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INTRA-OCULAR ANGIOSCLEROSIS AND ITS PROG-
NOSTIC AND DIAGNOSTIC SIGNIFICANCE

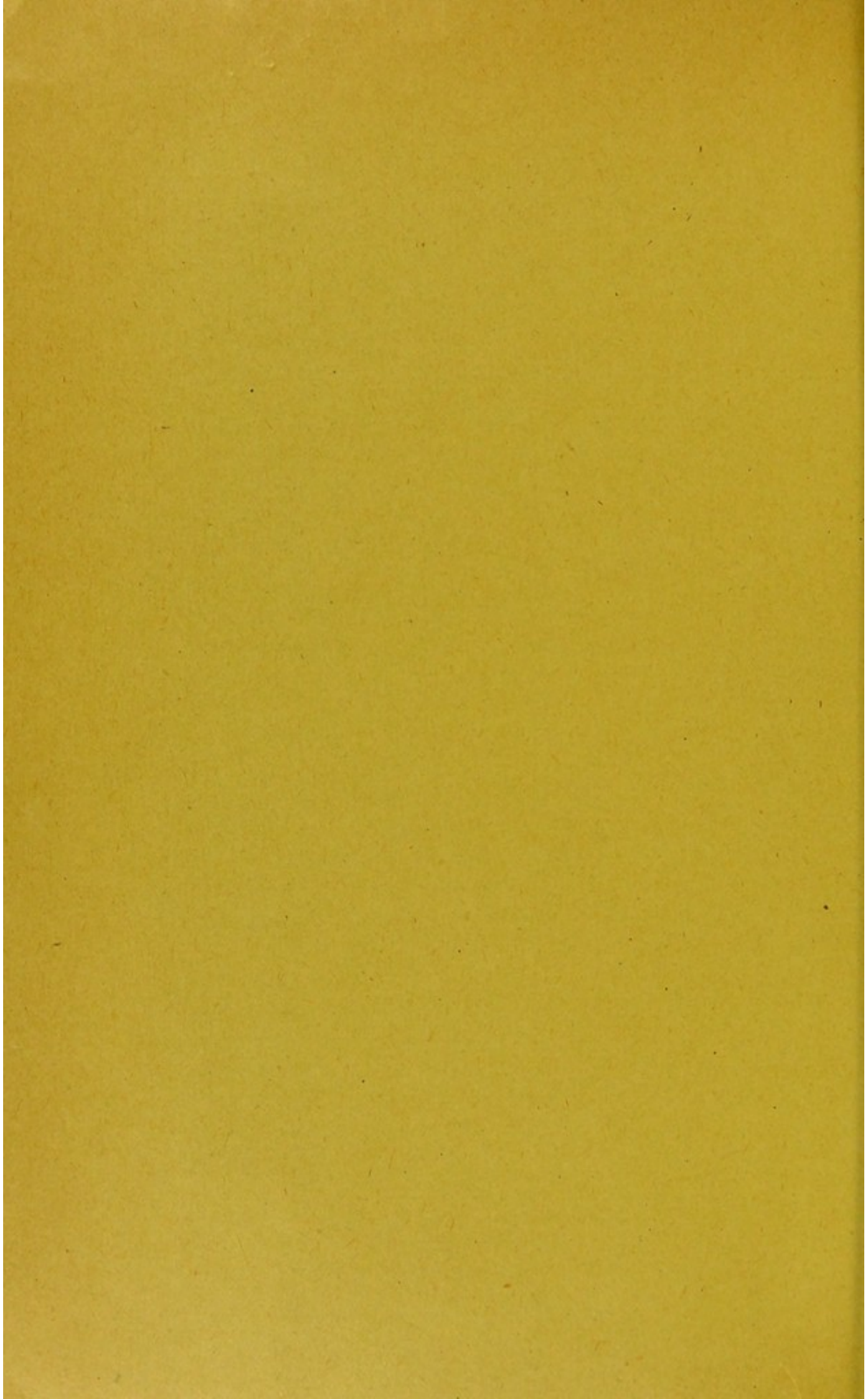
BEING AN ILLUSTRATED LECTURE DELIVERED BEFORE THE CLASS IN OPHTHALMOLOGY
OF THE PHILADELPHIA POLYCLINIC, NOVEMBER, 1906.

