# Unilateral Haemorrhagic Retinitis

and

# Haemorrhagic Glaucoma.

A Thesis

for the

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by

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# Unilateral Haemorrhagic Retinitis and Haemorrhagic Glaucoma.

## General Considerations and Literature.

The cases which I propose to bring forward and discuss in the following Thesis occurred in the Dispensary Department of the Glasgow Eye Infirmary and hence for the most part could not be kept under very close or long-continued observation. Nevertheless as they form a very sharply-defined group, and as they raise some points of considerable interest in the domains of general medicine and of pathology, I have thought well to put together such notes and illustrations of them as I was able to make.

In the term "haemorrhagic retinitis" I do not include for the present every case in which with signs of retinal inflammation there were perhaps a few haemorrhages in the retina. In the cases described below the haemorrhages were the predominant feature in the ophthalmoscopic picture. Signs of inflammation even, of which haemorrhages are so often the prodromata, accompaniments or sequelae, were subordinate. The haemorrhages in these cases have little in

common with those which may occur secondarily to an attack of intense neuritis e.g. choked disc, or with the scattered flame-shaped haemorrhages commonly seen in association with albuminuric retinitis. The characteristic ophthalmoscopic picture was an intense suffusion of the whole visible fundus with haemorrhages of all sizes and shapes and this occurring in one eye only.

The fact that these intense changes in the retina occur upon one side only is important in the inquiry as to their cause. We must be dealing here with a local and not a general cause; or at least if the haemorrhages be dependent on some general bodily state we must have some local pathological condition in addition; otherwise the affection would be bilateral since both sides would be equally affected by a general bodily condition. Thus the haemorrhages which occur in such constitutional diseases as diabetes, pernicious anaemia, Bright's disease, septicaemic etc., are practically always bilateral, or in the few cases where they occur on one side only they are small and few in numbers. What then is this local cause. Michel who seems to have been the first to classify these cases with regard to their cause (in 1878) answers, "a thrombosis of the central vein of the retina" a lesion which he proved pathologically in one case which came to post-mortem.

Michel (1) founds his description on seven cases, five men and two women. He fully reports four of them. The age was usually between 60 and 70; the lowest 51 the highest 81. In every case the affection was unilateral except that in one it followed in the second eye about a year after the first. All his patients were the subjects of marked arteriosclerosis, but in no case was there albumen in the urine. Dimness of vision, according to his description, sets in suddenly, sometimes in the night and without any subjective prodromata. Nevertheless it is not so sudden nor so complete as in embolism of the central artery, and in the further course of the disease the visual acuity may rise considerably even if only temporarily. Michel divides the cases into three grades of intensity according as (1) the vein is completely blocked, (2) the vein is partially blocked, (3) there is so slight a blocking that only a little damming back of the blood takes place. This division is only for convenience of description, there are of course intermediate degrees.

In the first grade the suffusion of the retina with blood is very intense. The disc margins are entirely hidden, and in an area of one to one and a half disc-diameters around it Michel compares the appearance to a "pool of blood." The haemorrhage here

is striped. The region of the disc however is not noticeably higher in level than normal. Outside of this area are innumerable circumscribed haemorrhages, flame-shaped and round, of all sizes, some clear red, others very dark. The vessels, hidden near the papilla, first come into view here, the arteries very small and hardly noticeable, the veins greatly enlarged, "like sausages", extremely tortuous, dark red, variable in diameter. In places they have small stripes along their course. In the macular region there is a yellow-gray coloration with a red spot of real extravasation.

In the second grade the changes are similar but less intense. The disc margins are almost covered by broad striped haemorrhages, and some small ones are also present on the disc itself, especially to the temporal side. The arteries are very small, the veins enlarged, sausage-shaped, dark-red and tortuous. Arteries and veins can however be followed to their origin in the papilla. In the periphery there are many round and oval, large and small apoplexies. In the third grade the haemorrhages are few. Some striated ones are collected round the disc, and some small round ones are present in the periphery. The chief feature

ophthalmoscopically is the great disproportion between the arteries and the veins, the arteries being thin and small and the veins greatly enlarged and tortuous - much more so than in optic neuritis of inflammatory origin. The contour of the disc is slightly veiled.

The prognosis varies in each of these grades. In the first, if the obstruction remain complete there may be temporary improvement, partial resorption of haemorrhages and some clearing of the disc; yet usually fresh extravasations follow, the vitreous becomes opaque, and sight is reduced to perception of light or nil. If, however, the lumen of the vein have been only partially obliterated, or if it be partially restored either by the disorganisation of the thrombus or by its organisation and the formation of passages through the new-formed fibrous tissue, the vision may rise again though never to normal, and the ophthalmoscopic appearances may become stationary in a more or less advanced condition of degeneration. In the third grade also vision does not rise quite to normal, and there is usually left at least a disproportion between the arteries and veins, and a slightly atrophic condition of the nerve-head.

The case microscopically examined occurred in a man of 58 who showed marked

general arteriosclerosis and had had a threatening of left hemiplegia. The ophthalmoscopic appearances were those of the highest grade of intensity. During 16 months he was under occasional observation and the fundus condition showed no great change, although there were at times new haemorrhages, and latterly the fundus details were dim. At the end of that period he died and the post-mortem showed besides general arterio-sclerosis, hypertrophy of the heart and mitral insufficiency; old standing pleurisy and hydrothorax; fatty liver and general anasarca. The cavernous sinus and the ophthalmic artery and vein were normal. In the central vein of the retina a cellular organized thrombus was found 1½ m.m. long, 6 m.m. from the ball, and 3 m.m. to 4 m.m. from the point of entrance of the vein into the nerve. It practically filled the vein leaving only a minute cleft between it and the wall and a few minute clefts in its substance. The central artery had a somewhat thickened adventitia but a normal lumen. For the rest the optic nerve was atrophic; the retina was distorted from the amount of blood in it but otherwise not very greatly altered, the spaces between the fibres of Müller broadened and often filled with fibrin coagula, the nerve fibre and ganglion cell layer much atrophied.

The tortuosity of the veins and the haemorrhages are of course fully accounted for by the venous obstruction caused by the thrombosis. Michel explains the smallness of the arteries, not very satisfactorily by supposing that the circulation is deficient in them because of the venous obstruction acting backwards through the capillaries. This, however, would probably cause engorgement rather than smallness, unless indeed it were that the thickening of the adventitia of the arteries interfered with the distensibility of the vessel. He holds it to be uncertain why the thrombus should have settled in the place where it did, and suggests that possibly there may have been some slight bend in the vessel which, in association with the slowing of the blood-stream caused by the general arteriosclerosis, might be sufficient to give rise to a thrombosis.

As regards the general condition of the other cases which did not come to post-mortem the second case - belonging to the second grade of intensity - showed accentuation of the heart sounds at the aortic area, and there was emphysema. In the third there was marked arthritic deformity, and in the fourth the first sound at the tricuspid area was impure and the sounds at the aortic area weak. In all there was wide-spread arteriosclerosis.

In the same year Angelucci, (2) as the result of pathological examinations reported two cases as thrombosis of the central vein in

which the clinical picture was quite different. His patients were young - 23 and 24 years respectively - and in both cases there was found post-mortem, endocarditis of the aortic and mitral valves. Blinding was sudden, complete and permanent as in embolism, and ophthalmoscopically there were no haemorrhages but only swelling of the veins. Doubt has been cast upon these cases by most subsequent writers. It has been held extremely unlikely that there could be stoppage in the outflow of an end artery without haemorrhages. There was also a lapse of some months between the ophthalmoscopic and pathological examinations, and as the patients died with increasing failure of the circulation, the thrombi found may have been recent and marasmic. There is also doubt as to whether the condition found was a real thrombus or only a post-mortem clot inasmuch as Angelucci himself says that the endothelium of the vessel was intact. Zehender (21) attempts to explain the lack of haemorrhages in Angelucci's cases and in two similar cases of his own by supposing a compression of the artery by the thrombus, or by its involvement in periphlebitic processes. No one else seems to have reported cases similar to those of Angelucci and Zehender.

Michel in the cases reported by him specially mentions that the tension was not raised, yet it has been long known that glaucoma may follow upon haemorrhagic retinitis. v Von Graefe (3) as long ago as 1859 anticipating many of the points in Michel's clinical

description gave a review of this subject. From the number of cases of the kind which he had seen he held that the combination was not accidental. The disease, he says, occurs usually in persons over 50 who have often wide-spread arteriosclerosis or evidence of changes in the walls of the retinal vessels. He had only once seen a case under 40. The onset of amblyopia is sudden, with central or eccentric scotomata or general dimness of vision. Chromopsia and photopsia are occasionally present but mostly absent. The ophthalmoscope shows the retina suffused with haemorrhages and the veins greatly enlarged, and later these haemorrhages may break through into the vitreous. Glaucoma does not necessarily follow, but in a certain proportion of cases it occurs in from a half to six months, either as an acute attack or as a gradual rise of tension, with severe pain and muddiness of the vitreous and it may be of the aqueous with blood. In which cases this will occur it is impossible to predicate. If left alone these cases may finally end in phthisis bulbi, with scleral staphylomata, while on the other hand if interfered with by operation, the prognosis for the operation is peculiarly bad and therapeutic treatment is quite powerless. Of 22 cases of the kind which v. Graefe had seen 10 showed no affection of the other eye 2 years later, 5 showed retinal haemorrhages but no glaucoma in the other eye, in 6 the other eye was subsequently attacked by glaucoma, and in 1 both eyes were simultaneously attacked.

V. Graefe gave no pathological examinations of such eyes, but in 1879 Deutschmann (4) reported the pathological examination of two cases probably of this kind, in which the eyes were enucleated for glaucoma. In one cases 8 days after a sudden loss of vision in the right eye many small haemorrhages about a third of a disc-diameter were found scattered over the fundus, and the retinal veins were tortuous. Nine months later there was pain and pericorneal congestion for which atropin was prescribed, and following upon this the eye became glaucomatous and was enucleated. In the other case the right eye became blind the first symptom being retinal haemorrhages. Nine months later the tension was up and the nerve excavated, and as iridectomy was followed by no improvement the eye was enucleated. In neither of these cases was there any thrombus of the central vein or its branches found, but there were haemorrhages in the substance of the optic nerve. In the second case the lumen of the retinal vessels was much reduced by a hyaline thickening of their walls. In both the filtration angle was shut off by adhesion between the iris and the back of the cornea. In this communication Deutschmann first drew attention to an appearance in the retina subsequently confirmed by other writers (Wagenmann, Stölting, Sankwitz, see also below Case 7 fig 25) namely an appearance as if the cone nuclei of the outer nuclear

layer were shifting out through the limitans externa into the inner segments of the cones.

Since then several cases have been reported in which glaucoma followed upon severe unilateral haemorrhagic retinitis, indeed most of the pathological material from which it has been attempted to work out the etiology of such a retinitis has been obtained by enucleations for intractable glaucoma. It is one of the objects of this thesis to show that "haemorrhagic glaucoma" is not a pathological entity but may originate in more ways than one. In the cases to which I have been referring the haemorrhagic retinitis was primary and the glaucoma followed upon it. I do not mean to deny that the glaucoma may be the primary lesion and that the haemorrhages may follow, partly from damage to the vessel walls from the continued high tension, and partly no doubt from the obstruction to the venous return through the compression of the veins where they curve sharply round the edge of the excavated disc. I believe that there is a third way in which haemorrhagic glaucoma may originate; the haemorrhages may arise when an operation is undertaken on an eye the subject at first of a simple glaucoma. Case 7 discussed below I believe to be an example of this class. No doubt the fundamental cause of the haemorrhages is the same in all these cases, viz. vascular disease within the eye. Evidently, however, in any given case of haemorrhagic glaucoma unless the sequence of events had been actually observed it might well be

impossible to say from the examination of the eye alone whether the haemorrhages or the glaucoma had been the primary lesion. A careful review of the clinical history of the case and, if it were available, the pathological examination might throw some light on the question. For the present we are dealing chiefly with the glaucoma which follows upon haemorrhagic retinitis.

