Section of Ophthalmology

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Angiomatosis Retinæ. Account of a Case, Including the Histological Results of X-ray Treatment

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THIS communication deals with a case of angiomatosis of the retina in which the large tortuous pathological vessels ended in an ophthalmoscopically invisible "capillary nævus" at or near the macula, no "massive exudate" having formed at the point of junction of the vessels. Fundus drawings show the appearances before and after X-ray treatment, and the histology of the retina and its vessels is shown in photomicrographs of sections obtained after excision of the eye, which was demanded on account of intra-ocular hæmorrhage followed by acute secondary glaucoma. The fundus appearance in this case closely resembled that of a case described by Mr. A. S. Philps at a meeting of this Section on February 10, 1939, and it was referred to in the discussion on Mr. Philps' case. My fundus drawings were reproduced in the *Proceedings*, 1938-39, 32, 1261.

The patient, a young man, aged 19, was first seen by me at the Tennent Institute on December 4, 1936, having been referred by Dr. J. Pendleton White for an opinion regarding possible treatment.

Defective vision in the right eye was discovered after an attack of influenza about September 1936, and he had recently suffered from epistaxis.

Vision in the right eye: + 0·5 D sph./ + 2·5 D cyl. axis 90° = $\frac{6}{18}$; in the left eye: + 2 D cyl. axis 90° = $\frac{6}{12}$.

Ophthalmoscopic examination of the right eye (fig. 1) showed great enlargement and tortuosity of the lower temporal artery and vein, which met and formed a twisted loop

in the midst of an ill-defined, dusky, reddish-grey area as big as the disc and embracing the macula. The two large vessels were similar in colour and calibre, and could not be distinguished as artery and vein. The other retinal vessels were also somewhat dilated and tortuous, but arteries and veins were easily distinguished. The disc and neighbouring retina were œdematous and the disc of somewhat florid colour. There were no hæmorrhages or exudates. The left fundus was normal.

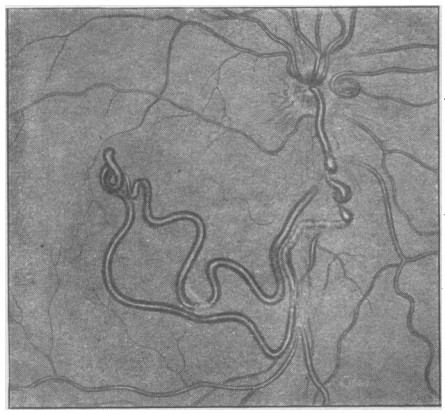

FIG. 1.

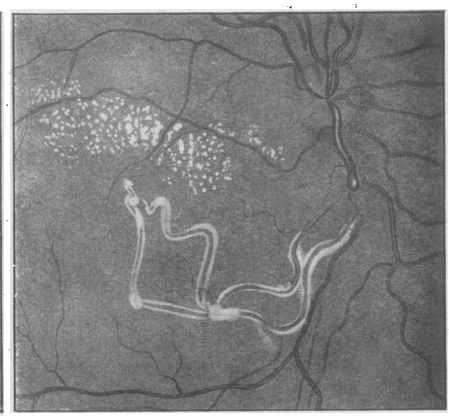

FIG. 2.

FIG. 1.—Angiomatosis retinæ. State of the right fundus before treatment. The pathological vessels are confined to the lower half of the retina.

FIG. 2.—State of the retinal vessels five months after X-ray treatment. The abnormal vessels in the lower half show thickening and opacity of their walls, and narrowing of the lumen.

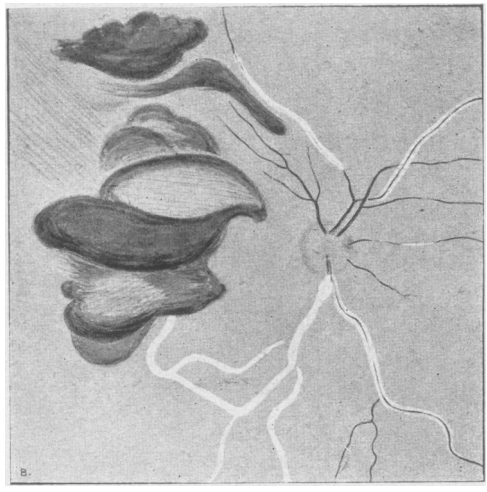

FIG. 3.—State of the fundus in the eye two years after X-ray treatment. (Binocular microscope × 7.) More extensive changes in the retinal vessels, especially in the lower half. Massive retinal hæmorrhages which had caused secondary glaucoma and led to excision.

A systematic general investigation was negative. There was not, nor has there ever been, any evidence of intracranial disease nor of hæmangiomata in any of the organs.

On the assumption that a hæmangioma was situated at the confluence of the two large vessels, four radon seeds giving a total of 4,000-5,000 r were placed on the sclera at the situation of the macula. A month later there was no change in the fundus picture, and radiograms showed that the seeds had not remained fixed in their original positions. He was dismissed, the vision of the right eye being $\frac{6}{36}$ (1 letter). Six weeks later he was readmitted, and X-ray treatment was begun on April 20, 1937.

