a patient on whom I operated for this more than twelve years ago. Previously to the operation he had headache without intermission for seven years, dating from typhoid, and he has had no headache since. But these operations are extremely tricky, and in higher heterophoria it is rather a responsibility to convert a comitant defect into a non-comitant one by operation. Here again, however, nature comes to the rescue and gradually restores more or less comitancy if a muscle is only tenderly dealt with by operation, on the principle of "reversion to organism," as we might call it.

The most successful surgeon is the one who understands nature best. You may perhaps wonder that I have said nothing about the evolution of the oculo-motor apparatus, but, Gentlemen, when I look at your intelligent faces, and the soul in your eyes, I cannot bring myself to believe that you are descended from maggots, and spiders, and pigs. The whole voice of nature, as I hear it, is against that fantastic theory. Nature is conservative and not progressive. She reverts to type as soon as the reason for departure from it disappears. When I see a piano, I do not at once jump to the conclusion that because the keys are in progressive series, therefore they must have evolved out of one another!

I thank you, Gentlemen, for your kind attention, and I have now the pleasure of laying this imperfect lecture as a little memorial wreath upon the tomb of our friend and benefactor, Robert Walter Doyne.

THE PATHOLOGICAL ANATOMY OF RETINAL DEGENERATION WITH MULTIPLE ANEURYSMS*

BY

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LEBER has already given a clear account of the clinical side of cases of retinal degeneration with multiple aneurysms with reference to some thirteen cases previously recorded^{1, 2}. On the anatomical side we have, however, but little knowledge, the only published case being that recorded by Morton and Coats³; and this only came under examination several months after complete blindness had resulted.

We have lately had the opportunity of examining a comparatively recent case of this rare disease. The clinical history of the case was as follows:

T. O., a boy of 15, in a school in Japan, was first seen on

*Based upon a paper read by Dr. S. Miyashita before a meeting of the Japanese Ophthalmological Society, on April 3, 1919.

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FIG. 1.—Fundus oculi of right eye as seen by the indirect method. Shows large tumour-like prominence and massive exudation.

FIG. 2.—Shows the tumour-like prominent part of retina containing a large number of enlarged and convoluted blood vessels.

FIG. 3.—Shows the same structures as in fig. 2, but more highly magnified so as to show the fibrin in the outer layers of the retina and the granular fat cells in the subretinal exudation.

FIG. 4.—Shows the enlarged blood vessels and hyperplastic connective tissue.

January 7, 1918. Neither his family nor his personal history gave any evidence of constitutional disease. At the age of three he was found to be shortsighted. In April, 1917, the right eye failed and developed a central scotoma. Under treatment this improved, but relapsed in October of the same year.

On examination in January, 1918, the patient was found to be moderately nourished, with no signs of any constitutional disease. Wassermann reaction was negative, but v. Pirquet's reaction was slightly positive. Both eyes were myopic. R.E. vision, fingers at 2 metres, after correction, (-1.75 D.), 0.1. L.E. vision, 0.1., after correction, (-1.5 D.), 0.9.

The left eye was, apart from the myopia, normal in every respect. The pupillary reaction of the right eye was sluggish and the fundus showed the following changes. Optic disc was hyperaemic and not sharply defined. The main central blood-vessels appeared normal, but the peripheral portion of the superior and inferior nasal and temporal vessels was dilated and convoluted. In the retinal periphery, particularly along the upper and lower temporal vessels, there were present a large number of small red globes like aneurysms, but none were to be found on the nasal side. The majority of these globes were round, but some were elliptical or irregular. The larger ones measured from one-third to one-half the diameter of the papilla, and two of them situated on the periphery of the superior temporal vein and several in the lower temporal periphery had a white spot in the centre. The majority were much smaller, at times set in a row, and at other places more or less scattered. One very conspicuous one, as large as the optic disc, was attached to the peripheral part of the superior temporal vein. Its centre looked gray. The vein seemed to pass through it. The red globes as a rule were mainly seen along the arteries.

In addition to the red globes, retinal exudation resembling the clouds in a summer sky was found in large white patches of a silvery brilliancy. Between the larger patches there were seen thousands of small brilliant white spots. None of the exudation obscured the retinal vessels which were to be seen passing over it, though in places the vessels were sheathed by white lines for a short distance. Near the termination of the inferior temporal vessels, where the red globes described above were most numerous, the retina was raised in the form of a tumour with a prominence of seven dioptries. This prominence was elliptical in shape, of pinkish colour with some haemorrhages on the surface. The visual field was contracted but the colour sense was normal.