To return to the literature; in 1889 Nettleship (5) in an article on chronic glaucoma mentions the case of a man aet 52 who had right-sided haemorrhagic retinitis when first seen, and a year later the eye was enucleated for glaucoma with pain. He mentions other cases possibly of this nature but in which the sequence of events was doubtful.

Anatomical proof of Michel's second grade was furnished in 1892 by Hagenmann (6a). A man aet 76, the subject of arteriosclerosis and who had had three attacks of agraphia, had a right-sided haemorrhagic retinitis of moderate intensity. One year later the eye became glaucomatous and amaurotic, and a corneal ulcer subsequently developed. The eye was enucleated, and the examination showed, some distance behind the lamina cribrosa, a spindle-shaped mass in the central vein partially filling the lumen; intimately adherent to the wall; finely granular but showing no nuclei. The vein wall was somewhat thickened. The iris showed signs of inflammation (perhaps

secondary to the corneal ulcer), and the filtration angle was shut off. Wagenmann holds the thrombus in this case to have been not marasmic, as Michel believes, but secondary to phlebitis and periphlebitis a condition perhaps associated with the occurrence of a trace of albumen in the urine.

Wagenmann, however, will not allow that thrombosis of the central vein is the lesion in all these cases of unilateral haemorrhagic retinitis. He gives two cases in which no thrombosis of the central vein was found. The first concerned a man of 60 with evidence of arteriosclerosis and cardiac hypertrophy but without albuminuria. In the right eye there was a severe haemorrhagic retinitis, the left eye being normal. Subsequently there was iritis, and the eye was enucleated for pain and glaucoma three months after the onset of symptoms. In this case the wall of the central vein was a little thickened, but the chief pathological change was in the arteries. The central artery was reduced to a third of its normal calibre by endarteritis, and the retinal arteries were greatly reduced in lumen the superior temporal being entirely obliterated. The filtration angle was shut off by connective tissue. In the other case, a man aet 42, in which also there was a haemorrhagic retinitis followed by iritis and glaucoma, a similar blocking of the retinal vessels near the papilla was found, but in this case there were present in the retina very definite signs of inflammation (round cell filtrations,) which were not present

in the other two. A case apparently similar, but of which I have been unable to consult the original report is given by Reimar. (34)

In the latter of these two cases Wagenmann considers that the vessel walls were damaged by some noxious agent, possibly microbic which most probably caused the haemorrhages by multiple lesions of the vessel wall and consequent thromboses. In the former of the two cases he believes that there had been multiple emboli in several of the branches of the central artery. Yet there are difficulties in the way of accepting of arterial emboli as a cause for retinal haemorrhages. In embolism of the central artery it is well known that haemorrhages are quite the exception and never profuse even if they occur at all. Yet the central artery is an end artery, and when embolism of such an artery occurs elsewhere - for instance in the lung - the blood flows back from the surrounding venous radicals and gives rise to a haemorrhagic infarction. Why does this not happen in the eye? Wagenmann explains that the intraocular pressure is sufficient to prevent the reflux of blood from the vein which would cause such an infarction. But, he argues, there is a difference in this respect between an embolism of the main stem of the artery and an embolism of one of its retinal branches. For in the former case the whole blood supply of the retina (unless from cilio-retinal vessels) is cut off, while in the latter there is still a blood supply to all the vessels both arterial and venous except the arterial twig which has

become blocked. Hence there will be a free flow of blood into the vein corresponding to the blocked artery from its collaterals within the eye, and so the infarction may take place. In the case in question Wagenmann explains that there had been an embolism of several arterial twigs but not of all, and hence the circulation in the eye was not altogether stopped and the infarctions took place in various regions of the retina and gave the appearance of a general haemorrhagic retinitis.

To support this complicated explanation it would be necessary to prove that branch embolism is actually followed by retinal haemorrhages, and recorded cases do not seem to bear this out. Nagel (7a) in a recent article points out that "in a vast majority of cases of embolism of a branch of the central artery haemorrhages into the retina are absent or are present only in insignificant numbers" and gives the names of no fewer than twenty authors who have reported cases which bear this out. I have myself only seen two cases of complete embolism and one of branch embolism (superior temporal) and in none of these was there haemorrhage. It might be argued that although this is true for an embolus entering a fairly healthy artery it may not be so for an artery affected with senile sclerosis. Wagenmann however, reviewing this case at a later period (1898) inclines to the view that the condition was one not of multiple embolism, but of multiple thrombosis.

Although Wagenmann denies in this case that there was any thrombosis of the central

vein, yet it may be pointed out in this place that Michel and Angelucci alone obtained their pathological material from post-mortem and therefore had the opportunity to examine the whole length of the optic nerve. The thrombus in Michel's case was 6 m.m. behind the eyeball, which is probably longer than the stump of optic nerve usually taken away in an enucleation, and hence it is possible that in Wagenmann's case and in others where no thrombus was found there may really have been one, but too far back to be present in any of the sections. Blockage of the circulation by a thrombus in the central vein would account for very profound changes in the retinal vessels especially when reinforced by glaucomatous tension. It is to be remembered that while veins are more liable to thrombosis from the greater slowness of the current in them, arteries are more liable to show thickenings of their walls.

Another case pathologically proved to show a thrombus in the central vein was reported by Weinbaum (8) in 1892. The causation in this case was obscure, the patient being a healthy man of 26 without heart, lung or kidney disease. Dimness of vision of the right eye came on suddenly and about two months later he began to have pain in the eye. When first seen the tension of the right eye was raised, the anterior chamber was shallow, the pupil was widely dilated and the iris seemed swollen and had streaks of haemorrhage on its surface. Through a hazy cornea striped

retinal haemorrhages were seen. After an unsuccessful iridectomy followed by haemorrhage into the anterior chamber, the eye was enucleated for persistent pain and glaucoma a little over a year after he was first seen. On organized thrombus was found in the central vein about 1¼ m.m. behind the lamina cribrosa and extending for ⅝ m.m. It resembled granulation tissue in structure but without vessels, and nearly filled the vein which was otherwise normal. The filtration angle was shut off by fibrous tissue, and there was very marked distortion of the iris by new-formed cicatricial tissue which had caused marked pulling round of the pigment layers of the iris - ectropium uvee (Cf. Case 7, fig 16). In this case the tension was raised when he was first seen so that there might be some doubt as to whether the glaucoma or the haemorrhagic retinitis were the primary lesion. The onset of sudden dimness of vision some time before pain speaks for the retinal disease having been the primary one, and it is pointed out by Weinbaum that the thrombus can hardly have been secondary to the glaucoma since it was behind the lamina cribrosa where rise of intraocular tressure could not affect it. The age of the patient throws no light on the subject, since it is against both primary thrombosis and primary glaucoma.

In the same year Furtscher (9) published the case of a woman aet 21 without evident organic disease but anaemic and liable to epistaxis. After a period of failing vision lasting two months glaucoma set in in the right eye with ocular and periorbital pain for which

enucleation was performed about three months after the occurrence of the first symptoms. The other eye showed retinitis proliferans and a few haemorrhages. Pathological examination showed the iris to be much distorted, adherent to the back of the cornea and showing ectropium uveae. In the central vein there was an organized thrombus .6 m.m. long, leaving only a small cleft-like lumen at one side. The vein wall was thickened and the retinal vessels also showed thickening of their walls from hyaline degeneration. There were many haemorrhages in the retina with formation of connective tissue and distortion of its structure.

In 1896 Stölting (10) reported the case of a woman aet 65 who had complained of dimness of vision in her right eye for four weeks. She had occasional cardiac irregularity but there was neither albuminuria nor glycosuria. Haemorrhagic retinitis was present in the affected eye; the margins of the disc were dim but there were no special alterations in the vessels. A month later tension was + 2 and in spite of temporary improvement after iridectomy, the eye was enucleated three weeks later for pain and glaucoma. Here there was no thrombus found. The central artery and vein which had a free lumen up to just behind the lamina cribrosa became there suddenly narrowed from hyaline degeneration of their walls, and there was much thickening of the walls of the retinal

vessels. The changes were more marked in the arteries than in the veins. The iris was adherent to the lens and pressed forwards against, but not adherent to the back of the cornea. These changes are very similar to those found by Wagenmann, and Stölting inclines to the same theory with regard to their origin, namely that there had been multiple emboli in the retinal arteries and that the changes in the vessel wall were secondary to this. The appearances were not typical, but he accounts for this by the length of time which had elapsed between the blinding and the pathological examination. An alternative theory which he propounds, namely that there might have been increasing disease of the vessels with haemorrhage either by rupture or diapedesis, he considers improbable because the central artery which must have participated in such disease had a free lumen right up to the lamina cribrosa. Besides, such a theory would not account for the lesion being strictly unilateral.

Suler (11) in 1896 described the following case. A man aet 39 with evidence of mitral and tricuspid insufficiency and consecutive nephritis, had noticed a gradual diminution in the vision of his left eye of two months duration. Only a dull red reflex was obtainable and the tension was slightly raised. A month later the eye was enucleated on account of

acute glaucoma and pain. The retina was suffused in every part with haemorrhages, and in the central vein, which resembled an artery in the thickness of its coats, a "clot" was found extending to the retina. As to whether this was an ante or post-mortem clot there is no hint in the report.

Sankwitz (12) in 1898 reported the pathological examination of another case of unilateral haemorrhagic retinitis with consecutive glaucoma. A woman aet 72 who suffered from cardiac symptoms and who had had right hemiplegic, complained of a sudden loss of vision in the right eye, five weeks before she was seen. Examination showed in the affected eye severe haemorrhagic retinitis with swollen veins and thread-like arteries; the iris was discoloured and showed posterior synechiae. The left fundus was normal. Later high tension and pain set in and the eye was enucleated about two months after the first onset of symptoms. Here the chief pathological change was in the central artery, which showed behind the lamina cribrosa great narrowing of its lumen from endarteritis. Just in front of the lamina its wall was bulged out into the form of a small aneurism, and at a corresponding point the vein suddenly became narrow and its hitherto free lumen contained cells and crumbling detritus. The retinal vessels both arteries and veins showed very marked changes, amounting here and there almost to obliteration, and their lumen contained in places crumbling thrombotic

masses. The corneo-iritic angle was shut off with connective tissue, and there was evidence of posterior synechiae and iritis from which the glaucoma had probably arisen. Bankwitz denies that the arterial changes in this case were secondary to any thrombosis in the central vein. The course of events he believes to have been as follows; as a result of a high grade of endarteritis a little aneurism formed upon the central artery in much the same way as often happens in the case of the cerebral arteries, and as had indeed probably happened in his patient's cerebral arteries (i.e. she had had hemiplegia.) In consequence of this aneurism the blood-current in the vein was obstructed, both directly by pressure on it, and indirectly by slowing of the arterial stream. Hence a venous thrombosis probably occurred resulting in profuse haemorrhages. If this be the correct pathology it will be seen that although the venous thrombosis was not the primary factor in the case, yet it was the factor which determined the haemorrhagic retinitis and the loss of vision. In this respect therefore the pathology is exactly the same as in the other cases already mentioned.