The X-ray applications were made at three points: above the right eyebrow, near the outer canthus, and in the malar region, directed in each case towards the posterior pole of the eye. There were 14 treatments spread over three weeks and the total dose delivered at the treated zone was 4,750 r.

He was dismissed on May 8, 1937, with a fairly well-marked local reaction, which reached its peak, with redness of the lids and conjunctiva, corneal œdema and some turbidity of the aqueous, about six weeks after commencement of the X-ray treatment.

Eleven weeks after the beginning of the X-ray treatment the first ophthalmoscopic signs of a retinal reaction were seen in the shape of a white lateral sheathing of the central loop of the large vessels. Visual acuity in the right eye was now $\frac{6}{60}$. In about a fortnight a constellation of white spots, like those constituting a "macular star" appeared above the macula, and there was a progressive increase in the extent and width of the vessel sheathing, which throughout the whole period of observation of the fundus, was confined to the large abnormal vessels in the lower half of the fundus (fig. 2). The white spots varied from time to time; disappearing almost entirely for a period. At a later date patches of woolly-looking exudate appeared near the disc. Small patches of pigment on a pale background were also seen in the lower central area.

During the succeeding twelve months he suffered a good deal from recurrent neuralgic pain in and around the right eye, and this was relieved by alcohol injection of the trigeminus. Vision deteriorated and it became more difficult to obtain a good view of the fundus owing to œdema of the cornea, folding of Bowman's membrane, K.P., flare in the aqueous, and opacities in the lens. Other by-effects of the X-ray treatment were: loss of eyelashes, depigmentation of the skin of the lids, telangiectasis of the eyelids and conjunctiva bulbi, closure of the lachrymal puncta and a general rigidity of the eyelids.

On April 2, 1939, two years after the X-ray treatment, there was sudden loss of vision in the affected eye, due to a gross intra-ocular hæmorrhage, and the tension rose to 30 mm.Hg. This increased, and the eye became painful, with hyperæmia of the iris, as well as of the conjunctiva.

The eye was excised, and both the local anæsthetic injection and the removal of the eye were impeded by the very rigid state of the eyelids, the conjunctiva and the orbital tissues.

An artificial eye is now worn, but the shrinking of the skin and orbital tissues gives a sunken appearance to the right eye, and the inelastic condition persists.

There has been no pain during the last two years.

After fixation in formalin the eyeball was bisected in an equatorial direction, and the posterior half examined with the corneal microscope (fig. 3). The main inferior temporal vessels were seen to be represented by apparently solid white bands. One or two others of the inferior branches had a similar appearance while others showed a very narrow blood column heavily sheathed in white. The vessels in the upper half of the retina presented mainly a reduction and variability of calibre with a minor degree of sheathing.

To a large extent the central area of the fundus was occupied or covered by hæmorrhages of various types. Some of these were obviously in the anterior layers of the retina, some in the deeper layers, and others of more massive character either subhyaloid or under the internal limiting membrane. At two points there was a large oval mass of greyish colour with some resemblance to a cyst. This was afterwards found to be a collection of serum, separated from a massive hæmorrhage.

The two portions of the eye were embedded in celloidin and serial sections cut in an approximately vertical direction.

Interest naturally centred for the most part in the condition of the retina and of the retinal vessels; and especially in the nature of the reaction of the pathological vessels and the angioma to the X-ray application.

The histological picture was inevitably disturbed by the gross hæmorrhage, the more so as there was an interval of two months between the occurrence of the intra-ocular hæmorrhage and the excision of the eye. Moreover, for about a year before the excision

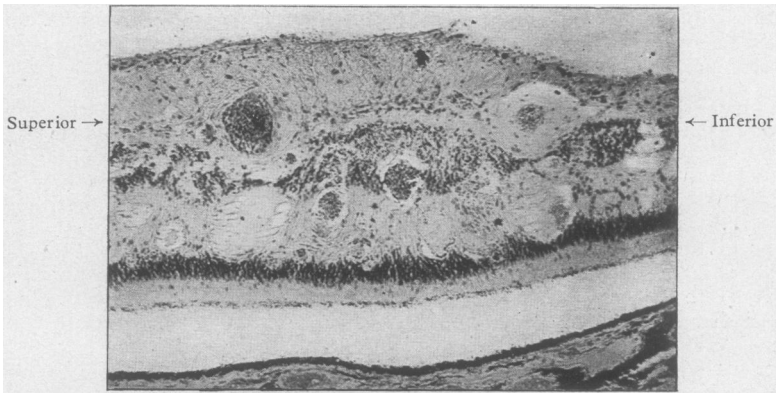

FIG. 4.—Section of retina adjacent to nasal disc margin. Shows contrast between inferior vessel with relatively thick hyaline wall and small lumen, and superior vessel with thick but less degenerate wall and relatively wide lumen. [Low power / Obj. 1″. Oc. 2.]