BY G. E. de SCHWEINITZ, M.D.,

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INTRA-OCULAR ANGIOSCLEROSIS AND ITS PROGNOSTIC AND DIAGNOSTIC SIGNIFICANCE

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AMONG the earliest ophthalmoscopic observations on atheroma of the retinal vessels is one by Hirschberg¹ in a patient aged fifty-one years, who died of an apoplexy five months after examination. From the pathological standpoint such changes had been investigated years before by Nagel, Schweigger and Iwanow. In 1889 Raehlmann² described the ophthalmoscopic appearances of the retinal blood vessels in general arteriosclerosis, with special consideration of the cerebral vessels. Ninety cases of general arteriosclerosis were studied for the purpose of determining the associated ophthalmoscopic changes in the retinal vessels, and lesions were found in practically all of them and decided alterations in 19 to 21 per cent. In 1890 Hirschberg and two of his assistants, Drs. Michaelson and Simon, examined ophthalmoscopically 50 old persons (98 eyes), and, exclusive of lenticular opacities, found only 22 eyes entirely normal. Vessel changes in the retinas in the form of alterations in calibre, narrowing of the arteries, white streaks along the walls, etc., were evident in fully one-half of these patients. In 1891, Drs. H. Friedenwald and George J. Preston, of Baltimore,³ recorded the ophthalmoscopic examination of 40 retinas in 23 patients suffering from general arteriosclerosis, and found only 7

¹ Centralbl. f. prakt. Augenheilk., 1882, vi, 329.

² Zeitschrift f. klin. Medicin, 1889, Heft 5 and 6.

³ Journal of the American Medical Association, 1891, xvi, 623.

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which appeared to be thoroughly normal. In 1892, Marcus Gunn ⁴ called attention to the ophthalmoscopic evidence of arterial changes associated with chronic renal disease and with increased arterial tension, and gave a particularly good description of the appearances of the retinal arteries under these conditions, and especially of the important sign which pressure of the sclerotic vessels may produce on underlying veins. In 1896, H. Friedenwald ⁵ added to his previous observations on the subject with a paper on the significance of constrictions and dilatations of the calibre of the retinal arteries based on the study of 33 cases of arteriosclerosis. In 1898 Marcus Gunn ⁶ published an admirable communication on the ophthalmoscopic evidence of general arterial disease, to which I shall presently make more extended reference. In 1899, before the Pathological Society of Philadelphia,⁷ and again in 1900, in an address before the Medical and Chirurgical Faculty of Maryland,⁸ I contributed some observations of my own bearing upon this topic. In 1902, Raehlmann ⁹ returned to this subject in a paper on the ophthalmoscopic diagnosis of sclerotic disease of the retinal vessels, from which I shall quote later. In 1904, L. A. W. Alleman ¹⁰ described in an excellent manner the retinal symptoms of vascular degeneration, based upon a study of 40 patients. In 1905, T. A. Woodruff ¹¹ briefly reviewed the most important retinal changes which may aid in the diagnosis of vascular degeneration. In 1906, before the American Ophthalmological Society,¹² I discussed the retinal signs of persistent high arterial tension, when this is a symptom of arteriosclerosis, making special reference to ophthalmoscopic examination in association with accurate instrumental measurement with a sphygmomanometer of the arterial tension. The whole subject of ocular arteriosclerosis was reviewed in a com-

⁴ Trans. Opth. Soc. of the U. K., 1892, xii, 124.

⁵ Archives of Ophthalmology, 1896, xxv, 177.

⁶ Trans. of the Opth. of the U. K., 1898, xviii, 356.

⁷ Proceedings of the Path. Soc. of Phila., 1897-1899, N. S. 1-2.

⁸ Maryland Medical Journal, June, 1900.

⁹ Zeitschr. f. Augenheilk., 1902, vii, 425.

¹⁰ American Medicine, 1904, February 20, 304.

¹¹ Trans. of the American Academy of Ophthalmology and Oto-Laryngology, 1905, p. 89.

¹² Trans. of American Ophthalmological Society, 1906, xi, part i, 87.

prehensive manner by Rohmer before the Société Française d'Ophthalmologie in 1906, and the address delivered in the Post-Graduate Course of Ophthalmology at Oxford, July 24, 1906, by George Coats,¹³ dealt with intraocular vascular disease, largely from the pathological standpoint, and in a most interesting manner.¹⁴ You will, therefore, see from this brief and very imperfect résumé of the literature, that this subject has occupied a large share of the attention of ophthalmologists during the last twenty-five years.