Wehrli (13) in 1898 reported the following case. A man aet 50, the subject of Bright's disease, had a haemorrhagic retinitis of the left eye, the right remaining free up to the time of his death nine months later. The retinitis was of moderate intensity with many white plaques round the disc, the picture

given showing much resemblance to fig 6 below. Glaucoma followed a month afterwards, and after an unsuccessful iridectomy the eye was enucleated six weeks later. Unfortunately the nerve and papilla were left in at the enucleation and were therefore not examined. The iris was adherent at its base to the cornea, the retina much thickened and distorted, the fibres of Muller in especial being much hypertrophied. The retinal vessels were greatly thickened by hyaline degeneration.

Jürk (22) and Würdemann (23) have also reported similar cases where a thrombus was found in the central vein on pathological examination, and Alt (24) describes a case where there were multiple thrombi of the retinal veins. These along with the names of Bourgon (25) Mannhardt (26), Schnabel (27), Borchene (28), and Clairborn (29), I only mention as I have been unable to consult the original communications.

Pictures of the ophthalmoscopic and microscopic appearances of the retina in thrombosis of the central vein are given by Haab (7c) in his Atlas, though in the case portrayed the haemorrhages are moderate compared with what they sometimes are. He also pictures a case which he considers probably a partial stoppage of the central vein. Haab (7b) in reviewing the whole subject in a recent article agrees on the whole with Michel's findings. He says that typical cases without albuminuria are rare; that heart disease is favourable to its

occurrence probably by producing early arteriosclerosis, and that working in a stooping posture is apt to bring it on. An excellent summary of the whole subject of thrombosis of the central vein will be found in this article.

The affection in its typical form seems to be far from frequent. Schobl (14) mentions having seen it three times in 180,000 patients. Ammann (30) however, who gathered all the cases from Haab's clinique, reports 20 cases in which Michel's picture of thrombosis of the central vein was present and 13 in which there seemed to be branch thrombosis among over 60,000 patients giving for the cases of thrombosis of the main stem a percentage of .03. In the course of a year I have been able to collect six cases which gave the clinical picture with more or less completeness out of a total of probably between 11,000 and 12,000, a percentage of .05. With regard to the conditions under which it occurs 15 out of Ammann's 20 were between 50 and 80 years of age, and 6 suffered from heart lesions. From the same point of view C. Moses (31) has reviewed 50 cases of complete or incomplete thrombosis of the central vein or its branches; 38 of these were over 50 and had arteriosclerosis, 4 who were under 50 had arteriosclerosis from Bright's disease; 1 showed much interference with the heart's action from a high degree of kyphoscoliosis, 2 had mitral insufficiency, 5 diabetes, 2 fatty heart and pernicious anaemia and 1 had thrombosis of the

cavernous sinus. One case occurred in a girl of 15 with profound anaemia, disturbed heart action and evidence of hereditary syphilis.

In looking back now over the reported cases where pathological examinations have been carried out, we find that the following explanations have been put forward by different authors to account for the occurrence of severe unilateral haemorrhagic retinitis. (1) Thrombosis of the central vein of the retina in the cases of Michel, Wagenmann (in one of his cases) Weinbaum, Juler (?) Noak, Eankwitz, Türk and Würdemann. (2) Multiple thrombosis of the branches of the retinal vein in Ott's case. (3) Multiple emboli or perhaps multiple thrombi of the branches of the central artery, the vein being free, as in two of Wagenmann's cases and in Stölting's case. (4) Haemorrhages in the optic nerve, found only in Deutschmann's cases. In the majority of the cases then which have been pathologically examined a thrombus has been found in the central vein. Thrombosis offers a very satisfactory explanation of the ophthalmoscopic picture, and it has already been pointed out that a thrombus may have been present far back in the vein in some of the cases in which none was found on microscopical examination of the enucleated eye. Nevertheless more pathological examinations would be necessary, and especially examinations in which the whole course of the central vessels was included before it could

be settled whether this lesion were constant or not.

As regards the etiology of such a thrombosis Michel is credited by most authors with saying that he believes it to be marasmic in origin. By "marasmic thrombosis" we usually mean a coagulation of the blood in the veins from slowness of the current in states of great bodily prostration (e.g. the marasmic thrombosis of the cerebral sinuses in the chronic diarrhoea of infants). But this degree of prostration was not present in the cases under consideration, and besides if the thrombus were purely marasmic in origin it would probably be bilateral. This, however, is not the kind of marasmic thrombosis to which Michel refers. He says that the venous current is slowed by the diseased condition of the arteries from arteriosclerosis, and that the site of the thrombus is probably determined by some accidental curve or narrowing of the vein. Wagenmann believes that the vein takes some share in the etiology, showing evidence of peri- or endophlebitis as a part of the general vascular disease.

This, however, is not to say that a purely marasmic thrombosis does not occur. A case given by Goh (15) seems to be of this nature, and it is noteworthy that the affection was bilateral. A man aet 25 showed a haemorrhagic retinitis in both eyes when he was in extremis three days before his death from septicaemia following upon stomatitis ulcerosa. During these three days there were

many fresh haemorrhages and the veins became much swollen. Pathological examination showed a recent thrombus in the right central vein, the vein wall being strictly normal in appearance. In the left eye in which the haemorrhages had been fewer there was no thrombosis of the central vein, but many thrombi in the retinal venous branches. Coh accounts for the central vein being attacked only on the right side by the fact that there was an unusually great curvature of it on that side. In this case, from the age of the patient there is evidently no question of arterioselerosis, and as the vein wall showed no evidence of inflammation we may take it that the thrombosis was marasmic in origin. Oxenfeld (32) has reported a similar case.

In looking over these recorded cases it is evident that the statement of v. Graefe is fully borne out, that glaucoma follows too often upon haemorrhagic retinitis to be a merely accidental coincidence, and we must now consider what the connection between the two states is. In a few of the cases there was an evident enough cause; thus in one of Deutschmann's (4) cases the glaucoma followed upon the use of atropin in a man of 72. In two of Wagenmann's (6a) cases and one in the case of Bankwitz (12) there was clinically observed an attack of iritis. But excluding these the others showed no other cause for the onset of glaucoma than the pre-existing retinal haemorrhages. One thing is noteworthy in surveying the recorded cases;

in no case after the onset of glaucoma was there a clear view of the fundus and indeed in most cases it was altogether invisible, the vitreous being beset with floating opacities. It seems, therefore fair to suppose that the retinal haemorrhages themselves do not cause glaucoma unless they break through into the vitreous. Stölting (10) gives the following explanation of the origin of the glaucoma:- The pouring out of blood into the vitreous causes the tension in the posterior segment of the eye to be higher than in the anterior, and hence the lens is pushed forwards carrying with it the iris and so blocking the filtration angle. At first there is merely apposition between the iris and the back of the cornea, but afterwards (probably when the endothelium has been destroyed by the pressure) actual fibrous union takes place. In Stölting's own case there was opposition but not adhesion; in all the others where it is mentioned there was actual adhesion (see also below Cases 7 & 8.) In addition to this there is the further factor that the constituents of the intraocular lymph-streams are altered by the presence of the blood so that their constitution is more colloidal, and hence they filter through the angle with greater difficulty.

This appears to be a reasonable explanation yet we must remember that haemorrhage into the vitreous even in large quantity is not necessarily nor I believe at all frequently followed by glaucoma. Pröbsting (16) in 1892

carried out a series of experiments on the results of the injection of fresh blood into the vitreous of dogs. He obtained separation of the retina and a condition resembling retinitis proliferans, but there was never rise of tension. I have also had a case under observation which illustrates the same thing. A young man aet 21 had had repeated attacks of amblyopia during four years in the left eye and during one year in the right. These attacks commenced with the appearance of "streams" before the eye, and after shortly reaching a maximum the amblyopia gradually cleared off in some week's time, leaving however, the sight rather worse than before. With the left eye he could barely distinguish light and shade though he saw rather better (counted fingers at about 8 inches) to the temporal side. With the right eye vision was 20/100 and the ophthalmoscope showed some curiously shaped retinal haemorrhages from one of which, on the nasal side of the disc, a web of haemorrhage stretched into the vitreous, moving a little with the movements of the eye. There were some dark floating opacities in the lower part of the vitreous. These retinal haemorrhages after nearly clearing away recurred in almost exactly the same form about a month later, with a new attack of amblyopia. This then is evidently one of those interesting cases of recurrent retinal and vitreous haemorrhage of young adolescents. There had apparently been haemorrhage in the left vitreous for from three to four years and yet

the tension was perfectly normal.

It is to be observed, however, that these haemorrhages occurred in a young man apparently otherwise in perfect health, and certainly with no arteriosclerosis and the dogs on which Pröbsting experimented were no doubt also healthy animals. It is probable that glaucoma will follow more readily from any cause in old persons with diseased arteries than in such subjects. That it may follow, however, even in the young is shown by a case of Hutchinson's (17) in which a man aet 23 had large retinal and vitreous haemorrhages and the eye was subsequently lost from glaucoma; later the other eye was attacked with haemorrhage but glaucoma did not ensue in it.

## Clinical Cases.

None of the following cases I think quite reach the degree of intensity described by Michel as his "first grade." In all there was visible at least some trace of the disc or of the vessels in its position. The "pool of blood" entirely covering the papilla was not typically present. The first case described might be classed as an intense degree of the second grade.

### Case 1.

Intense unilateral haemorrhagic Retinitis following upon Influenza. Jas. E., aet 62 a Carter.

Presented himself at the Glasgow Eye Infirmary Dispensary on May 29th, 1900, complaining of failure of vision in the left eye of about seven weeks duration.

The patient's heredity was satisfactory and he had always enjoyed excellent health. Some years ago he sustained a severe fracture of the leg, from the effects of which he was still lame.

History of Illness. About two months before he was first seen he had been laid up with an attack of Influenza. The chief symptoms complained of were, feverishness with much asthenia and disinclination for all exertion, loss of appetite and slight abdominal pain apparently dyspeptic in type i.e. coming on an hour or two

after meals and rather relieved for the time at least by the taking of food. On account of this attack he was in bed for a fortnight, and it was quite a month before he was going about again freely, and six weeks before he felt himself to be fully better. Three weeks after the onset of his illness, while he was still more or less confined to the house, vision failed rather suddenly in the left eye. It seemed as if a veil or mist had been drawn over the sight, and in addition especially in the morning and at twilight a large scotoma, not absolute in character, hovered about the fixation point. There were no prodromal phenomena of any sort. Vision had remained since in much the same state. If anything there was slight improvement. The vision of the right eye was quite unaffected.

Present Condition. The man was very well preserved for his years, and showed little sign of senility. The heart sounds were pure and the limits of cardiac dulness not abnormal. The radial arteries were somewhat tortuous, but signs of arteriosclerosis were not of very high grade.

The lungs showed no abnormality.

Urine Sp.G. 1030, Acid, no albumen or sugar. The external appearance of the eyes was normal, the pupils equal and responding to light and convergence, and tension was normal.

V.O.R. 20/30. V.O.L. counts fingers at 2½ ft.

