscopically in an alteration of the adventitia, by which the wall of the vessel is thickened without narrowing of its lumen, and also in a hyaline degeneration of the arterial wall. 3d. Local or patchy narrowing of the arteries, separated from each other by spaces of varying length in which the vessel has its normal caliber, the result of deposit in the vascular wall. In diffuse arterio-sclerosis the wall of the vessel has a more homogeneous, yellowish, glistening appearance, in which the structure elements are less distinguishable. In some places he has seen patches of arterio-sclerosis nodosa, occurring generally at the point of bifurcation. It is still an open question with Raehlmann how far these patchy proliferations of the intima, round or fusiform or irregular, as the case may be, have to do with the development of embolic and throm-

bolic processes. The vascular tension is probably increased in front of these narrowed spots, while it is diminished behind them. Complete closure of the lumen of an artery as a result of endarteritis nodosa occurs frequently. 4th. Aneurisms of the central artery and its branches.

In the venous system of the retina Raehlmann found white lines along the veins, narrowing of their lumen in local, circumscribed spots, and varicose dilatations also in circumscribed spots. He also met with pulsation in both arteries and veins, more frequently in the latter. Goldzieher has described the same pathological conditions in the eyes of a patient whom he first examined clinically, and whose eyes he subsequently examined microscopically. (*Centralbl. für prakt., Aug.*, 1889.) In one eye of this patient, besides the changes in and about the blood vessels, there was a circle of bright, glistening spots of varying size and shape, whitish-yellow in color, and without pigmental border, symmetrically grouped around the macula, which was intact. These patches of exudations proved to be entirely in the retina. He states that he has seen them in other cases, and that they were accompanied by impaired vision and an irregular central scotoma. The disc and periphery of the retina are in these cases usually normal, and the course of the disease is chronic and painless, and to these points I wish to draw particular attention, as they correspond closely with the clinical and microscopical appearances of the cases on which this paper is based.

CASE I. — In June, 1889, I was consulted by a gentleman, aged 72, at the request of his family physician. Four years previously he had consulted Dr. C. R. Agnew, who found signs of what he considered chorio-retinitis in both eyes, most marked in the left eye. After a careful examination of his case Dr. A. came to the conclusion that the trouble was due to gout, and recommended strict dietetic treatment. This was not carried out with any regularity by the patient, and the condition in the eyes grew slowly worse. The main difficulty had been an inability to read any small type. Some months before I saw him he first noticed that he could not see objects at a moderate distance as distinctly as usual, and on testing each eye separately

he found that the main defect was in the left eye. This defect has slowly but steadily increased. I found on examination, R.E., V = 20/200, with sph. + D 1.50 = 20/50; L.E., V = 2/200, unimproved, and decidedly better eccentrically. He could still read Jaeger 6 with sph. + D 5.50 with the right eye. The tension was normal in both eyes. The media were perfectly clear in both eyes. A careful perimetric examination of the field of vision showed the presence of a small, irregular central scotoma for form and color in the right eye, and a very large scotoma in the left eye.

The ophthalmoscopic examination revealed a very interesting condition of the fundus. The outline of the discs was very much blurred, resembling the first stage of choked disc, but without the œdematous swelling. In the left eye the arteries were much diminished in caliber, in several places the lumen being reduced to the merest thread, and requiring the closest examination to see the minute blood column that still existed. The white lines along the vessels, both arteries and veins, were very broad and distinct, and extended from the center of the papilla well out towards the periphery. In places, the veins seemed dilated like a fusiform aneurism, the vein on both sides of the dilatation being reduced in caliber. There were numerous patches of yellowish-white exudation in the retina, of varying size and shape, mainly grouped about the region of the macula and disc, but with no systematic arrangement, and one patch of considerable size in the macula itself. There were no hemorrhages in either eye. All these spots of exudation were in the inner layers of the retina. In the right eye vascular and exudative changes were much less marked. The white lines along the arteries and veins extended for only a short distance beyond the margin of the disc, and though the lumen of both sets of vessels was in places diminished, there was no apparent disappearance of the column of blood, and no fusiform or nodose dilatation of the vessels. The appearance of the optic disc, however, differed but little from that in the left eye. The action of the heart was strong, but somewhat irregular. There was a faint aortic and mitral murmur, and marked degeneration of the temporal and radial arteries. The urine had a specific gravity of