The patient was admitted to the hospital on January 14, 1918, and on the following day was given a subcutaneous injection of 0.1 c.c. of 0.1 per cent. T.O. - This was not followed by any reaction.

January 19. Intraocular tension R.E. 16 mm. Hg. (Schiötz), L.E. 20 mm. Hg. (Schiötz).

January 20. 0.2 c.c. of 0.1 per cent. T.O. administered. No general reaction, but some increase of the retinal exudation.

January 22. 0.2 c.c. of 0.1 per cent. T.O. No reaction.

January 26. 0.4 c.c. of 0.1 per cent. T.O. Increase of retinal exudation. Temperature rose to 38.3C. with shivering and headache.

February 2. The retinal exudation was still increasing and the tumour previously noted was larger. V. with — 2D., fingers at 2.5 metres.

February 5. The right eye was removed and immediately fixed in Zenker. It was embedded in celloidin and cut in serial sections.

Pathological examination showed the following result. Cornea, iris, ciliary body, lens and optic nerve were normal.

Choroid.—All blood vessels extremely dilated and the intervascular tissue slightly loose, so that the choroid, as a whole, was thickened. In the connective tissue were numerous mononuclear cells containing eosinophile granules, and, in places, round cells formed a perivascular infiltration. Hyperaemia was marked, but no distinct signs of inflammation were noted.

Retina.—The subretinal exudation consisted mainly of coagulated albumen containing numerous mononuclear granular fat cells and, also, a few polynuclear leucocytes, some blood-vessels, and a little fibrin. The amount of exudation was variable but never entirely absent. Where the exudation was most marked the retina was pushed forwards towards the vitreous, the edges of the exudation being always occupied by the granular fat cells. Near the ora serrata on the temporal side the retina was conspicuously thickened into the form of a tumour-like prominence.

The retinal structure showed the five following types of alteration.

1. Oedema of the retina was limited to the prominent part noted above; here the loose connective tissue showed cavernous spaces.

2. Hyperplasia of connective tissue was also limited to the prominent part of the retina. The fibrous tissue was of recent formation and consisted of spindle-shaped cells crossing in all directions and containing dilated blood-vessels.

3. **Blood-Vessels.**—Chiefly in the inner layers of the retina, and associated with the hyperplastic connective tissue described above, there were present numerous closely set dilated blood-vessels. These appeared in section either round, elliptical or kidney shaped, and sometimes presented an appearance like that of a cavernous angioma. Thrombi were to be found in the larger vessels sometimes completely occluding them. Along the inner wall of the vessels polynuclear leucocytes were generally to be found. The vessel

walls were thin and showed hyaloid degeneration and infiltration with fibrin and mononuclear lymphocytes. These latter were sometimes found surrounding the vessels, sometimes also infiltrating the entire section of the vessels and in other places more localized. These changes were found in places even where the retina was not prominent. The lymphocytes were sometimes found associated with leucocytes containing eosinophile granules. The enlarged vessels contained no elastic fibres, but these were present in the unchanged vessels even close to the altered vessels.

Summary of vascular changes.—Aneurysmal enlargement and convolution of small vessels in retinal periphery, infiltration by lymphocytes, formation of fibrin, destruction of elastic fibres, and formation of thrombi. All the retinal capillaries in the neighbourhood of these changes were enlarged.

4. **Haemorrhages.**—These were found along or near the dilated vessels in both the inner and outer layers of the retina, spreading between the hyperplastic connective tissue fibres.

5. **Fibrin.**—The retina contained a large quantity of fibrin, which was to be found in smaller quantity far from the haemorrhages and reaching only to the internal nuclear layer. The granular fat cells mentioned above were usually to be found in the outer layers of the retina. In the prominent part of the retina polynuclear leucocytes were also to be found.

As a result of these pathological changes in the retina the rods and cones were destroyed by the invasion of fat cells and fibrin, especially in the prominent part. The internal nuclear layer was normal except for the presence of the aneurysmal vessels. There was some hypertrophy of the glia fibres.