In order to refresh your memories, I throw upon the screen a water color of the *normal fundus* of the right eye examined by the direct method of ophthalmoscopy. You will observe that the optic nerve is nearly round, with a slightly pinkish tint, except at the centre, which is occupied by a whiter spot, the "light spot," which if it is deeper, forms the "physiological cup," and which marks the position of the entrance and emergence of the retinal vessels. The margins of the disc are clear, there is no striation of the retina or œdema of this structure hiding them. From the central light spot the principal retinal arteries emerge and into it the chief venous trunks empty. The arteries traverse the surface of the eye-ground, dividing into numerous branches and, passing above and below, spread in greater size and number over the temporal half of the retina, sending small branches toward the macula, and in less number over the nasal side. Fine branches arise from the central large trunks, or spring directly from the nerve, and pass outward and inward and also undergo numerous divisions. These vessels are not tortuous, and each one is evenly produced; that is to say, there are no notable variations in their calibre. The veins pass over the eye-ground in the same general direction as the arteries and in close relation to them; in some places they are crossed by arteries, in others they pass over them. They are slightly wavy, but not tortuous, and are larger than the arteries in the proportion of three to two. They also are evenly produced and are in no place pressed by an artery, with which they lie in contact. Each vessel presents a double contour, owing to a bright stripe which passes along the centre. This is the so-called "light reflex." It is more marked

¹³ The Ophthalmoscope, 1906, iv, 605.

¹⁴ Other important contributions to the pathological side of the subject of vascular disease of the eye are: Hertel, Archiv f. Ophth., lii, 191. Harms: Archiv f. Ophth., 1905, lxi, Heft 1 and 2.

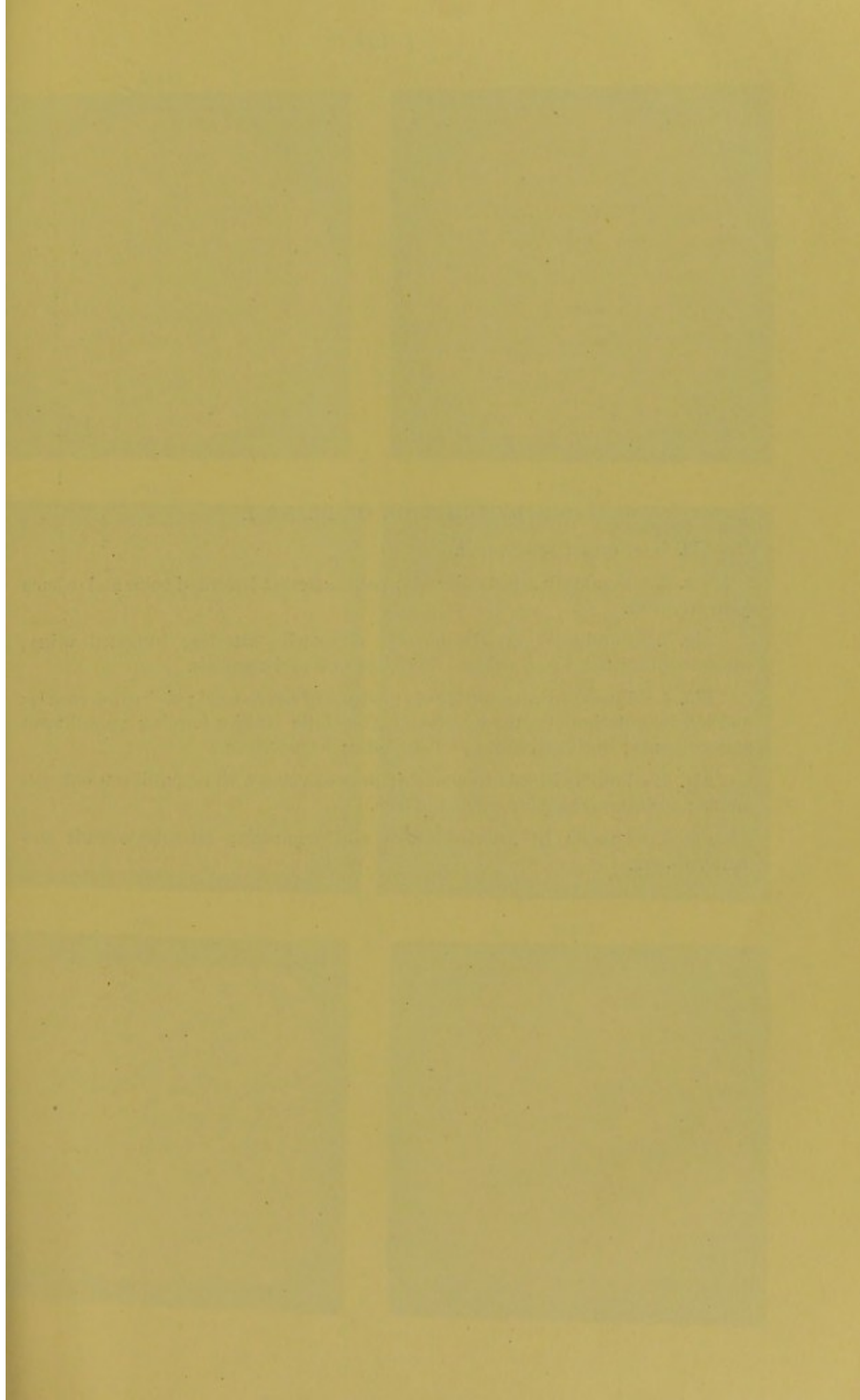
upon the arteries than upon the veins, and, indeed, is often absent as the latter cross the disc, being visible in minor degree where they lie at some distance in the retina. (Plate I, Fig. 1.)