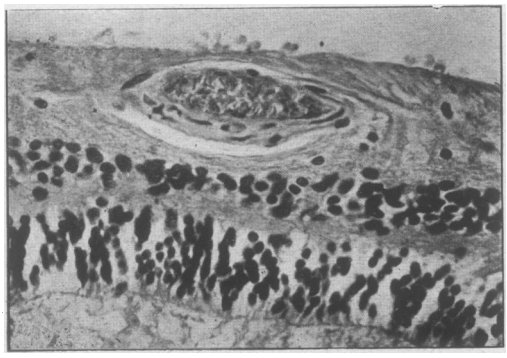

FIG. 5.—A vessel in the upper part of retina; hæmalum and eosin. The wall is thickened but the lumen is wide. [High power / Obj. ⅛″. Oc. 2.]

it was impossible to get a clear view of the fundus, on account of changes in the cornea and lens. But, at the same time, the picture seen in the excised eye by the corneal microscope, helped to bridge this gap, having a sufficiently close resemblance to the ophthalmoscopic drawing, made two years earlier, to enable us to correlate the clinical and the pathological changes.

Perhaps the most interesting and important clinical observation was the fact that the earliest changes seen with the ophthalmoscope occurred in the large pathological vessels;

and, indeed, so long as the fundus was visible there was no *apparent* change in the other retinal vessels. Twelve months later, however, inspection of the excised eye with the corneal microscope showed opacity in the walls of some of these, while sections revealed that there were practically no normal vessels in any part of the retina. The vascular changes, however, were notably more advanced in the lower region. Even the vessels on or near the disc show this contrast between those belonging to the lower and those belonging to the upper parts of the retina (fig. 4).

The clinical and pathological findings make it evident that the pathologically distended vessels were more radio-sensitive than the normal ones, and suggest that a smaller dose of the radiation, or similar doses spread over a longer period, might have produced the desired obliterative change in the big vessels without affecting the normal retinal circulation or producing the other harmful by-effects seen in this case.

One naturally hoped to find some histologically normal vessels and to be able to trace the sequence of changes from these to the completely obliterated vessels; but this was rendered difficult if not impossible by the radical differences between the vessels in the upper and lower halves of the retina above referred to.

It will be remembered that the accepted description of the vessel changes resulting from exposure to radium or X-rays (Colwell and Russ, 1934) is that there is a swelling of the collagenous elements in the vessel walls, proliferation and degeneration of the endothelial cells causing obliteration of the lumen, splitting of the elastic fibres and hyaline degeneration of the muscular coat.

If we examine some of the least abnormal vessels in the upper part of the retina (fig. 5), we find the walls thickened, and already showing a feebleness of the eosin staining, and a reduction in the number of nuclei. A single layer of endothelium is present, and the lumen of the vessels shows little, if any, distension or contraction. Only in a few instances can we find a thin layer of subendothelial tissue inside an internal elastic lamina. At this stage the Van Gieson and Mallory stains give the characteristic reactions of connective tissue; but Weigert's elastic tissue stain shows an increase of darkly staining elastic fibres throughout the whole thickness of the wall (fig. 6). Contrasting this with the vessels of similar size from the lower part of the fundus we find, in the latter, a similar weakness of the eosin staining, the zone nearest to the lumen being paler and poorer in nuclei, while there is a great reduction in the diameter of the lumen (fig. 7). Weigert's elastic tissue stain brings out the fact that the narrowing of the lumen is brought about by the presence of a thick, homogeneous feebly staining layer, within a coat composed of elastic tissue fibres similar to that described in the upper group of vessels (fig. 8).

It would appear then, that at this stage we see a notable swelling and degeneration of the collagenous elements of the outer coats of the vessels mingled with an abundance of elastic fibres, while in the pathologically distended vessels, associated with the angiomatous lesion, there is a greatly thickened intima, already reduced to a hyaline state.

The description already given applies to most of the vessels of the first group; but some of them show a reduction in the fibrillary character of the wall, and a feebler reaction to the Weigert stain, so that the wall throughout its thickness assumes a pale purplish colour and a structureless appearance (fig. 9). The lumen still remains comparatively open and there is no suggestion of the inner coat seen in the vessels of the second group. Among the latter, we can trace a sequence of more and more complete degenerative changes (fig. 10). The two coats remain distinguishable almost to the last stage, the outer one preserving a certain fibrillary structure, while the inner, sometimes separated from the outer by a sinuous refractile line, remains amorphous. Both become progressively paler, nuclear staining disappears from the outer coat, and the increasing thickness of the inner wall causes extreme narrowing of the lumen until we finally get a shrunken solid, hyaline, structure without a lumen and giving merely a pale purplish reaction to the Weigert stain.

There is little or no abnormality of the vessels in and around the optic nerve after it has left the eyeball.

I have made no attempt in this description to differentiate arteries from veins, as it seems to me impossible in these pathological vessels to do so with any certainty.