O.E.R. The fundus is strictly normal. The disc is well defined. Arteries and veins bear

normal relations to one another and show no signs of disease. No haemorrhages are anywhere visible.

O.E.L. (fig 1) Shows in every part of the fundus an intense suffusion with blood. Around the region of the disc these haemorrhages are especially large and numerous, and the disc margin is concealed by them. The central portion of the disc is however free from haemorrhages, and the point of exit of the vessels is visible, though there is a degree of haziness over the whole area suggestive of oedema. The disc is much congested. Of the vessels themselves the veins are much swollen and very tortuous, the arteries are certainly in comparison very small, but whether absolutely diminished in calibre it would be rather difficult to say. Beyond the disc the arteries are not traceable, but a loop of tortuous vein comes into view in places, the course of the vessel being elsewhere concealed by haemorrhages. The macular region is involved in the haemorrhages and presents no notable difference from the rest of the fundus. In the periphery, the haemorrhages are rather smaller and more scattered, but no large area of the fundus is free from them. No yellow flakes or spots, such as are often seen when haemorrhages have been present for some time, are visible. The media are quite clear.

This man presented himself again on June 29th, 1900, when the above appearances in the retina were practically unaltered. In the vitreous, however, there were many dark flakes floating. The tension was still normal.

Here then we have a case corresponding in almost every particular to the description given by Michel. The man was of the usual age - between 60 and 70 - free from albuminuria but showing at least some degree of arteriosclerosis; after an illness characterized by marked asthenia vision was suddenly lost in one eye by an intense haemorrhagic retinitis, the other eye remaining at the time when he was last seen entirely unaffected. The diagnosis of thrombosis of the central vein certainly offers a very tempting explanation of all these phenomena. The lowered blood pressure and general state of bad health following upon his illness would be predisposing causes which might well be aided by some local obstruction in the vessel itself, either in the form of a thickening of its wall or of some unusual curve in its course or perhaps of both. Venous thrombosis is a known though rare sequela of influenza. Thus Dr. Judson Bury, (18) describes the case of a lady who had first thrombosis in the veins of the leg after influenza, and who subsequently died of cerebral haemorrhage. I have also seen in the Wards of the Royal Infirmary a woman with right hemiparesis and other nervous symptoms which were thought to be traceable to an attack of influenza.

The following case also gave a history of an attack of influenza, though the dimness of vision did not occur during the actual course of the illness as in the last case.

Case 2.

Unilateral haemorrhagic Retinitis following upon an Attack of Influenza.
Mrs. Mc J., aet 40.

Was seen at the Glasgow Eye Infirmary Dispensary on May 8th, 1900, complaining of failure of vision in the left eye of nearly three months duration.

The patient's hereditary history revealed nothing of importance. She was married and had had five children of whom three had died from diseases of infancy (bronchitis, whooping cough and diarrhoea.) There was no evidence of any specific taint. Her pregnancies had always been normal in course, and she had had no difficulty in parturition. During the previous two years she had been liable to bilious attacks with vomiting, but these had not been present during the past six months. Apart from these she had had no symptoms pointing in the direction of Bright's disease, such as pain in the back, headache, oedema etc.

History of illness. In February, 1900, she had had an attack of Influenza. After a feeling of lassitude and illness lasting one week, but on account of which she was not confined to bed, she took a shivering fit and thereafter was too ill to rise during about another seven days. She was then able to get about again fairly well, but a fortnight later, while she was reading a book a sudden mistiness came over the sight of both eyes,

but in a much greater degree over the sight of the left. With the onset of this there were lightning like scotomata whether before both eyes or only the left she was uncertain, and it seemed as if a "ground glass" had been introduced between her eyes and the book so that she was unable to read. Vision in the right eye soon began to clear up again, and she considered that it was now as good as formerly but the condition of the left had shown no improvement. She thought that there had been some deterioration in her general health of late.

Present Condition. The woman was anaemic looking and did not seem to be in the best of health. No oedema was present at the time of examination. There was no proof of arteriosclerosis in the peripheral vessels. The limits of cardiac dulness were normal and the sounds pure. Nothing noteworthy was to be detected on examination of the lungs. The urine was thought to contain the faintest possible trace of albumen, but no unequivocal tube-casts were to be found.

V.O.R. 20/50 V.O.L. Counts fingers at 18 inches. The external appearance of the eyes was normal. The pupils were equal and responded to light and convergence. The field of vision of the left was a little contracted, (fig 2) the tension was normal.

O.E.H. The disc presented a normal appearance and there were nowhere any haemorrhages in the fundus. In the macular region there were some faint, scattered, yellow spots. The

nature of these was somewhat doubtful. They had not the pearly whiteness nor the stellate arrangement typical of albuminuricretinitis, but rather resembled the senile changes which are sometimes seen, usually however, at a more advanced age than in the present case. Possibly they may have represented a very early stage of albuminuric retinitis. I have once before seen a similar appearance in a woman of about the same age who had albuminuria, but in that case also I do not know whether a more typical appearance of albuminuric retinitis was presented later.

D.E.F. (fig 3) showed a picture very similar to that described in the last case. In this case, however, the region of the disc was more encroached upon by the haemorrhages, indeed it would have been almost indistinguishable from its surroundings had it not been for the point of entrance of the central vessels which, although a little hazy was clearly enough to be seen. The difference in calibre between the arteries and the veins was very marked here, the veins were thick, dark in colour, extremely tortuous, becoming dim or disappearing in places as if dipping beneath the underlying structures. The arteries did not seem to be actually diminished in calibre though very much smaller than the veins and pursuing a much straighter course. The haemorrhages generally were smaller than in the previous case, and probably as a result of this both arteries and veins could be better traced

into the periphery. There was little sign of degeneration or absorption taking place in the haemorrhages. In the macular region, which was strewn with small haemorrhages, a yellow mottling was visible probably corresponding to the appearance described in this region in the other eye. The media were fairly clear.

The further course of this case I do not know as she did not again present herself for examination. This was the more unfortunate because although the suspicion of Bright's disease was raised, in view of the extremely small quantity of albumen in the urine, and of the failure to find tube-casts the diagnosis could not be made with certainty without confirmation from subsequent examinations of the urine. It is to be remembered how many illnesses are diagnosed as "influenza" and although her illness presented points of resemblance to true influenza it might well have been dependent upon chronic kidney mischief, the course of which is frequently characterized by apparently slight illnesses. It need hardly be pointed out that ocular symptoms are not infrequently among the first to appear in Bright's disease. Whether, however, a chronic nephritis were present or not the argument advanced in the beginning of this thesis would still hold good namely, that in view of the intense degree of the changes, and of the fact that they occurred only on one side, there must have been some local as well as general causative agent. The extreme swelling and tortuosity of the veins would point to an obstruction to their

outflow. The case differs in some minor points from those of Michel; the patient was younger than the youngest (51) observed by him; there was no proof of wide-spread arterioscleroses; with the onset of dimness of vision there were entoptic phenomena which Michel expressly states not to occur. Haab (78) however, disagrees with Michel on this last point, and says that he has seen "prodromal obscurations and phosphenes", though usually the disturbance of vision occurs without these. Why the vision of both eyes was affected at first is not very clear; perhaps there was some macular haemorrhage in the right eye which had disappeared leaving the faint traces before mentioned when the woman was first examined. It would I think be very uncommon to find uraemic amaurosis without more general symptoms of Bright's disease than this patient presented; besides which the loss of vision was far from complete.

Case 3.

A young lady, a doctor, between 20 and 30 years of age, consulted me on April 26th, 1900, on account of a diminution of vision in the left eye of about one month's duration. She had always considered herself very healthy, her only serious illness having been an attack of bronchopneumonia two years before from which she had made a perfect recovery. There were no subjective signs of cardiac disease; no history of palpitation or

dyspnoea.

In February, 1900, she had a slight attack of Influenza which lasted about a week. This left her somewhat anaemic, and she was taking a tonic containing iron, arsenic and strychnine on this account. About the middle of March she found on awakening one morning that she could not see well with the left eye. She knew positively that it had been well the night before because she had happened to be looking at some objects with the other eye closed. Everything before the affected eye was indistinct, letters of even large type were all uneven and blurred, and she was unable to recognise faces in the street. Vision was especially bad in dull light or when she was tired. There never was any change in the external appearances or tension of the eye. At first there was a slight feeling of fulness as if the eye were slightly swollen, but there was never pain. A certain amount of improvement had occurred when she was first seen, but the visual acuity of the left eye was below 20/200, that of the right being 20/20 and Jaeger No.1 with ease.

The external appearance of both eyes was normal, the pupil responded to light and the tension was not raised.

O.E.R. The fundus was strictly normal.

O.E.L. (fig 4) showed an intense haemorrhagic retinitis, the fundus being strewn in every part with haemorrhages of all shapes and sizes but especially large and numerous near the posterior pole of the eye. The veins were

highly engorged, the arteries for the most part concealed. The media were perfectly clear. There were no signs of haemorrhage in the vitreous.

I had not the opportunity of examining the eye again, but the lady had it examined by Dr. Carline, (20.2.01) who stated that the haemorrhages had entirely disappeared; the disc appeared a little pale but not beyond the limits of the normal. Distant vision had risen to b/b and near vision to Jaeger No. 1. This improvement had taken place especially in the previous three months. Very distant objects still appeared a little blurred, the effect being compared by her to that of a large stop in a camera in place of a small one. Her general health had continued throughout good.

This is a very interesting, and in regard to its etiology a very obscure case. The ophthalmoscopic picture was that of thrombosis of the central vein, yet if such were the lesion how did it arise? It occurred at an age long before arteriosclerosis could be looked for except in connection with advanced chronic Bright's disease and even with this

arteriosclerosis would not be common.☆ Like the two previously reported cases she had had an attack of influenza, but the illness had not been nearly so prostrating as to give any grounds for believing that a marasmic thrombosis might have taken place, more especially as the dimness of vision did not occur till a week or two after the attack had passed off. Influenza by its weakening effects and probably by the action of some definite toxine can set numerous eye diseases going or cause those accidentally acquired to pursue a more intractable course, but it is not known to have any eye sequelae peculiar to itself.

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☆ Yet I have once seen rigid and thickened arteries with hypertrophy of the left ventricle in a child of 10. She suffered from advanced chronic nephritis with typical and advanced albuminuric retinitis. When last seen the child was rapidly sinking. See also a similar case in a girl of 12 reported by Arnold and Lawson, Trans. Ophth. Soc., of the United Kingdom, Vol. 18, p 140.

Possibly in the case under consideration the toxine of influenza gave rise to a phlebitis or periphlebitis as a consequence of which, helped by the anaemic state, there was a gradual building up of a thrombus which became completed and caused a blockage during sleep when the circulation would be at its slowest. This, however, is mere hypothesis, and even if it were granted to be possible we should be met with the difficulty of explaining why such a process of phlebitis and thrombosis should localize itself in the central vein of one eye while there was no evidence in any other part of the body of any similar disease.

This case is also of interest as showing that a haemorrhagic retinitis reaching quite up to Michel's second grade of intensity can clear up leaving practically no trace behind and with normal visual acuteness.

Case 4.

Unilateral haemorrhagic Retinitis associated with Cardiac Disease.

Margaret M., aet 25.

Presented herself at the Glasgow Eye Infirmary Dispensary, on August 24th, 1900, complaining of dimness of vision of the left eye of about a fortnight's duration.

