ON SOME POINTS
IN THE
PATHOLOGY AND MORBID ANATOMY
OF
GLAUCOMA.

. BY
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Received Nov. 26th.—Read Dec. 12th, 1857.

THE practice of excising the eyeball in hard and painful conditions of the globe, when all sight is lost, and when the diseased state of one eye places the other in jeopardy, was, I believe, first brought into practice in the Royal London Ophthalmic Hospital, in the winter of 1854-5.

Since then this operation has come into more general use; and I have had frequent opportunities of examining and dissecting eyes affected with glaucoma, directly after extirpation and whilst they were in a perfectly fresh state.

I wish to offer to the Royal Medical and Chirurgical Society a short record of the symptoms, the ophthalmoscopic phenomena, and structural changes, which have presented themselves in a large number of cases of this disease. Many have occurred in the Moorfields Ophthalmic Hospital, and I am glad to embrace this early opportunity of expressing how greatly I am indebted to the hospital staff for the courtesy and liberality which have enabled me to prosecute these investigations. I feel also great pleasure in expressing my deep obligations to Mr. Bowman, who has kindly permitted me to examine many cases which have occurred in his private practice, and given me many valuable suggestions.

Authorities have long recognised two varieties of glaucoma—the acute and the chronic, and this division has been adopted by Gräfe, in an excellent memoir on the ‘Treatment of Glaucoma,’ addressed to the French Institute.¹

Acute Glaucoma.—The symptoms of acute glaucoma have been so often and so graphically described, that I shall only briefly allude to them. They are, in short, violent pain in the eyeball; rapid blindness; a widely dilated, fixed pupil; a peculiar hazy greenish appearance of the lens, called glaucomatous; and great hardness of the globe.

The ophthalmoscopic phenomena are: a dilated state of the retinal veins, often tortuous and turgid with blood; small ecchymoses scattered over the surface of the retina; occasionally small blood-clots in the vitreous humour; pulsation in the central artery of the retina; and an excavated state of the optic papilla.

The pulsation of the central artery in glaucoma, and the excavated state of the optic papilla, were, I believe, first noticed by Gräfe, who insists upon their being pathognomonic symptoms of this disease. The pulsation in the arteria centralis is synchronous with the pulse at the wrist; I have counted it, beat for beat, with my finger on the radial artery. Gräfe points out that, when not present spontaneously, it may usually be produced by pressing on the eyeball with the finger. The cupped, excavated state of the optic papilla, persists for some time after removal of the eyeball. I found it well marked in an eye I dissected on 24th March, 1857 (in this case also the turgid retinal veins seemed to stop short at the margin of the optic papilla, whilst the arteries could be traced across the margin converging to a single trunk at the usual point of entrance).

In acute glaucoma the urgent symptoms often set in most suddenly, but a careful inquiry shows that, in a large number of cases, the sudden outbreak of the disease has

¹ ‘Note sur la guérison de Glaucome au moyen d’un procédé opératoire, adressé à l’Institut de France,’ par le Docteur A. de Gräfe.

been foreshadowed by a set of premonitory symptoms, which may, however, have been so slight as not to have excited the serious fears of the patient. These symptoms are:—occasional dimness, usually towards evening; shooting pains in the eyeball; *muscæ*, and sparks of light. Gräfe also calls attention to the diminished size of the field of vision. He thinks these prodromic symptoms occur in about three fourths of the cases. There are, however, cases in which these premonitory symptoms have not been observed. After some time, the sudden outbreak may be followed by a remission; the pain abates, and the patient may be able to see large objects, though indistinctly. We must not be deceived by this improvement; it is only a lull in the disease, and is sure to be followed by fresh paroxysms, which will sooner or later end inevitably in blindness. As the disease advances, large turgid veins appear upon the sclerotic, emerging in the ciliary region, and running backwards in a tortuous course. The iris loses its brightness, (in a case under Mr. Bowman's care in the Moorfields Hospital, March, 1857, a large dilated vein was noticed upon the iris,) and the aqueous humour has a yellow tint. The lens becomes opaque and swollen. The cornea loses its lustre, and becomes dim; its epithelium is raised in minute vesicles, and sloughing may take place: (this mode of termination was well exemplified by a case treated by Mr. Bowman at Moorfields, in June, 1847.) Gräfe has drawn attention to the insensibility of the cornea in glaucoma.