When the serial sections reach the macular region the retina is naturally greatly dis-

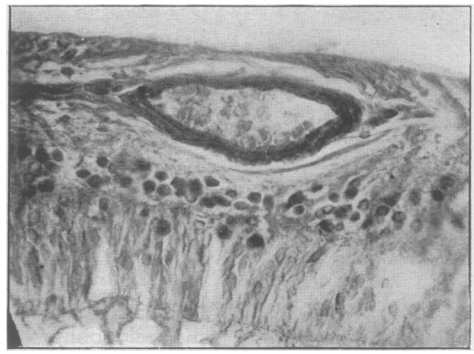

FIG. 6.—The same vessel: Weigert's elastic tissue stain. Proliferation of elastic tissue throughout the whole thickness of wall. No intimal thickening. [High power / Obj. $\frac{1}{6}$". Oc. 2.]

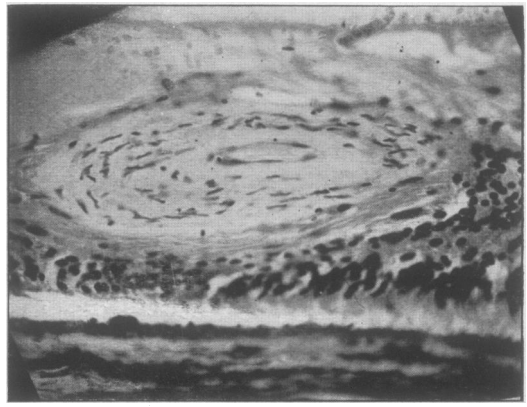

FIG. 7.—Vessel in lower half of retina; hæmalum and eosin. Great thickening of whole wall and very narrow lumen. [High power / Obj. $\frac{1}{6}$". Oc. 2.]

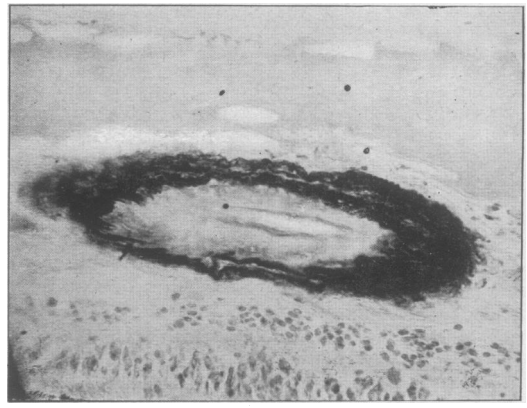

FIG. 8.—The same vessel: Weigert's elastic tissue stain. The wall consists of two portions: an intima, greatly thickened and completely hyaline, and an adventitia composed largely of darkly stained elastic fibres. The lumen is very narrow. [High power / Obj. $\frac{1}{6}$". Oc. 2.]

turbed by the bulky and extensive hæmorrhages and exudates; but we find here a compact mass of fibrous tissue covering an area about 2 mm. in diameter and occupying the nerve fibre and ganglion cell layers; with a maximum depth of about 0·5 mm. In this compact tissue, which stains to a brownish-red colour with hæmatoxylin and eosin, and which appears to be composed of glia, are found many irregularly shaped blood-filled spaces, as well as greatly distended capillaries and small vessels with more or less thickened walls. It seems fairly certain that this highly vascular area represents the angiomatous growth which forms the link between the large tortuous retinal vessels in the lower part of the fundus.

At a point in the lower half of the retina, a considerable distance below the macula, there is a small cluster of greatly distended capillaries in the nerve fibre layer. It is possible that this represents a small rudimentary angioma.

Throughout the greater part of the fundus included in the specimen, that is to say, over an area about 20 mm. in diameter, the retina presents a greater or lesser degree of disintegration. The changes are irregular in their distribution.

On the whole there is relatively good preservation of the retinal layers in the area below the disc and macula—the region of the pathological vessels. The upper half of the retina where the vessels were more normal, was the site of the first exudates (the constellation of white spots) and in the microscopic sections shows greater disturbance of the retinal layers.

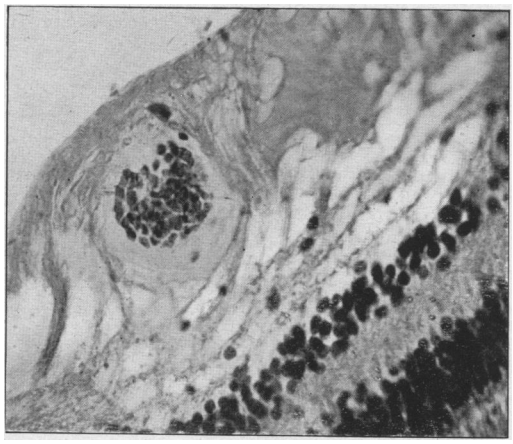

FIG. 9.—The most advanced change in vessels of the upper half of the retina: a thick hyaline wall with a relatively large lumen. [High power / Obj. $\frac{1}{6}$". Oc. 2.]