I turn next to a series of slides illustrating the pathological conditions with which we are concerned this afternoon. It is convenient to divide the fundus lesions, which are believed to be indicative of persistent high arterial tension when this is a symptom of arteriosclerosis, into those which are *suggestive* and those which are *pathognomonic*. For their description I take the liberty of quoting from one of my own papers:

“The suggestive signs include uneven calibre and undue tortuosity of the retinal arteries, increased distinctness of the central light streak, an unusually light color of the breadth of the artery, and alterations in the course and calibre of the veins (Fig. 2 of Plate I and Fig. 8).

“The pathognomonic signs include changes in the size and breadth of the retinal arteries of such character that a beaded appearance is produced; distinct loss of translucency; decided lesions in the arterial walls, consisting of white stripes in the form of perivasculitis; alternate contractions and dilatations of the veins, and particularly—and this is the most important of the signs—indentation of the veins by the stiffened arteries in the same manner as a solid rod would indent a rubber tube where lying across it. Sometimes the vein is simply flattened slightly at the point of crossing (Fig. 8), or merely pushed aside, or its calibre is contracted, so that beyond the point of crossing there is an ampulliform dilatation (Plate I, Figs. 3, 4 and 5). In addition to these well-known signs, there may be changes in the venous walls, so that they are bordered with white stripes, and the veins may be exceedingly tortuous and contain varicosities (Plate I, Figs. 3 and 4). Finally, there are œdema of the retina in the form of gray opacity around the disc or following the course of the vessels, hæmorrhages manifesting themselves as linear extravasations, or roundish infiltrations, or sometimes assuming a drop-like form” (Plate I, Figs. 2, 3 and 4).

1. *The Age at which these Signs May Occur.*—As Marcus Gunn has said in his well-known papers on this subject, old age alone does not produce these changes, or does not necessarily produce them in their most elaborate form. Perfectly healthy retinal vessels are