At a further stage, in many instances, sclerotic staphylomata occur; and these take place precisely in the situations where anatomical considerations would lead us to expect them; they are most apt to occur just behind the insertions of the tendons of the recti muscles, a part where the sclerotic is naturally thinnest. In one case I examined, (19th September, 1856,) there was so large a staphyloma beneath the internal rectus, that, at this point, the diameter of the globe from side to side exceeded that from back to front by nearly one third.

In another class of cases, after a long time, the hardness of the globe diminishes; the pain lessens, and then occurs only occasionally; the eye becomes quiet, and finally undergoes atrophy.

Chronic Glaucoma.—This differs from the acute chiefly by the insidiousness of its course, which is unmarked by those violent symptoms which characterise the outbreak of the acute form. It is equally intractable as the acute affection, and, like it, is sure to end in blindness. Whilst the transparent media are still clear, small hæmorrhagic spots may be seen with the ophthalmoscope upon the retina, just as in acute glaucoma, and the retinal veins are equally turgid. Another argument for supposing chronic and acute glaucoma to be merely different forms of the same disease, is the occasional occurrence of both in the same individual. An instance of this is now under treatment at the Moorfields Hospital.

Gräfe describes a form of amaurosis in which the optic papilla has an excavated appearance, but the other symptoms differ from those of glaucoma. The two affections are not identical, and must be carefully distinguished.

Morbid Anatomy of Acute Glaucoma.—In a large number of dissections of eyes affected with acute glaucoma, I have found the following morbid appearances uniformly present in a more or less marked degree.

Vitreous humour.—The vitreous humour is transparent and colourless, or it has a yellow tinge. According to Gräfe, it is, at first, yellow, and, subsequently, becomes colourless. It often contains blood-discs entangled in a delicate web of coagulated fibrine. The blood-corpuscles may be observed in all stages of withering. I have sometimes seen small filmy blood-clots imbedded in the vitreous humour, and tied by a slender point to the retina at the spot where the ruptured membrana limitans has allowed the blood to pass from the retina into the corpus vitreum. The vitreous humour has a much greater consistence than it naturally possesses; it does not readily flow away when the eyeball is

cut across, and it offers remarkable resistance when cut. With a view to relieve the tension of the globe, I have seen the sclerotic freely punctured with an extraction knife, after which, firm counter-pressure with the finger upon the opposite side of the globe only caused the protrusion of a very small bead of yellowish vitreous humour: such great firmness had it. The yellowish colour of the vitreous humour is derived from the colouring matter of the effused blood which soaks through it and stains it.

Retinal Tissues.—The veins are dilated, tortuous, and filled with blood. The inner surface of the retina is sprinkled with small ecchymoses, which, in point of fact, are small spots of capillary hæmorrhage. I believe this condition was first recognised by actual dissection, in glaucoma by Mr. Bowman, in a case which occurred in his practice in May, 1856. The hæmorrhage comes from the capillaries in the inner layers of the retina, and the effused blood either spreads laterally among the elementary structures of the retina, or bursts through the hyaloid membrane into the vitreous humour. The retinal capillaries are irregu-

larly dilated, and studded with small fusiform and globular enlargements, miniature aneurismal pouches. I have, now and

then, seen them on small vessels one remove from the capillaries ; but never, in a single instance, have the larger vessels and great trunks been affected in this way. The dilatations sometimes occur at the branchings of the vessels, and sometimes at intermediate points of their course.