Œdema of the nerve fibre layer is widespread. Within and somewhat beyond the area disturbed by the hæmorrhage there are round or oval cavities in the inner molecular layer, in the inner nuclear layer and in the outer molecular layer. Those in the two inner layers are for the most part empty in the sections, but those in the outer molecular layer, which may break through into the inner and outer nuclear layers, are in many places filled with red blood corpuscles, albuminous exudate or masses of fibrin, a picture resembling that seen in so-called albuminuric retinitis (fig. 11). This type of lesion is most fully developed in the lower central part of the fundus, where the cavities in the outer molecular layer are very large and are separated from one another by stout columns of hyaline looking material (fig. 12). These spaces break through the inner nuclear layer into the inner molecular, and the nuclei belonging to the former are found scattered irregularly along the columns separating the cavities.

Exudates of albuminous character are found between the internal limiting membrane and the nerve fibre layer, or even extending deeply enough to impinge on the external

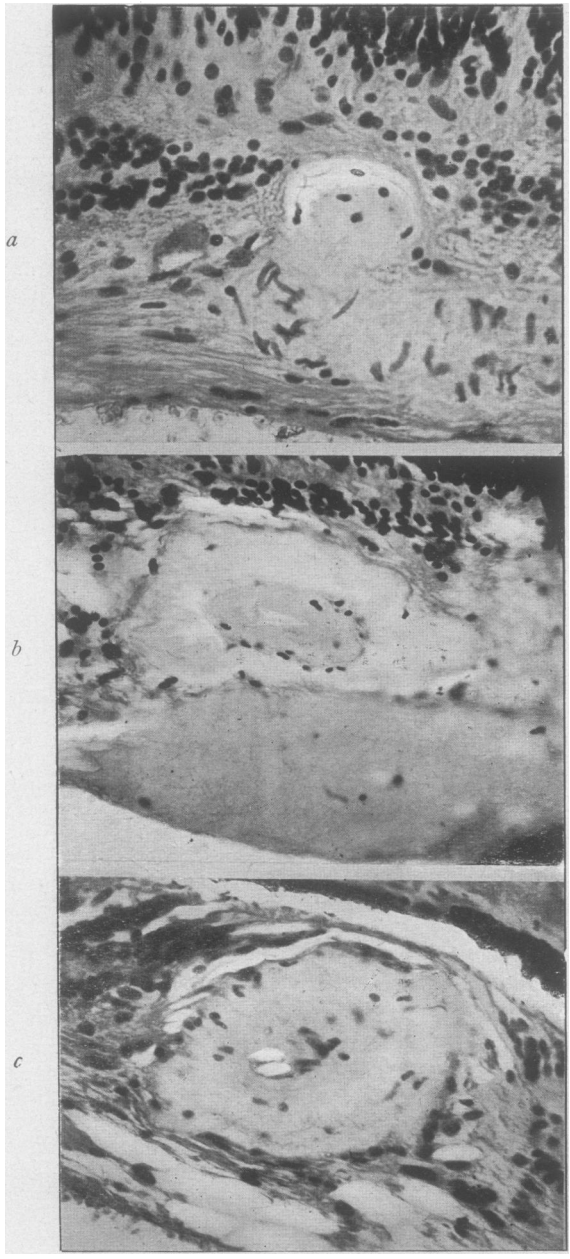

FIG. 10.—Three vessels, *a*, *b* and *c*, from lower half of retina, showing progressive changes. The adventitia, as well as the intima, becomes completely hyaline, the lumen diminishes and finally closes. [High power / Obj. $\frac{1}{6}$". Oc. 2.]

nuclear layer. Similar exudates are found between the rod and cone layer and the pigmented epithelium.

Hæmorrhages are a very prominent feature of the sections throughout the central area of the fundus, and they occur at all depths in the retina. Most prominent of all are those covering the macular region, which the microscope shows to be situated for the most part

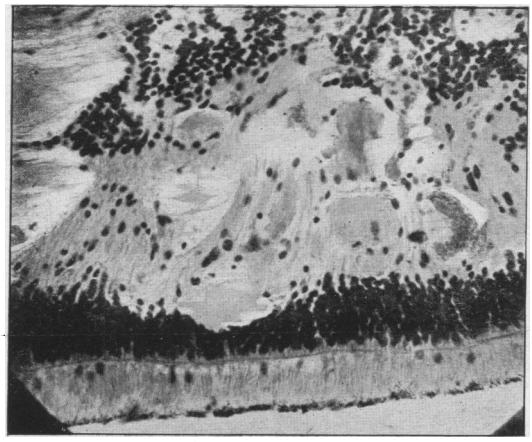

FIG. 11.—Disorganization of the retina, with formation of cavities in all layers, some containing masses of whole blood, serum or fibrin. The external nuclear layer is fairly well preserved but the internal nuclear is broken up and scattered. [Low power / Obj. 1″. Oc. 2.]