On comparing the ophthalmoscopic appearances with the result of the microscopic examination we note the following points.

1. The prominence noted in the retina corresponded to the portion found thickened and pushed forwards by the exudation in the subretinal space.

2. The red globes found on ophthalmoscopic examination were made up of the enormously dilated vessels and of the haemorrhages into the perivascular lymph spaces together with the thrombi.

3. The large white exudation corresponded to the granular fat cells lying along the edge of the subretinal space.

4. The smaller white spots were probably due to small collections of fat cells in the layers of the retina.

5. The sheathing of certain blood-vessels was probably caused by the infiltration of the vessel walls with round cells and granular fat cells.

To sum up the pathological changes observed. These all took place in the lower temporal periphery of the retina near the ora serrata. They consisted in definite degeneration of the vascular

wall with dilatation and convolution of the vessels. This was followed by thrombosis in the interior of the vessels, hyperplasia of the connective tissue and haemorrhages in the inner and outer layers of the retina, destroying the *membrana limitans externa*. The extravasated blood, almost completely defibrinated, spread between the retina and choroid. The retinal changes were caused, in our opinion, by the fibro-angiomas. They were similar to those described by Coats and Morton³. Hata⁴ reported this year a case of this disease from Tokyo University. The retinal changes observed by him only differed from those found in our case in the localisation of the new-formed connective tissue, this being found at the macula as well as the periphery. The choroid was more affected than in our case.

The amount of exudation in the subretinal space seems to indicate some inflammatory change, but we consider that this may really be entirely secondary to the degeneration of and thrombosis in the blood-vessels which would produce a violent disturbance of the circulation resulting in haemorrhage, and transudation and migration of granular fat cells, etc. Coats (l.c.) considered that the haemorrhage in the outer retinal layers was the primary change and was followed by hyperplasia of connective tissue fibres. We found no evidence of the organization of haemorrhage, but observed haemorrhage spreading between the hyperplastic connective tissue fibres. Moreover, the haemorrhage in our case was not confined to the outer layers, but also was found in the inner layers of the retina. Nor are we able to agree with Leber in considering embolism to be the cause of the aneurysmal formations. If we assume that the disease is of an inflammatory nature, tuberculosis appears to be the most likely origin, an hypothesis suggested by Leber. We have ourselves seen a case of retinitis undoubtedly tuberculous in origin, in which the appearances were very similar to those just described. It is noteworthy that the disease usually begins near the *ora serrata*, just where the blood stream is at its slowest. It was this fact that suggested to Leber the probability of embolism being of aetiological value.

We consider that the disease may be traced to a hereditary weakness of the blood-vessels. At a comparatively early age degeneration sets in in the smaller vessels, and, as a result, new and convoluted vessels of an inferior type of structure are formed. If the aneurysmal dilatations observed were due to some primary change, we should expect to find some evidence of destroyed elastic fibres; such evidence was completely absent.

We consider that the evidence points to the disease being of the nature of an angio-fibromatosis followed by a general degeneration of the retina. We are thus able to bring our case into close relationship with the disease known as *v. Hippel's disease*.

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DECENTRATION AND OBLIQUE CYLINDERS

BY

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WHEN prescribing spectacles it is frequently necessary to give a correction for a hyperexo- or a hypereso-phoria, and there are three ways of doing this.

The simplest, and the worst, way is to correct the hyperphoria in one eye with a vertical prism, and to correct the lateral defect in the other eye with a lateral prism. Suppose for instance that a patient needs a correction of 2∇ for a right hyperphoria and a correction of 3.5∇ for an exophoria; he might be given a 2∇ prism edge up before his right eye, and a 3.5∇ prism edge out before his left eye.

FIG. 1.

The second method is to divide the necessary correction between the two eyes. As is well known, prisms obey the parallelogram law, i.e., a prism set obliquely at an angle α with the horizontal may be resolved into two components:—(Fig. 1.)

V the vertical component, where $V = P \sin \alpha$.

H the horizontal component, where $H = P \cos \alpha$.

We are given V and H, and we have to find P and α . Of course

$$P = \sqrt{V^2 + H^2}, \text{ and } \tan \alpha = \frac{V}{H}$$

A simple and sufficiently accurate way of doing this is to draw AB horizontal representing in direction and units of length