The patient had suffered since early childhood from "heart disease". Its origin was obscure; there was no history of chorea,

rheumatism or other severe illness. She had been liable for many years to be laid up at times with attacks of cardiac insufficiency, and this had happened three months previously when she was in Edinburgh Royal Infirmary with cardiac disease and left "pleurisy". A fortnight before she was seen, while she was not confined to bed but was going about with as much freedom as she could ever use, there came a sudden mistiness over the sight of the left eye. The eye had been previously quite normal. There were no prodromal entoptic phenomena, and the dimness of vision was in the form of a uniform grey veil. Since that time vision had been getting worse rather than better.

Present Condition. Patient presented very evident symptoms of cardiac disease. She was very breathless on slight exertion; there was considerable cyanosis of the lips and cheeks and slight oedema of the ankles.

The cardiac impulse was forcible, appreciable over a large area of the chest wall but best palpable in the fifth interspace a little outside of the nipple line. The heart's action was very irregular and "tumultuous", and thrill was present though it was rather difficult to appreciate its relation to the apex impulse on account of the great irregularity. The limits of cardiac dulness were enlarged to the left. Auscultation revealed a rough murmur preceding and running into the first sound. The first

sound itself was loud and "slapping" in character. The diagnosis so far as the heart condition was concerned was; mitral stenosis with considerable general venous congestion and cardiac embarassment. The lungs showed no evidence of disease. The urine was unfortunately not examined.

Both eyes were normal in external appearance, the pupils equal and responding to light and convergence, and the tension not raised.

V.A.R. 20/20 and Jaeg. 1, V.A.L. distinguishes light and shade. D.E.R. Normal. The veins possibly a little engorged, but not remarkably so.

D.E.L. (fig 5) Radiating in every direction from the situation of the disc there were numerous haemorrhages scattered over the entire fundus. In this case many of these were very large, and most of them showed large yellow plaques in their substance such as are usually held to indicate that haemorrhages are absorbing. In many places the appearance was that of a yellow plaque streaked and rimmed with dark haemorrhage. The haemorrhages presented almost universally the striated appearance characteristic of extravasations into the nerve fibre layer. Only the central area of the disc was free; the edge was encroached upon from every direction. What remained of the disc was very red and hazy looking, but did not present the appearance of much swelling. The difference in the appearance of the vessels was very typical. Except for a very occasional appearance in a part of the

retina free from blood, the arteries were only visible on the small portion of uninvaded disc surrounding the point of entrance of the central vessels. The veins on the contrary were very prominent in the ophthalmoscopic picture, though also hazy or interrupted by haemorrhages in places. They were thick, very dark, and took a zig-zag and twisted course and could be fairly well traced into the periphery.

This case was not subsequently traced. Like the last one described it occurred in a young woman, but the etiology is here more easily made out inasmuch as there was very definite cardiac insufficiency with general venous congestion. This in itself will not produce retinal haemorrhages but it forms a predisposing circumstance to the occurrence of some local lesion in the ocular circulation, which, as has been before discussed, might most probably be in the form of a thrombus. It was by no means clear in this case why, with only a fortnight's duration of dimness of vision there should have been so many yellow plaques in the fundus, a condition which is usually associated with haemorrhages of long standing. She herself had no doubt at all that the eye was previously quite healthy, and indeed the history seems to point not to a previous amblyopia accidentally discovered, but to the definite onset of haemorrhage in an eye previously normal or nearly so. It might be said that the yellow plaques were of old standing but that the present dimness of vision arose from the recent haemorrhage. The plaques were however, I think, too

extensive not to have been associated with a very marked diminution of vision had they been previously present, and though it is true that a very considerable degree of amblyopia in one eye is often overlooked by patients, if the other eye is sound, yet probably if this had been the case in the present instance the woman would not have made so definite a complaint of mistiness coming over normal sight. I have not arrived at any satisfactory explanation of the appearance. Observation of its further course might have cleared up the difficulty.

Case 5.

Unilateral haemorrhagic Retinitis of moderate Degree with small Arteries and greatly swollen tortuous Veins. Edward C., aet 44, a Boilermaker.

Presented himself at the Glasgow Eye Infirmary Dispensary on February 9th, 1901, complaining of dimness of vision in the right eye of a fortnight's duration.

Heredity. His mother died of apoplexy at the age of 59; his father of a "fever".

There were five of a family of whom three died in infancy.

Personal History. When eight years of age patient had smallpox; when twenty-four "inflammation of the lungs" (acute); when twenty-nine tonsillar abscess. Two years ago he was troubled for a time with palpitation, perhaps brought on by heavy smoking. It did

not cause him to leave his work and had not been present since. He had never had headaches, pain in the back, oedema or other symptoms of nephritis. There was no specific history, and he was moderate in the use of alcohol. He smoked about 3 oz. of heavy tobacco per week. His work was on the whole light, and did not involve much heavy-lifting or stooping. The bowels were regular.

A fortnight before he was first seen he noticed upon rising one morning an appearance of fog or smoke before the right eye. He was sure that the eye had been quite normal the night before, indeed the right was his best eye, and he was accustomed to shut the left when he made a minute examination of any object. The smoke was at first moving and curling before the eye and there were occasional lights and flashes. There was no pain in the eye or head, and his general health was quite as usual. He had been undergoing no unusual exertion on the previous day, and had been indulging in no excesses. During the following week the condition of the eye remained unchanged; there were occasional movements in the clouds in front of it. Since then there had been a slight improvement in vision.

Present Condition. Patient was a healthy looking man. There was no arcus senilis, the radial arteries were slightly tortuous and rather too easily felt. The pulse numbered 82; was regular and of normal tension. The chest showed something of the barrel-shape

of emphysema, and the cardiac dulness was perhaps a little encroached upon, but neither in the breath sounds nor in the heart sounds was there anything noteworthy.

Urine Oc. Sp.G. 1010. No albumen or sugar.

Both eyes were normal to external examination. The pupils were equal and responded to light and convergence, and tension was normal.

V.G.R. 20/200. V.G.L. 20/70.

The fields of vision were practically normal and the colour sense was good.
O.C.R. (fig 6) The most noticeable point in the ophthalmoscopic picture was the great enlargement of the veins which stood out very darkly, and were extremely tortuous and broad. The arteries on the other hand were certainly not enlarged, and were possibly smaller in calibre than the normal. There was a faint haziness about the whole retina especially in the region near the disc suggestive of oedema, and in places the veins were not quite so well seen, as if there were a semi-transparent veil over them. The disc was congested and its margins ill-defined especially on the temporal side. On the nasal side the choricidal ring could be faintly distinguished. Scattered over the visible fundus there were many striated and punctiform haemorrhages. These were more numerous and larger in the vicinity of the disc than in the periphery, more especially the vicinity of the inferior nasal vein. O somewhat large but rather broken up haemorrhage stretched out from about the centre of the temporal border of the disc in

the direction of the macula, and there was a punctiform haemorrhage apparently right on the site of the fovea. The disc itself was streaked with some small haemorrhages, and there was a little irregular one in the centre. The haemorrhages in the periphery were mostly in the vicinity of the veins, but also occurred in parts of the fundus where no large vessels were visible.

D.E.L. showed a high degree of mixed astigmatism, roughly about +1D / -3D a finding confirmed by the ophthalmometer. The fundus itself except for the haziness of details induced by this astigmatism was normal, and the relative calibres of arteries and veins were not noteworthy. Correction brought the visual acuity of this eye considerably up, though only to 20/40.

On March 23rd, 1901, the visual acuity of the right was 20/100, on April 6th it had risen to 20/70 with a little difficulty, the visual acuity of the left being as before 20/70. He had been in good general health in the meantime. The ophthalmoscopic appearance in the left eye was unchanged. In the right the disc was of a somewhat dirty-white tint and the details of it not very clear. The arteries seemed rather small and the veins were very much less swollen than formerly, and pursued a much straighter course, in fact they could not be said to be greatly abnormal. All the haemorrhages had entirely

disappeared except for a small speck on the disc near the centre, and a little to the temporal side, evidently a remnant of the haemorrhage described above in this position. Ground the disc to the temporal side there was a narrow yellowish band showing some pigment disturbance, the appearance having some resemblance to a myopic crescent. The refraction of the eye was approximately emmetropic. (fig 7)

This may be taken as a typical example of Michel's third grade of intensity. In its further course also it agrees with Michel's description, inasmuch as in about two months time the haemorrhages had practically disappeared leaving a slightly atrophic condition of the disc. There remained, however, but little disproportion between the relative sizes of arteries and veins as described by Michel. As regards the etiology there was some degree of arterioscleorosis in the peripheral vessels, and if we suppose the lesion to have been a thrombus, the starting point would seem to have been merely the lowered blood pressure during sleep, since there was at the time nothing in the state of his general health to act as a predisposing cause for thrombosis. There was no evidence on physical examination of cardiac insufficiency or of kidney disease, and neither his personal habits nor the nature of his work seemed to throw much light on the subject. As there was no colour scotoma it may be taken that his heavy use of tobacco had not done his eyes

any damage, indeed it would be extremely questionable if tobacco intoxication would in any way predispose to (as it certainly would not in itself cause) the ophthalmoscopic changes described above. The low visual acuteness of the left eye is fully accounted for by the high grade of astigmatism which was present.

Case b.

Unilateral Haemorrhagic Retinitis of moderate Intensity; a Trace of Albumen in the Urine.

Jas. R., aet. 47, Roadsurfaceman.

Presented himself at the Out-door Department of the Glasgow Eye Infirmary on September 29th, 1900, complaining of dimness of vision in the left eye of about 2 months duration.

His mother died of apoplexy; otherwise there was nothing remarkable in his heredity. He had enjoyed in general excellent health, but a year before he had been ill for ten weeks with "congestion of the right lung and diarrhoea". No symptoms pointing in the direction of Kidney disease were to be elicited.

Two months previously there had been sudden failure of vision in the left eye for which he was unable to assign any cause. Since that time his sight had neither become much worse nor much better. Usually there was merely a general mistiness over the whole field of vision, but sometimes in addition black specks seemed to hover before the eye.

Present Condition. Patient was a strong and very healthy looking man. Signs of arterial degeneration were doubtful in the radial arteries and not present in the temporal. There was no arcus senilis. The limits of cardiac dulness were normal, the heart sounds free from murmurs, and examination of the lungs revealed nothing abnormal. The urine was acid, Sp. G. 1010. No sugar. On standing for some time with Nitric Acid in the cold a very faint cloud came down. The presence of albumen was considered at first doubtful, but a subsequent examination two months later gave the same reaction, and as extremely scanty but still quite definite casts (hyaline and epithelial) were to be found, we may believe that there was chronic kidney mischief.

The external appearances and tension of both eyes were normal, and the pupils were equal and responded to light and convergence.

V.O.R. 20/20 and Jaeg. 1. V.O.L. 10/200.
O.E.R. The fundus was normal. There were no macular degenerative changes and no abnormal appearances of the vessels. The refraction was emmetropic.
O.E.L. (fig 8) The haemorrhages in this case seemed to be all, or nearly all confined to the nerve-fibre layer. Except for a few small round ones in the vicinity of the macula they all presented the striated appearance characteristic of haemorrhages in that layer. They were mostly small and pretty evenly scattered throughout the visible fundus though there were some larger ones in

connection with some of the vessel branches. The veins were somewhat engorged and tortuous, but not nearly to the same extent as in the cases previously described, and similarly the arteries were not greatly abnormal in appearance. The position of the disc was quite easily distinguished yet its edges all round were fuzzy and indistinct. This was especially the case to the nasal side where also congestion was very marked. The disc was but little encroached on by haemorrhages. There were no signs of degenerative spots in the macular region.