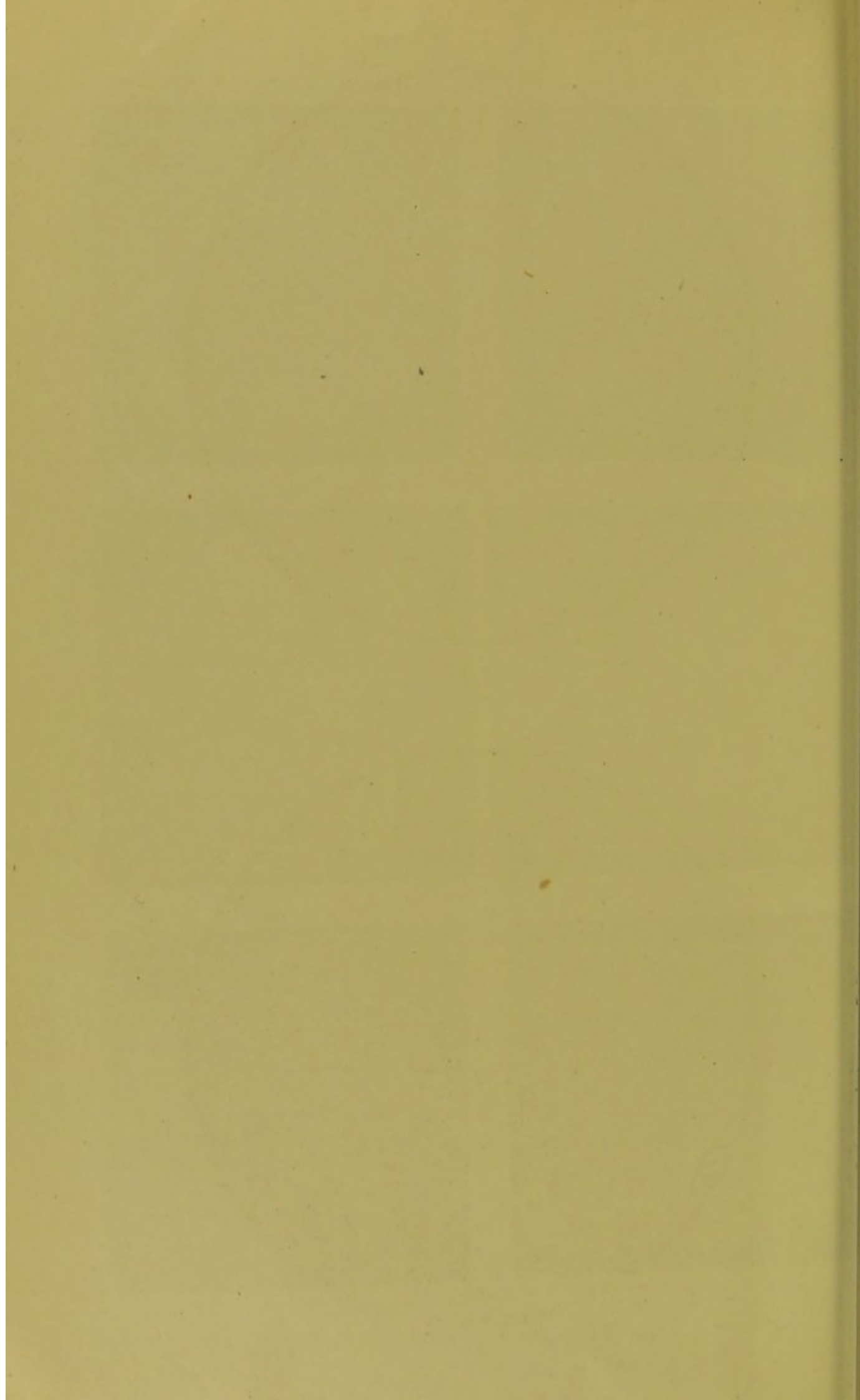
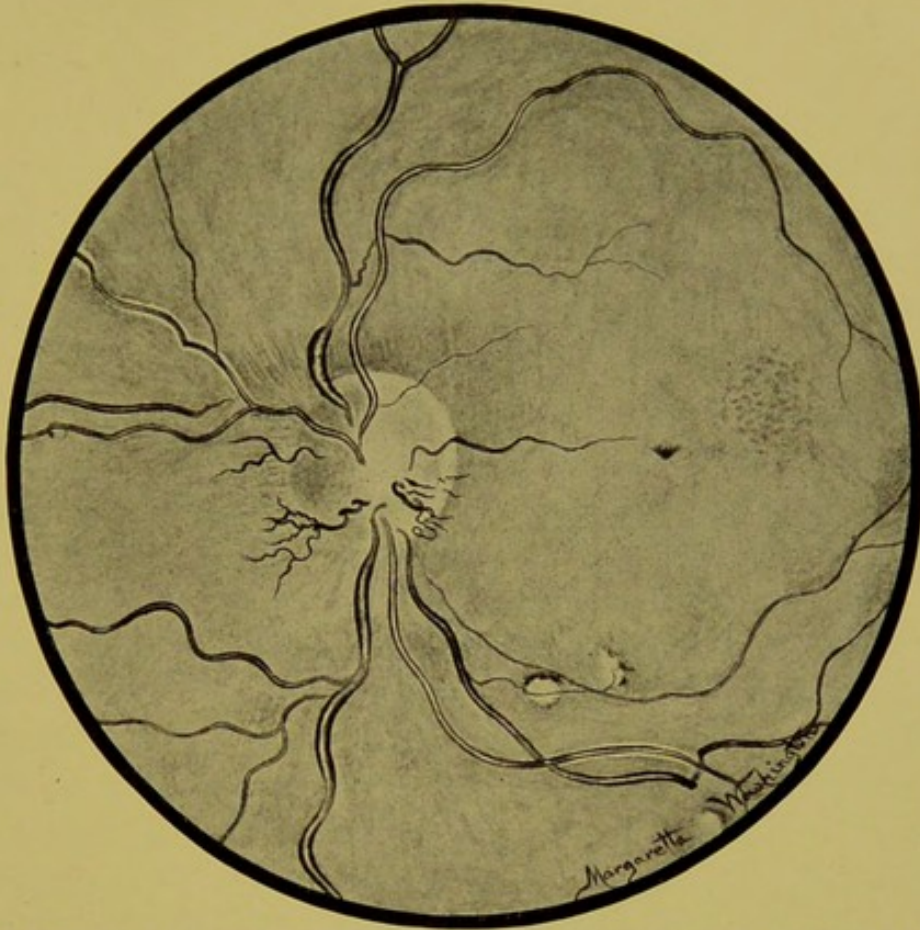


FIG. 7.



Fundus in arteriosclerosis; perivasculitis; silver-wire arteries; indented veins and new-formed capillaries on disc. (After Rachmann.)

FIG. 8.