These pouches, and the vessels communicating with them, are usually crammed full of blood-corpuscles. In the hæmorrhagic spots, the tissues immediately around the

dilatations are infiltrated with blood-discs, which have escaped from some of the pouches which have burst, and they are stained with blood-pigment to a still greater distance.

Excepting these dilatations, the retinal capillaries have a healthy aspect, and they do not present any traces of fatty or atheromatous change. The coats of the small arteries are hypertrophied, and the nuclei in their walls are highly developed. The other retinal structures retain a natural appearance during the early stages of this disease.

Lens.—The lens during life had a greenish glaucomatous appearance, but it now has a yellow tint when viewed by transmitted light. The bedding of the lens in the hyaloid fossa of the vitreous humour, and the thinness of the posterior half of the capsule, are circumstances favorable to the existence of osmotic currents between these two structures, and it is probable that they both derive their colour from the same source, viz., the hæmatine of the blood.

Choroid.—Besides fulness of its vessels, I have not found any morbid changes in the choroid, excepting when staphylomata have occurred beneath the recti muscles. In an eye which I examined in September, 1856, there was a very large staphyloma beneath the internal rectus, and a smaller one under the outer rectus. In this instance, the choroid corresponding to the staphylomata was pale; the pigment was irregularly distributed; and the tissues were opened out, if I may so express myself. The choroid preserved its natural connection with the sclerotic and retina respectively; and these membranes were not separated by any collections of fluid. The vitreous humour was firmer than natural, and more abundant; it contained small blood-clots, and had a yellow colour. The retina was marked with numerous ecchymoses. In this instance, then, in which large staphylomata were present, the choroid and retina were not separated by any serous effusions. The examination of several subsequent cases has given similar results; and I believe that in glaucoma, in the acute stages at least, serous effusions between the choroid and retina never occur.

The subsequent changes in the choroid and retina in the advanced stages of the disease have an atrophic character. In some cases I minutely examined, in the summer of 1856, I found the dilated capillaries of the retina had become dark and granular; they were charged with minute oil-globules and often contained a few grains and little masses of red pigment. In short, these vessels were in a state of fatty degeneration, and the neighbouring parts of the retina supplied by them participated in the atrophy, and formed small spots of a dull, stone-gray colour.

The structural changes which take place during the early stages of the disease all point to a state of great vascular excitement in the retina. An increased flow of blood augments the internal pressure on its walls. In practice, the degree of tension of the globe is measured by its hardness. Every one who has injected an eyeball knows how very hard it becomes when fluid is forced into it with an injecting syringe, and the hardness varies directly with the pressure. The cupped state of the optic papilla is also produced by the internal pressure, and Gräfe says the degree of excavation gradually increases as the disease advances. Probably, in consequence of the altered state of the optic papilla, the exit of the blood through the vena centralis is embarrassed, and a strain is thrown backwards upon the capillaries and arteries. The delicate capillary wall cannot sustain this strain, and hence the dilatations, the ruptures, the hæmorrhages. The arteries, from their efforts to overcome the impediment, become hypertrophied. With every beat of the heart the tension of the globe is momentarily augmented, and simultaneously the return circulation through the central vein is stopped: the column of blood is checked and a pulse appears in the artery.

The violent pain, and the paralysed state of the iris, depend on the great pressure upon the ciliary nerves. When this pressure is relieved artificially, these symptoms are diminished. I have already mentioned a case in which some of the vitreous humour was let out to diminish the internal pressure. In this instance the operation was directly followed by contraction of the pupil; the iris acting on the side corresponding to the puncture in the sclerotic. Gräfe, in his memoir above cited, attributes the blindness which occurs early in acute glaucoma to the great pressure upon the retina and optic nerve, rather than to changes in the retinal tissues. In this way he explains the excellent results he has obtained in the treatment of acute glaucoma by the operation which he has originated, and which he has submitted to extensive trial.