The patient was seen again on November 24th, 1900. He had been in good health since his previous visit, and he thought that there had been a little improvement in his vision to the temporal side. Central vision had, however, sunk a little - to 6/200. The ophthalmoscopic appearances had only slightly changed. The disc seemed a little swollen and was probably redder and more hazy. The veins were more engorged, and near the disc seemed in places buried under a hazy film. The arteries were unaltered and the haemorrhages much as before; showing, however, a few yellow plaques among them. The right fundus was as before perfectly normal.

It might be doubtful whether this case strictly belongs to the same class as those already described inasmuch as the marked appearance of strangulation of the venous system present in them was absent here, the

relative calibre of the arteries and veins not being greatly abnormal. Even a slight obstruction of the central vein is said to cause a marked engorgement of the retinal veins. The question arises whether it was perhaps a case of unilateral albuminuric retinitis perhaps to be followed later by similar disease of the other side. ☆ Such cases are of course known, but in the present instance the retinitis was not of the ordinary degenerative albuminuric type, but was chiefly characterized by haemorrhages, and whether there was a noxious agent of general action or not, the argument already advanced will hold good, namely that where symmetrical structures in the body are attacked only upon one side we must look for some local cause upon that side in addition to any poison which acts upon the system generally.

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Uwert's case is probably the best known in which at the post-mortem only one kidney was found. In the present instance, however, it is unlikely that a man with only one kidney and that sufficiently far advanced in disease to give rise to retinitis would be going about apparently in his usual health.

Thus in the present instance the retinal haemorrhages were tolerably profuse and confined to the one eye, and therefore we are bound to suppose some disease or obstruction of the vessels in that eye which was not present on the other side, although of course the toxic agent resulting from the nephritis must have affected both sides equally.

The onset of dimness of vision was not gradual in this case, as is usual in albuminuric retinitis but sudden and followed after two months by little or no improvement, and this would point to some definite lesion having taken place at that time such as would cause a sprinkling of haemorrhages in every part of the fundus. One is certainly tempted to believe that this must have been a thrombosis, yet as already pointed out the appearances were not typical of the accepted ophthalmoscopic picture of thrombosis. The man was younger than the average usually given though not younger than the average of my own cases. Signs of arteriosclerosis were doubtful and there had not been any weakening illness for so considerable a time as practically to exclude its influence in the case. The ophthalmoscopic appearances may have changed considerably in the two months before he was seen, but of course this is mere conjecture. The fact that the man was apparently in his usual health would not exclude Bright's disease from a share in the causation of the retinal disease, since it is a common

experience in Eye Cliniques to find patients with well marked albuminuric retinitis from whom no history of subjective symptoms can be elicited even on careful inquiry. The disease seemed to be advancing rather than receding when he was last seen. Looking at the case generally I should be inclined to say that there was proof of local vascular disease in the left eye to the etiology of which a chronic interstitial nephritis probably stood in some relation. Of the exact nature of this vascular disease I should leave the question open.

In the two following cases I had an opportunity of carrying out a pathological examination on eyes affected with unilateral haemorrhagic retinitis. In both cases the eyes were enucleated for haemorrhagic glaucoma with persistent pain in spite of operation, a source from which nearly all the material described in the literature of the subject has been derived. Unfortunately I did not see either of them till after enucleation had been performed, so that I have had to take the clinical details of the affected eyes from the Ward Journals which were not of course written with a view to special research. I was, however, able to make a detailed examination later of the patients' general condition, and of the state of the unaffected eyes.

Case 7.

Glaucoma of right eye; Iridectomy followed by intraocular Haemorrhage; Persistent Pain; Enucleation; Haemorrhagic Retinitis.

Jane Mc J. aet. 40.

First presented herself at the Glasgow Eye Infirmary Dispensary on August 1st, 1899. She was under the care of Dr. J. Spence Meighan, to whom I am indebted for permission to use the case. The following notes of her condition at the time are from the Infirmary Journals.

The right eye was glaucomatous. Tension +1; the cornea hazy; the pupil irregular and dilated. There was no perception of light. After treatment as an out-door patient she was admitted to Hospital on August 29th, the tension being +3 and the eye painful, and on the following day an upward iridectomy was performed. The iris was fragile and difficult to remove, and tension was noted not to be lowered after the operation.

1.9.99. Anterior Chamber full of blood. Conjunctiva red. Not much pain since the operation. T+1.
3.9.99. Chemosis; less pain; Gtt. Eserin in right.
6.9.99. A little haemorrhage on the dressings; Painless. Still blood in the Anterior Chamber and the iris invisible.
9.9.99. Haemorrhage from the wound last

night. Much easier to-day.
13.9.99. Enucleation of Right under Chloroform. 3 ? in left.
19.9.99. Dismissed.

The following notes were made by me about twenty months after her first appearance in April, 1901.

Her previous health had been on the whole good, but twice (13 and 5 years ago) she had had "inflammation" of the right lung.

For some years back she had been subject to headaches and had suffered much from palpitation and dyspnoea on exertion. There was no history of oedema or of pains in the back, etc. She had had seven children of whom one suffered from hip-joint disease and one had died in infancy from inflammation of the bowels. The pregnancies and labours had all been normal, and there was nothing either in the history or in her appearance to suggest specific disease.

About one month before she first sought advice she began to be troubled with pain in the right eye and forehead gradually increasing until it became constant and very severe. The eye appeared red and inflamed and the sight became gradually worse until in about a fortnight it was completely lost.

The patient was a somewhat sparely-built woman. The radial and temporal arteries were decidedly rigid to the feel. The pulse numbered 96 per min. and was highly irregular in force and rhythm. The cardiac apex beat was in the fifth interspace about 3½ inches from the mid-sternal line.

Apart from the irregularity of the heart's action it presented no abnormality; there was no thrill. The upper border of cardiac dulness was at the 3rd. rib, the right was displaced to about the right border of the sternum, and the transverse measurement was about 4¾ inches. The heart sounds were extremely irregular both in force and rhythm but no definite murmur could be detected. What appeared to be the second sound both at the apex and in the tricuspid area was re-duplicated giving a "cantering" action. Physical examination of the lungs revealed no abnormality.

The urine was acid. Sp.G. 1002. There was no sugar but a definite trace of albumen, and scanty hyaline casts were found microscopically.

The patient was illiterate but with the left eye counted the letters of 20/20 with the utmost ease. The left eye was normal in external appearances; the pupil responded to light; the tension was not raised; the anterior chamber was somewhat shallow. The right socket was healthy. The field of vision was slightly contracted temporally. (fig 9) R.E.L. The disc presented no noteworthy features and the veins showed no engorgement but were in comparison with the arteries of about normal breadth. In places they showed some irregularity in their calibre in the form of a concentric narrowing and slight dimness extending for a short distance along their course, the appearance being suggestive of a localized narrowing of the blood stream by a thickening of the vessel

wall. Where the arteries crossed the veins there was an appearance of an interruption or constriction of the vein as if the artery also had less transparent coats than normal so that the vein did not show so clearly through them.☆ There was no trace of cupping of the disc, and nowhere were there any haemorrhages or traces of previous haemorrhages.

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☆ Appearances similar to this have been described by Marcus Gunn (Trans. Ophth. Soc. of the United Kingdom 1897-98 p. 35b) who holds such changes to be important evidence of general arterial disease, and in support of this shows that many of the cases which presented them subsequently died of apoplexy. The present instance would tend to confirm his views inasmuch as though she had no untoward general symptoms when last seen, yet there was great cardiac irregularity, and the occurrence of haemorrhagic glaucoma in the right eye may be taken as proof of vascular disease in it.

Pathological Examination of the enucleated Eye.

Macroscopic Examination, (by Dr. Leslie Buchanan.)

"The eyeball is of peculiar shape, being oval vertically at the equator. The cornea is of the normal curvature. The anterior chamber is very shallow from pressure forwards of the lens which is large, measuring 10.5 m.m. by 4.5 m.m. The anterior chamber is rather more than half filled with blood which is partly fluid. The iris is adherent to the base of the cornea peripherally; there is no evidence of disease of the ciliary body, and neither in the iris nor in the ciliary body is there any inflammatory effusion.

The vitreous is deeply tinged with blood colouring matter, and possibly contains blood itself. There is certainly a considerable quantity of blood lying on and in the retina as if from the larger retinal vessels. There are large numbers of haemorrhages in different parts of the retina and at different depths in its substance. There is one very extensive haemorrhage lying on the surface of the retina at the inner part of the optic nerve entrance. The optic nerve entrance is not cupped but seems swollen."

Microscopic Examination.

Cornea. The surface of the corneal epithelium presents a less smooth and more ragged condition than normal, and the cells have a crushed together and flattened appearance, but this is possibly the result of some accidental circumstance in the fixing and hardening (in formalin). Between the epithelium and Bowman's membrane there is a granular-looking line of little highly refracting particles (? minute spaces or crystals). The membrane of Bowman itself is normal, and shows none of these, but in the corneal fibres especially the more superficial they occur very numerously. (fig 10) The deeper fibres near the membrane of Descemet are nearly or altogether normal. Where the corneal fibres pass into scleral fibres the granular appearance ceases entirely. The membrane of Descemet takes on a haematoxylin stain (Ehrlich's) more diffusely than normal, and the endothelium is not well preserved. In the corneal limbus there is marked distension of the episcleral vessels, a distension which is also markedly present further back. There is perhaps some cellular infiltration of the limbus but it is not marked. (fig 11) Infiltrations of blood are present in some sections but probably occurred at the time of enucleation.

Iris and Ciliary body etc., The iris is less spongy in structure than normal and more fibrous. It contains numerous vessels the

walls of which show thickening especially of the Tunica media. The intima is intact and not proliferated. (fig 13) The sphincter iridis is represented by a distorted clump of muscle fibres at the end of the iris. (fig17) Pigment cells are present in places in the iris especially in the region of the distorted sphincter, but they show nothing of their usual branched form and are merely rounded balls. (fig 17) The root of the iris is firmly adherent not only to the ligamentum pectinatum, but also for some distance to the membrane of Descemet. (fig 12) As a result the canal of Schlemm is fully a millimetre removed from the angle of the anterior chamber and quite shut off from it by fibrous tissue. (fig 12) Where the iris is adherent to the cornea it is very thin and its structure is very dense. In the angle of the Anterior Chamber there is a considerable mass of well-preserved blood-corpuscles, probably the remains of the haemorrhage which occurred after the operation as mentioned in the clinical history. (fig 13) Blood corpuscles are found in large numbers among the fibrous tissue which connects the iris with the back of the cornea. Behind the position of the membrane of Descemet they probably occuby what remain of the spaces of Fontana. Where the blood is not present no spaces of Fontana are visible among this tissue. The Canal of Schlemm contains blood in some sections and is empty in others; in some

there is a large blood-filled vein in close connection with it. (figs 14, 15)

On the Anterior surface of the non-adherent portion of the iris there is a new-formed connective tissue of spongy texture, somewhat resembling iris stroma. Its meshes are crammed with blood corpuscles, among which however the light supporting stroma is visible and is seen to contain some small vessels, some of which can be traced into connection with the vessels of the iris. (fig 16) It would be difficult to say whether this haemorrhage has occurred in a new-formed tissue, or whether it has occurred in the iris itself and split off a superficial layer. It might possibly be that a stroma had grown from the iris tissue into a haemorrhage which lay upon its anterior surface, but this explanation seems to be negatived by the fact that the whole haemorrhage is separated off from the Anterior Chamber and the blood in it by a definite layer of tissue which joins the membrane of Descemet peripherally. At the pupillary margin of the iris this layer of tissue is attached to the uveal pigment layer which it seems in contracting to have pulled considerably round, causing marked ectropium uveae and also disorganizing the pigment-cells and causing them to form the ball-like masses mentioned before. (figs 16, 17.) In some sections there is a large haemorrhage quite within the pupillary margin of the iris, which is also supported by a light stroma connected with the iris tissue. This haemorrhage is considerably more

disorganized than the others; the forms of the corpuscles though visible are badly preserved and do not stain so deeply with eosin as usual. (fig 17.)