Early stages of arteriosclerosis of the retinal vessels.



often visible in persons who have reached old age, even seventy or eighty years of life. Doubtless the visible changes, again to quote Mr. Gunn, usually begin about forty and fifty. I have seen them, however, at an earlier age, one of the most pronounced examples being a man aged thirty-seven years. Referring to intra-ocular vascular disease and the nature of the pathological processes at work, George Coats remarks that if all changes which involve thickening of the wall and increased rigidity are included in the term angiosclerosis, it is not entirely a pathological process, but in part a senile change which is naturally present in the vessels of old persons, and in part the result of a pathological stimulus. It is difficult always to separate these two components. Given the stimulus, however, he further remarks, the changes may appear far in advance of the usual age, as, for example, in juvenile nephritis. In other words, as I have elsewhere said, if the cause of persistent high arterial tension is sufficiently prolonged, these alterations may occur at any age at which the pathological condition to excite them arises.

2. *The Earliest Indications.*—The suggestive signs have already been enumerated and must not be disregarded if they are associated with other symptoms of arterial disease. Three signs may be seen early: (a) A markedly corkscrew appearance of certain arterial twigs, either of those which skirt the macula, or, more significantly, of one or more small branches which arise from the larger vessels of the main distribution which themselves are apparently normal. A particularly striking manifestation is one not infrequently seen of a single twig which descends vertically from a transverse branch and assumes this corkscrew appearance. This manifestation is the one to which Alleman has given the name "crinkled retinal vessels," and which he figures in somewhat exaggerated degree in one of his diagrams (Plate I, Figs. 4 and 5). Such phenomena indicate that the whole artery is not affected, made evident, as has been often noted before, especially by Mr. Gunn, not only with the ophthalmoscope, but, where opportunity afforded, by means of the microscope. Thus, Coats points out that serial sections from the small vessels will frequently demonstrate notable differences within a very short space. For example, in the central artery of the retina the region of the lamina is often selected for the endarteritic process, where the vessel may be almost obliterated, although a few millimetres

from this position it is practically normal. What has been termed the irregularity of the distribution of vascular disease in the retinal vessels is, therefore, well known, both from the ophthalmoscopic and the microscopic examinations.

(b) A flattening of a vein where it is in contact with an artery, and at this spot it has somewhat the appearance that a strap would have if laid across a solid tube, provided the vein passes over the artery (Fig. 8). If the artery passes over the vein, the appearance is analogous to that which would be produced if the strap were placed across the lower surface of the tube. The vein is only slightly compressed at this stage, but is not yet indented sufficiently to produce an ampulliform dilatation of the vein beyond the point of crossing. It has seemed to me that this appearance is a little more frequent in the inferior temporal retinal vessel distribution than elsewhere, or, perhaps, to speak more accurately, appears there first.

(c) The nerve-head has an appearance often loosely described as "congested." But this appearance is different from that presented by the so-called streaked hyperopic disc; it is unlike the flannel-red surface of the papilla which merges into an equally flannel-red eye-ground, so commonly the result of prolonged eye-strain, exposure to bright light and intense heat, or seen in certain constitutional conditions; and, finally, may be distinguished from the early stage of neuritis, with the somewhat juicy red aspect of the disc. It is a dull red appearance which is presented, and differs from the franker congestions as the unhealthy flush of a cheek differs from the brighter color of a normal blush. Subsequently the later signs develop—marked indentation of veins, with ampulliform dilatation beyond the point of pressure, varicosities, "silver wire" arteries and perivasculitis, hæmorrhages, etc. (Plate I, Figs. 2, 3, 4, 5, and Fig. 7).

3. *Significance of the Signs.*—The general clinician in his study of arteriosclerosis, and particularly of its early stages, interests himself especially, for the purpose of diagnosis, to quote from Alfred Stengel, with the study of the pulse, with the character of the first sound at the apex and the second sound at the aortic area, with the phymogram, and with the results obtained by instrumental measurement of arterial tension. It is particularly

the purpose of the present lecture to interpret the value of the ophthalmoscopic indications, and their value has not escaped the attention of general physicians. Thus, Stengel has stated that the ophthalmoscope may reveal the positive evidences of vascular disease before angiosclerosis has become marked, and maintains that this instrument may afford an aid in early diagnosis when other methods fail. Moreover, the condition of the pulse, the character of the heart sounds, the increased tidal wave on the sphygmogram and the elevation of tension recorded by the sphygmomanometer may be caused, as Stengel has said, by numerous and varied conditions of the system, organic and nervous in origin, which elevate pressure and in which arteriosclerosis has no part, except, perhaps, as a consequence.

Now, the ophthalmoscopic signs which I have described are sure evidences certainly of local angiosclerosis, and frequently of the persisting high arterial tension either of general arteriosclerosis, or of its manifestations in special organs; and, therefore, eye-ground examination is of paramount importance in the early recognition of vascular disease, and may render signal aid in the interpretation of symptoms caused by the derangement of the functions of important organs, which in their turn depend upon sclerotic changes in their smaller vessels, although there is as yet no decided alteration in the general circulation.