1018, was slightly turbid, light amber in color, and contained neither albumen nor sugar. There were no casts, but it was loaded with uric acid crystals, which were deposited on standing. His family physician confirmed the existence of gout, and agreed with me that the main treatment must be dietetic. After consultation with his family, it was decided to send him to Carlsbad, and he went, carrying a letter to Dr. Kraus, under whose care I placed him. He went through a carefully-regulated course of treatment by the waters, with strict attention to his diet, by which his general health was very much improved, but there was no improvement in his vision. On his return, I made a careful examination of the fundus of each eye, but could discover no appreciable change in the condition of the blood-vessels or the retina. While there was no demonstrable increase in the number or size of the patches of exudation, there was certainly no improvement. Peripheral vision was still fairly good, but he could not read at all with the left eye, and only with great difficulty with the right eye. This patient is still living, and the eyes show no perceptible change.

CASE II. Early in March, 1890, I was consulted by a lady, aged 45, whose vision had been failing for some months, and who had never worn glasses. In November of the previous year she had suffered from an attack of "la grippe," which assumed the form of asthma and bronchitis. She soon recovered from this attack, but suffered from a relapse, which was accompanied by severe neuralgia of the left side of the face and teeth, and by marked œdema of both lids on the left side. She had long suffered from gouty symptoms, and was saturated with the disease, the chief symptoms being an obstinate general eczema and renal colic. She had one living child and had had four miscarriages. Syphilis could be absolutely excluded. She had been to Aix-les-Bains on several occasions, and had always been greatly benefited by the waters. When I saw her, there was marked œdema of the left upper lid and orbital tissue, but no other external evidence of trouble. There was compound hypermetropic astigmatism in both eyes, the right eye being the more defective, and with the refractive error corrected, her vision was 20/30—in each eye. She could read Jaeger 5 with sph.

+ D. 2.50 ⌒ cyl. + D. 1.50, axis 180°, but could not read any smaller type. The cornea, anterior chamber, and iris were normal. There were slight peripheral opacities in both lenses, most marked downwards and inwards. There was an irregular central color scotoma in each eye, but no limitation of the field for form. The muscles were normal in their relation for all distances.

The fundus showed the following conditions: The optic discs were reddened, and their outline was decidedly blurred, as if smeared with a yellowish exudation. Both arteries and veins were reduced in caliber, and the white lines along the vessels were very broad and extended well towards the peripheral branches, particularly in the arteries. At the posterior pole were a series of patches of exudation, of varying size and shape, grouped irregularly around the macula, most of them being in the inner layers of the retina. There were no hemorrhages in the retina in either eye. The heart was quite feeble and irregular in action, and there was a faint mitral regurgitant murmur. She had never suffered from an acute inflammatory attack of gout.

Repeated and very careful quantitative and qualitative analyses of the urine were made, the whole quantity passed in the twenty-four hours being examined, and this never exceeded fourteen or fifteen ounces. Its specific gravity varied between 1020 and 1028, its reaction was decidedly acid, and it was always dark-colored and turbid, and on standing cast a bulky precipitate. It always contained a small quantity of albumen, but no sugar or casts were ever found. The precipitate was made up almost entirely of uric and phosphatic acid crystals.

This patient was immediately placed on the following dietary: For breakfast, weak black tea with a very little milk but no sugar, eggs scrambled or soft boiled, a small portion of fresh fish, and occasionally an orange or some grapes. Dinner in the middle of the day consisted of fish, poultry or game, underdone beef or mutton twice a week, some fresh-boiled green vegetables, toast, or graham bread. No butter, starches, sweets, or pastry were allowed. A glass of lithia water with an effervescent lithia tablet was allowed at each meal. Under this dietary her gen-