The ciliary body presents on the whole nothing remarkable, except that there are a few small extravasations in its anterior part. (fig 18.)

Retina. The Pars Ciliaris Retinae shows some formation of vacuoles between its two layers of epithelium, otherwise it is not remarkable.

At the Ora Serrata there is great distortion of the retina. Only at about 2 m.m. distance from the ora are the two nuclear layers of the retina separately distinguishable, and about the same place the rods and cones first show anything like their normal structure. In this position also there are some haemorrhages in the nerve fibre and ganglion cell layer. At the Ora Serrata itself no retinal layers are to be made out. There is merely an irregular network with many spaces and here and there nuclei dotted over it. (fig 19.) The hexagonal epithelium is considerably disturbed at this point. Further back the arrangement becomes more regular. There is a somewhat thick layer having the appearance of the nerve-fibre layer, and external to this a layer of round nuclei usually in a single stratum, sometimes several deep. Of the rods and cones there are only distorted remnants. No certain traces of the ganglion layer can be found.

The hyaline membrane is adherent to the Ora Serrata and shows a few cell forms most probably chiefly wandering corpuscles. (fig 19.)

The remainder of the retina shows in every part numerous haemorrhages. In some places they are small and situated chiefly in the more internal layers (the nerve-fibre, ganglion cell and inner reticular.) In other situations they are extremely large, infiltrate the whole retina and cause very marked distortion. Some large haemorrhages are seen lying in front of the retina a position from which probably many others have been washed away in preparing the specimens. (fig 20) There are also many subretinal haemorrhages. (fig 21) At the site of some of the larger haemorrhages the retina is thickened as much as 4 to 5 times. This thickening is mostly caused by a new-formed tissue closely packed with blood corpuscles, having the appearance not so much of ordinary connective or cicatricial tissue as of an immense overgrowth of the fibres of Müller. (figs 22, 23) Some irregular indications of the nuclear layers of the retina are still preserved among the red blood corpuscles. This thickening contains numerous vessels often showing signs of endarteritis and perivasculitis.

At the site of the subretinal haemorrhages the retina shows much thickening and folding and an indefinite arrangement of its layers, the two nuclear layers especially tending to run into one another. (fig 21.)

Where the retina is not infiltrated

with blood the nerve-fibre layer is highly atrophic, showing a loose vacuolated meshwork probably representing chiefly or entirely the supporting framework of the retina, the Müllerian fibres, with hardly a trace of the nerve elements of the nerve fibre-layer. (fig 24) There is no regular layer of ganglion cells though an isolated cell is to be found here and there. The inner reticular layer, while fairly preserved in parts is elsewhere much atrophied and represented by thin horizontal lines with hardly any reticulum amongst them. (fig 24) The inner nuclear layer shows a good deal of vacuole building with scattering of its nuclei. The outer reticular layer is indefinite and a good deal encroached upon by the nuclei of the outer nuclear layer. (fig 24.) This latter is the best preserved of the layers, and even in the places of greatest thickening and distortion can always be followed, indeed in some of these places it seems thickened. The rods and cones are separated from the pigment epithelium by a homogeneous coagulum (containing in places the subretinal haemorrhages) figs 21, 25.) ☆

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☆ U. Gracle (loc. cit.) discussing the nature of this coagulum says that it is an alkali albumen; that it does not occur in normal eyes but represents a subretinal fluid present during life.

Though their forms are distinguishable in places especially where the retina is folded, they are in no place well preserved, and in some localities are almost entirely destroyed. The appearance described by Deutschmann of nuclei passing from the outer nuclear layer among the rods and cones is I think present in places though rarely. (fig 25)

The retinal vessels show a certain amount of thickening of their walls chiefly of the adventitia. The change affects both arteries and veins, and in rare instances amounts to almost complete obliteration. (fig 24) Nevertheless many normal looking vessels are to be seen, and taken as a whole the deviation from normal is not a marked one.

The pigment epithelium adheres to the chorioid and shows nothing remarkable.

Papilla and Optic Nerve. The lamina cribrosa is perhaps a little excavated but not to a great extent. (fig 26) The papilla is not swollen and there are no haemorrhages in its substance. In the papillo the vessels do not show very remarkable changes; the vein may be said to be normal while some of the small arteries show pericarteritis. (fig 27) The central artery itself, however, does not show any marked thickening of its walls on the papilla. The whole papilla is small and atrophic looking.

The optic nerve is also small and atrophic. (figs 28, 29) The nerve bundles stain diffusely and badly and have a granular

appearance, and there is an undue number of nuclei among them. The large faint-staining (? neuroglia) nuclei present in the normal optic nerve are not to be found, the nuclei here being small and deep-staining. There is an appearance among the nerve bundles in places of grouping of the nuclei into little masses. The connective tissue septa are increased in bulk and more cellular than normal, and there is some cellular infiltration of the connective tissue around the central vessels. (fig 20) The whole lumen of the vein is not included in any of the sections, but the portion which is present shows some thickening of its wall, and in some of the sections there is evidence of proliferation of the endothelium. These changes, however, are not of high degree. The central artery is normal. A little vascular twig lying between the artery and the vein has its lumen blocked by a fibrous-looking mass in which are one or two nuclei. (fig 30)

The Lens presents nothing noteworthy.

## Critique.

The chief pathological changes found in this case were; (1) adhesion of the root of the iris to the back of the cornea at the corneo-iritic angle; (2) haemorrhages in the anterior chamber, the iris, the vitreous and the retina, (3) atrophy of the conducting

nerve elements in the retina and in the optic nerve. We must now discuss the sequence in point of time of these phenomena, their dependence upon one another, and their relation to the general state of health.

Two different hypotheses might be put forward which would account for the appearances and be in accordance with known facts. (1) We might say that in a woman the subject of vascular disease, and probably as a result of this vascular disease, there had occurred a haemorrhagic retinitis in the right eye probably with simultaneous or subsequent haemorrhage into the vitreous, and that, following upon this, there had occurred an attack of glaucoma very little amenable to operative treatment. That such a glaucoma may follow upon haemorrhagic retinitis is amply borne out by the numerous cases cited in discussing the literature of the subject.
(2) On the other hand it might be said that a primary glaucoma having occurred in a woman the subject of vascular disease, there took place a profuse intraocular haemorrhage from the diseased vessels within the eye when the eye was opened at the time of the iridectomy. Such an intraocular haemorrhage more or less profuse is of course well known to occur occasionally after operative interference in those with diseased vessels. The vascular disease presupposed in both these hypotheses is in the present instance sufficiently proved by the rigidity of the radial arteries, the great cardiac

irregularity without definite murmurs, and the condition of the vessels in the other eye (see p.60) The presence of albumen in the urine also, whether primary and the cause of the cardiac condition or secondary to it, furnishes a cause of vascular disease. ☆

Since glaucoma was present when the woman first presented herself it is impossible definitely to say whether or not there had been retinal haemorrhage before its onset. Further, since there is no note of any ophthalmoscopic examination before operation (probably because the media were too hazy to permit of it), it is quite possible that there were no haemorrhages until the iridectomy was performed.

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I should be inclined to regard the renal condition as primary here in view of the low specific gravity of the urine, the evidence of arteriosclerosis in a person of 40, and the "cantering" action of the heart without definite murmurs but with a slight degree of enlargement.

The mass of evidence both clinical and pathological inclines me to believe that the iridectomy was indeed the starting point of the haemorrhages. That is to say, that there was first a simple primary glaucoma, but that owing to the diseased state of the vessels the iridectomy was followed by an intraocular haemorrhage. From the clinical side the history of the onset was typically that of primary glaucoma and not that of haemorrhagic retinitis. It was not with quick or sudden mistiness over the vision, but with pain in the head and eye and gradual loss of sight in a fortnight the eye meanwhile showing symptoms of irritation. Again intraocular haemorrhage was actually observed clinically in the anterior chamber after the operation. Pathologically the well preserved form of the blood-corpuscles in the anterior chamber confirmed their recent origin. In the iris and in the retina the corpuscles were equally well preserved, and although it is true that they would not so soon lose their forms in these tissues as in contact with the aqueous humour, yet we may probably take it that these haemorrhages also were of comparatively recent origin. On the other hand the changes in the iris were evidently of some standing; there had been sufficient cicatricial contraction to greatly distort the sphincter pupillae and to cause much ectropium uveae, changes hardly likely to have occurred in the fourteen days between the iridectomy and the enucleation.

The iris also was adherent to the back of the cornea by well-formed connective tissue. The duration of the symptoms from the first complaint to the time of enucleation was two and a half months, and the appearances in the iris were quite consistent with so long a duration. Indeed there was evidence of disease of the iris when she was first seen inasmuch as the note runs that the pupil was "irregular and dilated"; and at the time of operation also the iris was found to be "atrophied and fragile." There seems then to be proof that there had been a chronic inflammation of the iris of considerable duration, and it is worth considering whether it may have been not secondary to the glaucoma but the cause of it. The point can hardly be settled from the pathological evidence but at any rate clinically the glaucoma was of the so-called primary type, i.e. it was not preceded so far as the history tells us by any other evident disease of the eye.

If the above reasoning be correct we might attempt to put down the sequence of events in the present case somewhat as follows:- According to the modern theory of glaucoma we should say that, from some cause or other the filtration angle of the anterior chamber became blocked. This cause may have been in the first instance merely a plastering of the iris against the cornea from some accidental difference in the tension of the anterior and vitreous chambers which carried forward the lens; or, as I

have just said, it may have been from the first an actual adhesion due to a very chronic process of iritis not showing itself by very definite subjective signs. At anyrate, however it arose, there was subsequently firm adhesion betwixt the iris and cornea, and hence the filtration of the eye was seriously interfered with and the glaucomatous state firmly established. This glaucomatous state would have a still further prejudicial effect upon the vessels of the eye already probably diseased from general causes as before discussed. The conducting elements of the retina and optic nerve would also suffer severely under the continued high tension, and this accounts for their atrophied condition in the sections. Upon this eye now, the operation of iridectomy was undertaken, which proved to be difficult, and followed both immediately after the operation and subsequently, by little or no decrease of tension. This was probably due in the first place to an intraocular haemorrhage occurring at the time of operation, and at anyrate there is no doubt that such had occurred two days after when the eye was first dressed and the anterior chamber found "full of blood." In consequence of this haemorrhage, and very probably of subsequent repeated haemorrhages, the tension did not come down, the pain continued, and finally enucleation became necessary.