As Rohmer has said, it would be eminently interesting to be able, according to the state of the vessels of the fundus of the eye, to draw a clear conclusion with reference to the condition of the cerebral vessels. Based upon many observations, we suppose that they furnish information on this point; but, as he further states, the question to be decided is if there really is an analogy between the state of the cerebral vessels and those of the retina.

That limitations must be set and reservations made, and that the ocular circulation cannot be regarded as a mere expansion of that of the cerebral in the manner in which the nerve is an expansion of the grain, to use the language of George Coats, is fully recognized. This author insists that disease of the retinal vessels is not positive proof of disease in any other vascular area, although it furnishes a presumption in favor of its existence, a presumption which will be strengthened if any general cause of arteriosclerosis

can be found. He further maintains that the absence of retinal change is no positive proof that other vascular areas are intact, and this is certainly the case, just as the absence of choked disc is not positive proof of the absence of brain tumor. Hertel, although fully in accord with those who maintain that the ophthalmoscopic recognition of sclerotic retinal vessel changes is an important help in the diagnosis of sclerosis of the cerebral vessels, declares that a negative eye-ground examination does not disprove the presence of the cerebral lesions. Thus, he examined microscopically the eyes of a number of old persons in whom no abnormality had been visible with the ophthalmoscope, and yet found angiosclerotic changes in a certain proportion of them; hence his assertion which has just been quoted. Raehlmann, too, who is enthusiastic in his recognition, and most accurate in his depiction of these retinal lesions as valuable aids in the diagnosis to which I am now referring, has never permitted himself from a negative ophthalmoscopic examination to draw the inference that there was failure of sclerosis in the cerebral vessels. He maintains, however, that both affections, that is, cerebral vascular disease and angiosclerosis of the retinal vessels, frequently accompany one another, and as we know from many researches that sclerotic disease of the vessels most frequently attacks alone the carotid distribution, and especially the distribution of the internal carotid, under certain circumstances we are in a position to diagnose dangerous disease of the blood vessels of the brain with the ophthalmoscope, even where the vessels of the rest of the system fail to give indications of this pathological process.

I believe that, in so far as the early signs of arteriosclerosis of the so-called cerebral type are concerned, the ophthalmoscope is apt, at a very early stage, to show the retinal lesions which I have depicted and which are therefore significant. Doubtless there is much difference of opinion in regard to what is meant by a negative ophthalmoscopic examination, and it is for this very reason that I have spent some time in pointing out to you what I believe to be the earliest suggestive signs in the retinal vessels. I have many times, by careful search of an eye-ground, found them when they have escaped my attention on a previous occasion, or the attention of some other examiner; and I believe, with Hirschberg, that these examinations of the eyes of old

persons should be made after their pupils are dilated, not, of course, with any strong mydriatic, like atropin, but with such a drug as euphthalmin, the effects of which can be promptly neutralized with eserine, and, therefore, free the patient from any danger of an attack of glaucoma. No one is prepared to say that because the retinal circulation is apparently normal the cerebral vessels are also normal, but neither should any one pronounce an eye-ground to be normal unless he excludes by such careful examination as I have described all of the signs, not only those which are positive, but those which are suggestive. Certainly we may say, with Marcus Gunn, ophthalmoscopic examination is one of the most ready clinical means for the early detection of important arterial changes. Personally, I think we may go further and say if its findings are positive they are diagnostic.

Evidence is accumulating that it is not alone with the cerebral types of arteriosclerosis that the retinal lesions are found. To be sure, it would require a large amount of research and careful ophthalmoscopic examination in connection with clinical investigation, and also, when opportunity was afforded, with post-mortem examination, accurately to determine the comparative frequency of retinal changes in the groups of arteriosclerosis exclusive of the cerebral types. Concerning some investigations along these lines I shall have more to say in a future lecture. The point evidently is that ophthalmoscopic examination should never be omitted as part of the investigation of any suspected case of early arteriosclerosis, and if indented veins and other ocular signs are present, then its findings are positive and in a certain sense more valuable than any one of the four symptoms now so many times described; and if they, too, are present, the diagnosis becomes practically certain.

In so far as the *prognosis* of intra-ocular vascular disease is concerned, it necessarily is related to that which surrounds the eye itself and its visual properties, and that which affects the general economy.