eral condition materially improved, and in the latter part of June she went to Carlsbad and placed herself under the care of Dr. Kraus. Under his carefully-regulated treatment, as to diet, baths, and drinking the spring waters, her improvement was very marked, not only in her general health, but also in her vision, so that when I saw her in October, 1890, her vision was 20/20 — in the right eye, 20/20 + in the left eye, and she could read Jaeger 1 with ease. The fundus, however, did not show much change. The white lines along the vessels were as distinct as ever, and seemed to have extended still farther towards the periphery. There was no occlusion of the arterial lumen, however, at any spot. Some of the patches of exudation seemed to have shriveled or contracted in size, and there were apparently no new spots of exudation. The scotomata in the field were smaller. This patient has rigidly maintained the dietary laid down for her, and her general health is very satisfactory. An ophthalmoscopic examination is made at intervals of several months, but no material change has been noticed, so that it seems fair to assume that the diseased process in the eyes has thus far been stayed.

CASE III.— In April, 1891, I was sent for to see a lady from the West, who had suffered from inflammatory gout for many years, and was so crippled by the disease that she moved with great difficulty. She was 68 years of age, and for some months had been conscious of a failure of vision in reading, and very recently for all objects at any distance. She had noticed that her eccentric vision was better than her central vision. The media were clear and the iris normal. R. E., V = 20/50 unimproved; L. E., V = 20/200 unimproved. There was a hypermetropia of one diopter, and with sph. + D 5 she could still read Jaeger 6 with the right eye. With the left eye she could read nothing smaller than Jaeger 14. The tension was normal. There was no limitation of the field for form in the right eye, but there was an ill-defined central scotoma for color. In the left eye there was an irregular scotoma both for form and color. The ophthalmoscopic examination showed in the right eye well-marked white lines along the arteries and veins, extending well beyond the margin of the disc, and one small patch of yellowish

exudation in the retina just above the macula. In the left eye the symptoms were much more marked. The caliber of both veins and arteries was decidedly reduced, and the white lines along the vessels were very broad. There were numerous patches of exudation in the retina, round and oval, and both superficial and deep, grouped round the disc and macula, but there were no hemorrhages. In two of the smaller arterial branches the lumen seemed entirely obstructed. This patient passed about 32 ounces of urine in the 24 hours, having a faint brown color and acid reaction, with a specific gravity of 1022, and a rather copious deep-yellow sediment was deposited after standing a few hours. It contained no albumen, no sugar, and no casts, after repeated examinations. The deposit consisted of uric acid crystals and urates. She stated that her father and several other members of her family had suffered from a similar loss of vision after they had passed middle life, and that they were all gouty. This patient was under my constant care for more than a year, and is still seen at intervals. At no time have there ever been any symptoms, either objective or subjective, of renal disease.

After a careful study of this case, it seemed almost hopeless to expect any benefit from regulating the diet, owing to her age and the advanced stage of the general malady, and I contented myself with prescribing a general tonic regimen, including small doses of whisky, and free lithium carbonate dissolved in lithia water. The changes in the blood-vessels and the retina slowly advanced, the white lines creeping towards the periphery, and the patches of exudation increasing gradually in size and number. On two occasions small hemorrhages occurred on the disc margin of the right eye, apparently capillary in origin. In October, 1892, she had a slight cerebral hemorrhage, causing temporary impairment of speech and transient numbness in the right arm and leg, from which she entirely recovered, and there has been no recurrence of these symptoms. She is still very helpless, and can only move about with great difficulty. The heart's action is feeble, but fairly regular. The condition of the urine is somewhat improved. She passes about the same amount, but it has a lower specific gravity, and there is less sedimentary deposit.