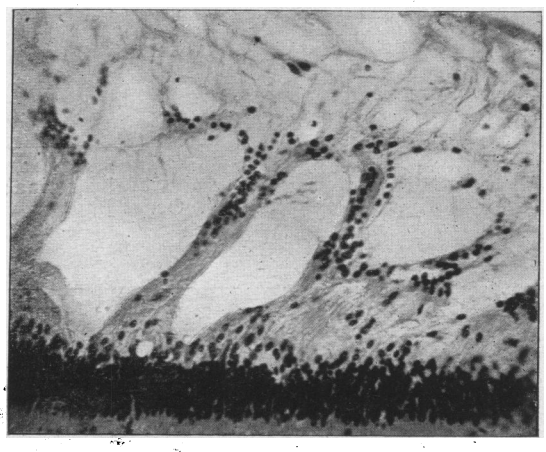

FIG. 12.—Large cavities occupying the external molecular, the internal nuclear and the internal molecular layers. [High power / Obj. $\frac{1}{6}$″. Oc. 2.]

under the internal limiting membrane. In two situations coagulation has squeezed out a serum which lies in front of the massive hæmorrhage (fig. 13) and is represented in fig. 3 by the grey cystic-looking masses with a delicate striation apparently due to traction folds.

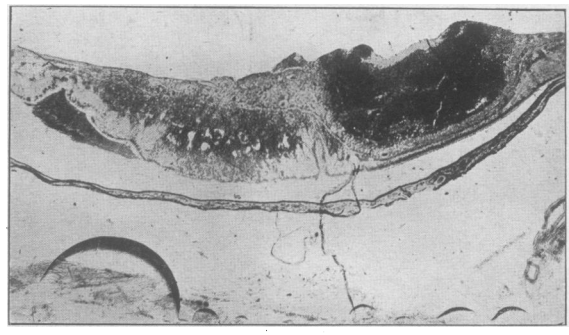

FIG. 13.—Section through the area of massive hæmorrhage, which also contains the angioma. Hæmorrhages are pre-retinal, intra-retinal and sub-retinal. The large dark hæmorrhage is within the angiomatous growth and lies entirely anterior to the inner molecular layer. [Low power / Obj. 1". Oc. 2.]

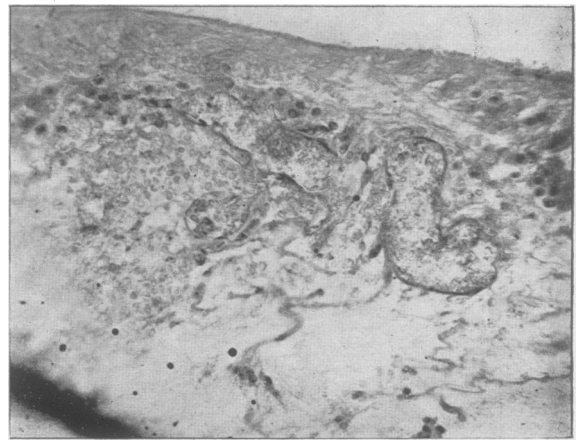

FIG. 14.—Dilated, thin-walled capillaries surrounded by retinal hæmorrhages [High power / Obj. ⅛". Oc. 2.]

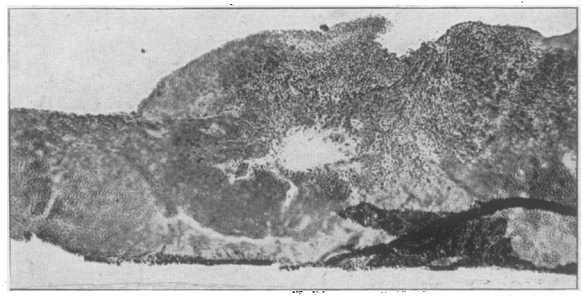

FIG. 15.—Site of the retinal angioma, which is situated within the nerve fibre and ganglion cell layers. Rupture of internal limiting membrane with escape of hæmorrhage into vitreous cavity. [Low power / Obj. 1". Oc. 2.]

In addition to these intraretinal hæmorrhages there are hæmorrhages under the retina (between the rods and cones and the pigmented epithelium) as also in front of the internal limiting membrane. There was, of course, gross hæmorrhage into the vitreous, and a layer of blood corpuscles is found on the anterior surface of the retina in most of the sections. The least disturbed part of the retina is the external nuclear layer which, for the most part, presents a fairly normal and uniform depth throughout the sections. The rods and cones are well preserved in some places, destroyed in others. The ganglion cells in general are either scanty or entirely absent. The retinal pigment epithelium is intact in most of its extent.

It is difficult to judge the condition of the choroid in these formalin-fixed specimens, but it shows variations in thickness and apparently in vascularity from one point to another. In one area about the temporal limit of the large hæmorrhages, and some six millimetres below the macular level, the choroidal vessels have thickened hyaline walls, and over the same area (some 6 mm. in extent) the pigmented epithelium is of very irregular thickness, the retina is thin, with loss of its rods and cones and partial destruction of the other layers, and there is migration of pigment into the atrophic retina.

Far forward, behind the ora serrata, some sections show a collection of " ghost cells ", mingled with pigmented cells (no doubt shed from the hexagonal pigment layer) lying between the rods and cones and the pigmented epithelium.