As you know from previous studies, and as I have already pointed out to you, in the later stages of this condition hæmorrhages frequently appear. If they are centrally placed, their very position interferes with vision, or they may invade the vitreous, may result

in proliferating retinitis, may cause glaucoma, and, if extensive, detachment of the retina. It is not my purpose, however, to discuss ocular lesions this afternoon, except to point out that the frequent association of glaucoma with intra-ocular angiosclerosis, not only in the form which you have witnessed this afternoon but in that variety which depends upon so-called embolism, but, more properly, obstruction of the central artery of the retina, and upon thrombosis or obstruction of the central vein of the retina, must be regarded as more than a coincidence, as recent elaborate pathological investigations have very well shown. The relation of these vascular changes to the development of cataract, to the production of certain types of optic nerve atrophy, and certain varieties of retrobulbar neuritis, represent interesting topics for discussion, which must be omitted, however, from the present lecture.

To one matter, however, I would like to refer, which I have also recently emphasized at the meeting of the American Ophthalmological Society, namely, a curious and persisting form of *asthenopia*, which is observed in patients, frequently women in the late forties, after presbyopia has been established or is already well marked, an asthenopia, moreover, which fails to yield to ordinary optical therapeutics. In other words, these patients are unrelieved by glasses or by treatment directed to unbalance of their ocular muscles. They frequently present the ordinary symptoms of neurasthenia, which in their turn are doubtless the outcome of beginning arteriosclerosis; and it is interesting to observe that this asthenopia disappears, or is often materially relieved, if the high arterial tension which instrumental examination will not infrequently reveal, is reduced to the normal by means of proper dietetic measures, and particularly by the intelligent use of nitroglycerine and allied drugs.

We are interested, however, particularly in the *general prognosis*. As Coats puts it, the finding of retinal vascular disease turns our thoughts not to what is the prognosis with regard to sight, but how far is the disease to be regarded as an indication of the condition of other vessels in the body, and how far, therefore, does it affect the prognosis with regard to the life of the patient.

From the time when Hirschberg first depicted atheroma of the retinal vessels in a patient who died of an apoplexy shortly after-

wards, the intimate connection between these ophthalmoscopic changes and cerebral vascular disease has been forcibly impressed upon the minds, not only of ophthalmologists, but general clinicians. It is no difficult matter to trace the history of patients in whom these retinal vessel changes have been discovered, and find that not infrequently they have shortly afterwards succumbed to a cerebral apoplexy. Moreover, they are not only indicative of arteriosclerosis and of endarterial disease in the cerebrum, but have an important relationship to the study of chronic nephritis, because, as has been well stated, they may be seen in cases in which the usual retinitis has not yet developed. They may be part of the symptomatology of arteriosclerosis, which may be associated with or eventuate in renal sclerosis; and as we know that a large percentage of the cases of chronic interstitial nephritis are primarily arterial in causation, this relationship increases in importance. These retinal vessel changes may be discovered where the usual retinitis may not supervene, because it is possible for them to be significant of a process which may terminate fatally before the development of fully established nephritis.

These facts, and others which I have previously detailed, being accepted, and they are accepted by all careful clinicians, a concluding word should be said in regard to our duty as ophthalmologists in the routine examination of patients who come, often totally unaware of any constitutional disability, provided we find even the earliest indications that I have detailed to you of retinal vascular disease. Alleman has forcibly called attention to this subject; it is plainly pointed out in Mr. Gunn's important paper, and I have tried to emphasize it in what I have written on the subject. In a word, suppose a patient comes for an ordinary change in presbyopic glasses, and has, therefore, reached an age at which vessel degeneration may begin to appear, and the ophthalmologist finds early signs, even though they be only suggestive, of angiosclerosis of the retinal vessels,—that is, a cork-screw appearance of individual branches, a slight thickening of the perivascular lymph sheaths, a beginning brick-dust appearance of the optic nerve-head, a flattening of a vein against an artery (particularly where this appears in the inferior temporal circulation), or a bending in a curve of the vein overlying the artery (a sign which sometimes ante-

dates its flattening or its indentation—a bending as if it had been lifted at that point by the beginning sclerotic vessel), he should not be turned away without more elaborate examination. Under these circumstances he should be sent to his own physician, or to any competent clinician who can carefully examine his cardio-vascular system, with all the modern methods of precision, and particularly with those instruments by means of which the arterial tension is accurately tested. If these signs confirm those in the retina, proper treatment, dietetic as well as medicinal, may save him from the development not only of lesions in his eye, but of lesions in his body elsewhere, and notably of his brain, which, if they occur, may prove fatal.

Time does not permit me to burden this lecture with case histories, but had I sufficient time at my disposal I could impress the lesson by the recitation of many startling cases. My object is accomplished, however, if I have called your attention to these matters, so often described in the articles to which I have made reference, and have made you cautious, not only as ophthalmologists, but, as you ought to be, as doctors, in the thorough ophthalmoscopic and general examination of every case that comes to your consulting room for ocular examination.