CASE IV. In April, 1891, I saw a man, aged 40, who gave the following history: He had always had somewhat imperfect vision in both eyes, owing to the scars of old corneal ulcers dating from childhood. He had been at various times a sailor, miner, ranchman, and bar-keeper, and had always "lived high" when his means allowed it. For some months the vision in the left eye had grown much worse, so that he was only able to read the largest type. When I saw him I found the following conditions. There were faint opacities in the cornea of each eye, that in the right eye being central, R. E. $\frac{20}{30}$—unimproved: L. E. $\frac{20}{100}$ + unimproved. With the exception of the cornea, the media were clear. He could read Jaeger 3 with sph. + D 1.50 with the right eye, but with the left eye he could only read the heading of a newspaper. There was no scotoma for form, but there was an irregular color-scotoma in each eye. The ophthalmoscope showed in the right eye one small hemorrhage on the margin of the disc, but none in the left eye. In both eyes the white lines along the arteries were quite marked, particularly in the left eye, where they were also evident along the veins, and the lumen of both sets of vessels was narrowed, not only on the disc, but in the left eye for some distance beyond it. The outlines of the discs were blurred and smooched, and the discs themselves much reddened. There were no patches of exudation in the right eye, but in the left eye there were a number of irregular, yellowish patches above and to the nasal side of the macula, all apparently in the inner layers of the retina, and one patch on the temporal margin of the disc. The heart was irregular in action, but otherwise apparently normal. The urine contained albumen, but no sugar or casts. There were about 30 ounces passed in 24 hours, which was acid, had a specific gravity of 1026, and deposited a thick brown precipitate, consisting mainly of uric acid and urates. The man denied syphilis, and had no trace of any specific lesion. There was a mild eczema on his hands and feet, and some chalky deposits in the middle and third fingers of both hands. He had drank all sorts of wines and liquors for many years, but he had never used tobacco. The patient was very stout and his breathing was very short, and after any exertion he would have

attacks of mild dyspnœa. His diet was at once regulated, and his daily supply of alcohol was reduced to the lowest amount of whisky. He was put on a general tonic treatment, with digitalis and small doses of potassium iodide, and he was advised to drink lithia water freely. Under this treatment his general condition began to improve, but its progress was slow, while the condition of the eyes grew steadily worse. The scotoma increased in size, the central vision deteriorated, the outlines of the discs became absolutely lost, though there was no swelling and no rupture of retinal blood vessels. During the first month of treatment, sub-conjunctival ecchymoses occurred in both eyes, and this happened several times during the following four months. The patches of exudation increased in number and size in the left eye, and new ones appeared in the right eye, all grouped around the macula or in the vicinity of the optic disc. The degeneration in the walls of the blood vessels became more and more marked. The white lines broadened and extended farther towards the periphery, the deposit being more marked in the arteries than in the veins. The lumen of both arteries and veins became narrower, and towards the end of life there were several small arterial branches, which were apparently entirely occluded. In both eyes there developed in several places fusiform aneurysmal dilatations of the arteries, and on the distal side of these dilatations the artery was always markedly reduced in caliber. The white lines were always broader and more marked at the points of bifurcation of the vessels.

In the latter part of September the patient developed pneumonia on the right side, and while ill with this pulmonary complication had a cerebral hemorrhage, which produced partial hemiplegia, though his speech was but little affected. He did not rally from the pneumonia, and died on the sixth day, apparently from heart failure. I was fortunately enabled to make an autopsy, and removed the posterior halves of the eyes and optic nerves as far as the chiasm for examination. The autopsy showed very extensive degeneration of all the intracranial vessels, both arteries and veins. The circle of Willis was very stiff and rigid. The larger arteries retained some elasticity, but the smaller vessels were very stiff and in places

had apparently lost all power of contractility. There was a small clot in the right anterior lobe, near the lower margin of the paracentral lobule, and another one in the right corpus striatum, near the supero-anterior surface.

The microscopical examination of the retina, optic nerve, and choroid proved very interesting, and absolutely confirmed the ophthalmoscopic diagnosis. All of the sections of the retina, choroid, and optic nerve showed the signs of wide-spread arterio-sclerosis and phlebo-sclerosis. The adventitia and media were decidedly thickened, but in most of the sections the main increase was in the intima, which in many places was so marked as almost to obliterate the lumen. This was particularly noticeable in vessels at some distance from the margin of the disc. The proliferation in the adventitia was largely granular, while the thickening in the intima was mainly due to hyaline proliferation. Numerous fusiform, aneurysmal dilatations were found in the arteries, almost always at the point of origin of a branch, and on the distal side of these dilatations, the caliber of the vessel was markedly narrowed. No extravasations of blood were found, except a small one in the right eye near the disc, and previously noted with the ophthalmoscope. The nerve fibres on the papilla and in the retina were markedly varicose, and separated by spaces filled with finely granular matter. The connective tissue fibres were but little altered. In the retina there was a decided thickening of the nerve-fibre layer due to infiltration between the fibres of a mass of fine granules, aggregated in heaps, with occasional distinct cells provided with a cell-wall, and filled with the same granular contents. These aggregated masses of fine granules and cells extended through all the layers of the retina except the external layer, but the bulk of them were in the nerve-fibre layer. The capillaries on the optic disc were greatly dilated, and accounted for the hyperemic condition seen with the ophthalmoscope, but between the capillaries were patches of fine granules, similar to those in the retina. Sections of the optic nerves as far back as the chiasm showed the same changes in the walls of the blood vessels as existed in the retina, but there were no special changes in the nerve fibres back of the eyeball, and the nerves could be regarded as healthy.