The optic nerve shows some atrophy of the nerve bundles and thickening of the fibrous septa. In the pial sheath there are many dilated blood-vessels, some with thickened walls.

In sections of the anterior segment of the eyeball, there are pathological changes in the cornea, iris and ciliary body. The corneal epithelium varies in depth and there is some desquamation of the superficial cells. At the limbus there is vascular pannus and round cell infiltration of the cornea and episclera. Over a small area in the lower part of the cornea there is destruction of Bowman's membrane. Descemet's membrane and the corneal endothelium are intact; but there are a few K.P. The ciliary body and processes show some atrophy. There is partial closure of the angle of the anterior chamber. In some of the iris vessels there is a thickened hyaline wall with narrowing of the lumen.

Comments

The occurrence of a capillary angioma of the retina in or near the macula is unusual if not unique. Junius (1930), describing a case in which the angioma was peripherally situated, but caused a central scotoma, remarked that up to that date no case of angioma at the macula had been reported: and I have failed to find any reference to such a case subsequent to the date of Junius' contribution.

It is unusual also for a case to come under observation before the angioma has become visible either as a red vascular nodule or as a pale globular tumour—an angiogliosis of the retina. It is noteworthy that in the present case, even after the vessels had reacted by showing thickening and loss of transparency of their walls there was no visible tumour mass at the macula.

Another case which was exceptional in this respect was that reported by Worms and Pinelli (1930) in which one of the large vessels returned to the papilla and the other seemed to sink into the retina, no angiomatous nodule being visible.

The difference in the reaction of the pathological and the normal vessels to the X-ray application is a point of interest and importance. While a few vessels in the upper half of the retina showed a very thin subendothelial internal coat, there is a very striking difference between this and the greatly thickened and degenerate intima seen in the vessels of the lower half; and it is difficult to believe that the first type of reaction was simply an earlier stage of the second. If we compare vessels from the upper and lower halves whose walls have reached the stage of complete hyaline degeneration we see that the first have still only one distinguishable coat, and an open lumen (figs. 5, 6, 9), while the second have two distinct coats and a more or less complete obliteration of the lumen (figs. 7, 8, 10). The latter condition seems to be that described as an endarteritis (or

endophlebitis) obliterans. The former does not fall into that category, and the sections do not show any transition stages between the one and the other.

The vessel changes in this case do not tally in all respects with those usually described as resulting from experimental exposure of healthy vessels to X-rays. Where cellular and nuclear staining is still present there is no evidence of proliferation of the endothelium. In the vessels of the lower retinal region the thickening which is causing narrowing and obliteration of the vessels is probably a proliferation of the subendothelial intima. Even in the capillaries of the angioma itself, there is no endothelial proliferation.

The demonstration of the histological explanation of the ophthalmoscopically "sheathed" vessels is of some interest. In a joint paper with Michaelson and Heggie (1938) it was shown that a white sheathed vessel, identified microscopically, presented atheromatous thickenings of the intima which had undergone a lipoid degeneration. It was pointed out that one would not be justified in assuming this to be the condition in every case of vessel sheathing, all that could be claimed was that the appearace of sheathing demanded the occurrence of a qualitative as well as a quantitative change in the vessel wall. In other words, a retinal vessel may be represented by a thread-like blood column although the vessel wall is ophthalmoscopically invisible, so long as the wall is thickened by mere increase of its normal structures. "Sheathing" only makes its appearance when these structures undergo hyaline, lipoid or some other form of degeneratino. In the present case there is no patchy thickening of the vessel wall, no eccentricity of the lumen, and no sign of a lipoid change. We find another kind of qualitative change, namely a hyaline degeneration of the whole vessel wall. It is interesting again to note that in many vessels of the upper part of the retina there was sheathing but no thickening of the intima, merely a hyaline degeneration of the thickened adventitia.

While it is possible, in microscopic sections, to identify the angioma at the macula, the extensive hæmorrhage makes it difficult to study this satisfactorily. No clear view of the fundus was obtained during the twelve months before excision of the eye, and, although the view of the fundus of the excised eye given by the corneal microscope was in a sense a substitute for ophthalmoscopic examination at that date, the hæmorrhage which had occurred two months earlier made it difficult to say to what extent the other retinal changes were due to the natural progress of the vascular lesion, the X-ray treatment or the damaging effects of the hæmorrhage.

The simple structure of the angioma serves to bring it into line with the view of E. T. Collins, v. Hippel, Leber and others that the primary lesion is derived from angioblastic sources, and is in fact a vascular tumour of congenital origin. The greater part of the tumour mass at this stage is composed of glia cells and fibres enclosing the wide capillaries and irregularly shaped blood-filled spaces, both lined with a single layer of endothelium. The gliosis would appear to be a secondary change due to local tissue reaction.

With regard to the source of the hæmorrhage, there is no direct association of the hæmorrhages with the larger vessels, indeed there is a comparative freedom from hæmorrhage in the lower part of the retina where these vessels are most abnormal.