In the choroid the blood vessels showed the same changes. The adventitia and intima of the arteries were thickened, the proliferation being more marked in the adventitia. The veins also showed the presence of phlebo-sclerosis. No very marked pigmentary changes were found, though there was some slight atrophy of the hexagonal cells. No exudation of any kind was found in the choroid, except in the thickened coats of the blood vessels.

CASE V. — In October, 1891, I saw a lady, aged 65, who for some months had noticed a gradually increasing blur before the right eye, and recently the same indistinctness of vision had appeared before the left eye. The media were perfectly clear, and the external aspect of the eyes was normal. R. E., V. = 20/40 +; L. E., V. = 20/30 — unimproved. The ophthalmometer showed a hypermetropic astigmatism of one diopter, axis 90°. She could read Jaeger 5 with sph. + D 3 with either eye. The tension was normal, and there was no scotoma or other limitation of the field. The ophthalmoscope showed an interesting picture. The optic discs were very red but not swollen, and the outlines completely blotted out, though œdema was scarcely perceptible. The appearances resembled those of papillitis without the swelling, and were more marked in the right eye. The vessels, both arteries and veins, showed the white lines well marked almost to the equatorial region of the eye, the deposit in the adventitia being, however, very irregular. In places there seemed to be a fusiform, aneurysmal dilatation in both arteries and veins, the lumen of the vessel on the distal side of these swellings being nearly obliterated. This change was more marked in the arteries than in the veins. At the first examination no spots of exudation were discovered, but as time passed, and her vision grew worse, patches of exudation in the retina began to make their appearance around the macula, and between the latter and the papilla. This patient had always been gouty, and for some years had maintained more or less carefully an anti-gout dietary. The heart's action was feeble and irregular, but no organic lesion was made out. There were about 26 ounces of urine excreted in the 24 hours, which was dark yellow in color, acid, and deposited a copious

brown precipitate. There was a small amount of albumen, but there were no casts and no sugar. The precipitate consisted mainly of uric acid and the urates, with some phosphatic crystals. It was very interesting to note the gradual development of the patches of exudation in the retina, and the very slow extension of the disease of the vascular walls. The very marked development of this lesion in the retinal vessels, together with the condition of the urine and the gouty history of the patient, pointed to extensive disorganization of the vascular walls throughout the body, and to a probable rupture of some one of the cerebral vessels at no distant day. The vision slowly grew worse, so that eight months after my first examination she could no longer see to read even the largest type with either eye. Under dietetic management and the free use of lithia, the condition of the urine improved very much, but the uric acid never entirely disappeared. Thirteen months after her first visit to me she was, without warning, attacked by all the symptoms of an "apoplexie foudroyante," and died in three hours, without regaining consciousness. I was permitted to make an autopsy, and to remove the posterior halves of the eyes for microscopic examination.

On taking off the skull-cap, the venous congestion of the dura-mater and pia-mater was very marked. All the arteries of the brain were found extensively diseased. In the substance of the left frontal lobe, about half way between the middle and inferior frontal convolutions, and dipping backwards towards the fissure of Rolando, was a very extensive clot, as large as an olive, and in the paracentral lobule near the median line was another extravasation, not so thick, but extending for nearly an inch in every direction, breaking down the brain substance completely. The middle and anterior cerebral arteries were in spots almost completely rigid, and their walls very thick and brittle. The same condition existed to a marked degree in all the arteries at the base of the brain. There was another small clot in the pons near its under surface and far back, about as large as a split pea. The optic chiasm, optic nerves, and posterior halves of the eyeballs were carefully removed and hardened for future examination. In making the sections from

the nerves, my attention was directed principally to the condition of the blood vessels, and in all of the sections of the nerve, retina, and choroid there were evidences of extensive arterio-sclerosis and phlebo-sclerosis, resembling closely the lesions described by Thoma. The adventitia and media were enormously thickened by hyaline and atheromatous infiltration, and the intima was in many places so increased in diameter as to project into and narrow the lumen of the vessel, particularly at the points of origin of small vessels. In two places in the retina there was found complete obliteration of one of the smaller arterial branches by the thickened intima, and one section showed a similar obliteration of the lumen of one of the smaller veins. The changes in the coats of the blood vessels were relatively as marked near the equatorial region of the retina, as it was near the papilla. In most of the arteries the thickened adventitia was accompanied by a corresponding thickening of the intima, but in the veins this proportionate relation did not seem to hold good, the changes in the outer wall being here more marked.

As regards the nerve fibres on the disc and in the retina, they were here and there separated by oval spaces, which were sometimes empty and sometimes contained a mass of fine granules. There was in places slight varicosity of the fibres, and in the retina there was a decided thickening of the nerve-fiber layer, and to a less degree of the connective tissue elements. Those sections which included patches of exudation showed marked thickening of the nerve-fiber layer, the thickening being due to a mass of granular bodies, some being distinctly provided with a cell-wall and nucleus, while others seemed mere aggregations of granular matter. Within the limits of these patches of exudation the nerve fibres showed many varicosities. The same variety of granular exudation or degeneration was found in all the deeper layers, except that of the rods and cones, but to a much smaller extent. The exudation was largely confined to the nerve-fiber layer, and in several instances the spaces between the nerve fibers seemed absolutely empty. There were no hemorrhages found in any of the sections. The region of the margin of the disc showed

marked changes in the capillaries, which were generally dilated, though their walls were thickened. This condition existed all over the disc, and accounted for the extreme red appearance seen with the ophthalmoscope.

The blood vessels of the choroid showed similar lesions to those found in the retina. The adventitia and intima of the arteries were thickened, the former showing the greater change. In a few sections the veins also showed a distinct thickening of their coats. The layer of hexagonal pigment cells showed patches of atrophy in some places. There were no patches of exudation found in any of the sections, and though spaces were found in the connective tissue frame-work, which sometimes contained a few granular masses of small size, they did not differ materially from the ordinary chronic signs of senile degeneration of the choroid. There was no conspicuous pigmentary change found in any of the sections. Sections made from the optic nerve back of the eyeball showed the same marked changes in the central artery of the retina, mainly in the adventitia. There was little or no change in the aspect of the nerve fibers in this part of the optic nerve from that which is met with in a state of health.

In conclusion, the points to which I draw attention are as follows:

1st. The changes in the fundus are always bilateral, though rarely symmetrical, in the two eyes.

2d. The degeneration in the walls of the blood vessels and in the retina cause marked impairment of central vision, little or no impairment of peripheral vision, and never end in blindness.

3d. The loss of central vision is always progressive up to a certain point, unless the cause of the disease is recognized early in the outset, and immediately and properly handled. Improvement of the vision after the disease is established cannot be expected.

4th. Hemorrhages into the retina are rare, except in the beginning of the disease. Their absence later is probably due to the fact that the strength of the vascular wall is increased by the deposit, though its elasticity is diminished.

5th. The most marked feature in the fundus is the development of the arterio-sclerosis and phlebo-sclerosis. This is seen by the ophthalmoscope in the vessels of the retina, and the microscope shows that the degeneration exists as well in the vessels of the choroid and optic nerve.

6th. Another almost equally pathognomonic symptom is the peculiar yellowish granular exudation in the retina, located by the ophthalmoscope around the posterior pole of the eye, and generally leaving the macula intact, and proved by the microscope to be mainly in the nerve-fibre layer, though found in all the layers except that of the rods and cones.

7th. The changes in the optic nerve fibres seem to be almost entirely intraocular, and cannot be traced for any great distance back of the eyeball.