Possibly the greater part of the hæmorrhage came originally from the angioma itself, and the neighbouring capillaries. There are many of these greatly distended capillaries buried among the hæmorrhages. Their walls seem to consist of a single layer of endothelium incomplete in some places as if from rupture (fig. 14). At one point in front of the angioma there is a rupture of the internal limiting membrane through which blood has escaped and is lying in a compact mass on the anterior surface of the retina (fig. 15).

In every case of hæmorrhage the attempt to explain its occurrence requires the consideration of three possible factors: the condition of the blood, the state of the vessel walls and the height of the blood-pressure. Most, if not all, of the hæmorrhages in this case are seen in similar forms in cases of hypertensive disease, with or without renal involvement; but it is very doubtful if the hypertension *per se* is an important factor. In the present case, there was neither hypertension nor any physical defect in the blood. It seems natural to attribute the hæmorrhage to the state of the blood-vessels. It is generally believed that the glial and connective tissue proliferation which produces the characteristic "massive exudate" in Coats' disease may be the result of sudden or gradual escape of blood from congenitally fragile vessels; and it is not improbable that in such

a case as this, a globular mass would have developed in the course of time in a similar way. Moreover, one of the early results of experimental radiation of vascular tissues is destruction of the endothelium of the vessels. And the integrity of the walls of the capillaries in this case would be further damaged by the anoxia resulting from impeded circulation in the partially obliterated vessels.

In the presence of hæmorrhages and exudative changes closely resembling those found in hypertensive retinitis it is surprising that there is almost complete absence of the so-called "ganglioform degeneration" of the nerve fibre layer.

I should like to express my thanks to Dr. J. Pendleton White for his courtesy in sending this case for investigation, and for giving permission for its publication.

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Discussion.—Dr. BALLANTYNE in reply to the Chairman said that originally the tension was 30 mm. in the affected eye, but it went up after that. The tension digitally, as compared with the other eye was very high—probably in the region of 40 or 50.

Mr. A. SEYMOUR PHILPS showed two coloured drawings (fig. 1) of the eyes of a patient which had been shown at a meeting of the Section in 1939 (*Proceedings*, **32**, 1260). She was a woman of 35, with an arteriovenous communication in each eye. She complained very much of headaches and at a later stage of noises in the head. When the case was

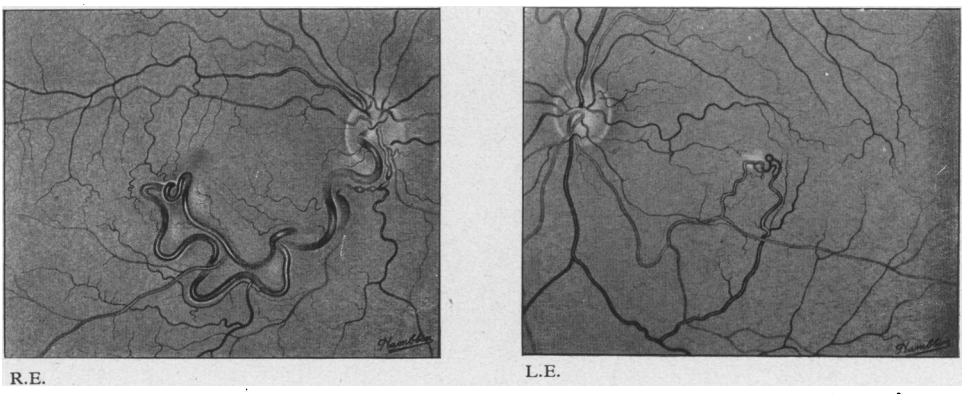

FIG 1.—Right and left fundi showing arteriovenous communication in each.

shown to the Section some members recommended diathermy, but others urged that it be left alone. The case was left alone, and the vision, which had been $\frac{6}{9}$ in each eye, became $\frac{6}{6}$. It was almost impossible to distinguish the arteries and veins except in so far as the branches of the vessels were not affected. In one eye the condition was not quite so marked as in the other at that time, but six months later she came back because vision in that eye had gone down suddenly; it was in fact $\frac{6}{60}$ and there was white exudate at the arteriovenous communication. No treatment was given, the patient was told to go home and rest. The vision improved to $\frac{6}{12}$ and there it remained, he believed, at the present time. The patient still complained of rhythmical noises in

the head, and had been twice admitted to hospital for a general neurological examination. The surgeon was now of opinion, however, that there was no general neurological lesion. The patient had listened so often to the descriptions of symptoms from which she should be suffering, that she had learnt them by heart.

Dr. BALLANTYNE said the point Mr. Philps raised about the vision having gone down owing to the presence of exudate, and the exudate disappearing, was rather interesting, and it would be useful to know what the precise reaction was, because nothing of the kind had been described so far. In most of the cases hitherto described and illustrated there had been a nodule at the confluence of the two big vessels, which no doubt began with the simple angioma. But what the explanation was of this transient change of the macula it was very difficult to say. Mr. Philps would of course follow up his case, and see what other changes took place.

In reply to Mr. R. H. Bickerton Dr. Ballantyne said that in his case there were quite clearly no similar changes in the other eye.