In saying that probably this case began as a simple primary glaucoma I do not mean

that there may not have been a few retinal haemorrhages before the onset of the glaucoma. In view of the evidence of vascular disease it is very probable that there were. What I would maintain is, that there is no proof, and the clinical and pathological facts are against the assumption that there had been a severe unilateral haemorrhagic retinitis such as we have seen examples of in the literature and in the cases already described, before the onset of the glaucoma. The case therefore belongs strictly to a somewhat different category from those mentioned in discussing the literature, in which the haemorrhagic retinitis very definitely preceded the glaucoma, and if it be true that in them the haemorrhagic retinitis was the cause of the glaucoma the cause in the present instance must be something different. Yet although the pathological cause of the glaucoma is different, the pathological cause of the haemorrhages is much the same; for both in these cases and in the present one there was wide-spread arteriosclerosis and therefore presumably vascular disease of about equal degree in the two eyes. To this there was added a further local strain on the circulation of one eye, in the present instance in the form of a sudden lowering of previously high tension by an iridectomy, and in the other cases by a blockage of the main venous outflow by a thrombus. Thus in each case there was superadded to a general

vascular disease a local lesion acting only on one eye, and the result in both instances was the same viz. unilateral retinal haemorrhage.

One would say a priori that where a so-called "primary" glaucoma attacks one eye only there must be some local cause in this eye which is not present in the other. It is not uncommon to find glaucoma attacking an eye which is not, at least so far as we can tell clinically the seat of any other disease and in which therefore the glaucoma must be termed primary, the other eye meanwhile remaining very long or perhaps always free. Must we not say that in such an eye there must be some local lesion not present in the other to which the glaucoma is really secondary although clinically it must be called primary? No doubt there is blockage of the filtration angle but in a primary glaucoma what has caused such a blockage?

In the present instance twenty months after the occurrence of glaucoma in the first eye there was still no glaucoma in the other, but as I have just said this does not exclude primary glaucoma. The shallowness of the anterior chamber in the sound eye may perhaps indicate that there had been a similar condition in the eye attacked which helped to render it susceptible to a glaucomatous attack.

It was unfortunate in this case that only one half of the optic nerve was available for examination, and that that half did not

contain the whole of the central vein. It can only be said that in the part preserved there was no evidence of a thrombus.

The following similar case occurred in the Eye Infirmary under the care of Dr. Treeland Fergus to whom I am indebted for permission to use it. This case also I did not see till after enucleation and therefore the first part of the notes is from the Infirmary Journals.

Case 8.

Glaucoma of left eye; sclerotomy followed by no permanent Improvement; Enucleation; haemorrhagic Retinitis.

William W. aet. 52, a Plumber.

Was first seen at the Dispensary Department of the Glasgow Eye Infirmary on August 23rd, 1900. The note then made shows that the anterior chamber in the left eye was very deep; the pupil widely dilated; tension+2; the fundus illuminable with difficulty and no details visible; V.O.R. 20/20. V.O.L. Shadows. He was admitted to hospital two days later, and on August 27th a sclerotomy was performed which was followed for a time by marked relief from pain. Subsequently, however, pain and high tension returned, and the eye was enucleated Oct. 1. Of the condition of the right eye the only note made was that there was no cupping of the disc. The

visual acuity when he left hospital was, letters of 20/30.

On January 31st, 1901, I had an opportunity of seeing him and of going more fully into the case. His hereditary history was satisfactory. Both his father and mother died in old age. Of a family of eight, three died in infancy, and the other five were alive and well. Two years previously he had had an attack of Influenza followed by "congestion of the lungs" chiefly affecting the left side. Since this illness he had suffered from considerable breathlessness on the least exertion. There were no subjective symptoms of kidney disease. He had had no previous trouble with his eyesight. In the course of his work he was not subjected to any heavy lifts or strains, nor did it involve much stooping. He denied the excessive use of alcohol.

History of Illness. The details of the onset of amblyopia were as follows. A fortnight before he was first seen he had been exposed during a whole day to wet and stormy weather, and on awakening the next morning he noticed a mistiness over the sight of the left eye as if he were looking through a ground glass. There were no positive scotomata in the field of vision, and no entoptic phenomena. There were no halos around lights but only a general dimness before them. During the next five days the sight became gradually dimmer until it was almost lost, but there was no pain either in the eye or around it.

severe periorbital pain now set in in the forehead and cheek, so that sleep was almost entirely prevented up to the time of his admission to hospital. Sight meanwhile was practically abolished.

Present Condition. The patient was a stout, florid man and showed during examination an evident degree of breathlessness. The pulse numbered 108, and was full and bounding and of somewhat high tension. The radial and temporal arteries showed only a little evidence of arteriosclerosis. The area of cardiac dulness was not enlarged, and the cardiac sounds though loud and forcible were free from murmurs. Examination of the lungs showed only an occasional wheezing bronchial râle. Urine; Alkaline. Sp.G. 1010. There was a distinct amount of albumen; no tube-casts were found at a first examination, but at a subsequent one (6.4.01) unequivocal hyaline and granular casts were found. There was no sugar.

V.O.R. was 20/40 and a letter or two of 20/30; with a +3.5 D Sph. lens he could read Jaeger 1 with a little difficulty. The eye was normal to external appearance; there was no arcus senilis; the pupil responded to light and the tension was not raised. The field of vision was practically normal, and the colour fields had normal relations to the field for white and to one another. (fig 31) There was no central colour scotoma.

O.E.R. The media were clear. The disc showed a little obscuration of its edge on

the nasal side especially in the upper quadrant; this was very slight however, and doubtfully of pathological import. There was certainly no glaucomatous cupping of the disc but rather a little swelling. The arteries and veins were about normal in their absolute and relative calibres; there was no swelling or undue tortuosity of the veins. The same appearance as in the last case of interruption or constriction of the veins where they were crossed by the arteries was present here, but it was in a more marked and typical form.

This patient was seen again on April 6th, 1901. He had in the interval been in rather better general health though still pretty breathless. The pulse numbered 118 and was of high tension as before. He could read letters of 20/30, the tension was still normal, and the ophthalmoscopic appearances unchanged.

Pathological Examination of the Enucleated Eye.

Macroscopic Examination (by Dr. Leslie Buchanan.)

"The measurements of the eye are as follows:- Antero-posterior diameter 24.5 m.m; horizontal equatorial 24 m.m. The lens is very large measuring 10.25 m.m. in equatorial and 3.3 in polar diameter; it is considerably pushed forward obstructing the corneoiritic angle and completely filling the interciliary space. The coats of the

eye retain normal curves; the cornea is clear, the pupil semidilated, and there is considerable adhesion between the iris and the lens capsule. There is a corneal wound in the lower inner quadrant of the corneo-scleral margin to which the iris is adherent. The ciliary body shows no evidence of change. The vitreous is clear in front, opaque behind from the existence of blood-clot. There is very considerable thickening of the retina near and around the optic nerve entrance, and it is separated from the choriod by a thin layer of gelatinous substance. The optic nerve is not cupped but swollen 2 m.m. above the choricid; there is separation of the retina and thickening probably due to acute neuroretinitis. There are many retinal haemorrhages."

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Microscopic Examination.

Only portions of the eye were available for microscopic examination, namely a piece of the anterior segment including cornea, iris, ciliary body and ora serrata, and a piece of the posterior segment not including the optic nerve entrance but showing the retina in close proximity to it.

Cornea. The cornea on the whole presents normal characters. The sclerotomy wound is present in the sections and has become partially torn apart in preparing them. (fig 32)

It is situated a considerable distance in front of the filtration angle, and its edges show a shallow cellular infiltration some of the nuclei being tripartite. ☆

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☆ This and other sections which I have seen suggest to me a strong doubt as to whether a sclerotomy can really fulfil its supposed purpose of opening up an adherent filtration angle, especially if a v. Graefe's knife and not a keratome is used. The scleral opacity at the limbus begins in front of the corneoiritic angle, so that it is impossible to see exactly the position of the knife. On studying the accompanying photograph (fig 32) it will be seen at a glance what an excessively delicate operation it would be to open up the filtration angle without going through the adherent iris into the posterior chamber, and how far back the knife would have to be entered to do so. Besides, even if it were done, would the wound not heal up again by fibrous tissue more firmly than before so that the drainage would be no better, unless indeed a cystoid cicatrix with inclusion of the iris were formed which would hardly be a legitimate aim in ophthalmic surgery. So far as my own observation goes sclerotomy is but a temporary measure in dealing with glaucoma, though I admit that I have not seen enough of cases to dogmatize on the subject. On pathological grounds, however, I would question whether its action is not much the same as paracentesis of the anterior chamber only on a more extensive scale.

This infiltration is probably not more in degree than is proper to the normal healing of a wound. The vessels in the corneal limbus, and also the episcleral vessels further back are enlarged. There is however no evidence of any active inflammation.

Iris, Ciliary Body, etc. The root of the iris is adherent to the back of the cornea both in the position of the ligamentum pectinatum iridis and also for a little piece of the membrane of Descemet. The spaces of Fontana are thus obliteroted, and the canal of Schlemm situated at a considerable distance behind the angle and quite shut off from it. (fig 32) There is perhaps some round-cell infiltration around the canal of Schlemm. The adherent portion of the iris is thinner than the free, but the iris itself presents on the whole normal characters. There is no ectropium uveae. A few blood corpuscles are present in the angle of the anterior chamber. (fig 33)

The ciliary body is normal; the pars ciliaris retinae is normal in its anterior part but shows some vacuolation of its inner layer nearer the ora serrata. Some hyaloid membrane is adherent to the pars ciliaris.

Retina. At the ora serrata all the layers are disorganized and indistinguishable. There and elsewhere in the retina the

distortion is much more marked than in the last case. The retina is strewn with haemorrhages of all sizes, the smaller in the inner layers, (fig 34) the larger infiltrating the whole retina. There are also subretinal haemorrhages lying in a homogeneous coagulum which separates the retina from the pigment epithelium and is probably an artefact, though no doubt representing some collection of serum during life. At no place in the sections is there evidence of any considerable haemorrhage having broken through into the vitreous; the limitans interna seems everywhere intact. While the retina is considerably thickened, it is much more uniformly so than in the last case, and shows nowhere an appearance resembling fig 22. Owing to the suppression in large measure of the proper retinal elements the fibres of Müller show out with great clearness, especially in the inner layers of the retina. Between these fibres the reticulated structure of fibrine is seen in places, with blood corpuscles and a few nuclei in its meshes. (fig 36) So far as any of its normal structural elements are concerned the nerve-fibre layer may be said to have almost disappeared: the ganglion layer is also only represented by an isolated cell or two. The two reticular layers are usually distinguishable; the inner is on the whole the best preserved; the outer is in places a good deal mixed up with the nuclei of the nuclear layers.

In places, however, the reticulations are almost obliterated, and there only remain the horizontal lines representing the supporting framework from the fibres of Müller. (figs 35, 36) The two nuclear layers are usually fairly distinguishable, though there is a tendency to scattering of the nuclei. The outer nuclear layer is much better preserved than the inner. (fig 34) In the rather mixed up outer reticular and outer nuclear layers, and lying between the fibres of Müller there are present certain rounded homogeneous looking bodies, staining well with eosin, and bearing a considerable resemblance to the subretinal coagulum already spoken of. (fig 37) Sometimes these bodies lie among reticulated fibrine, and they probably represent serum coagulated by the hardening fluids. The serum would probably come partly from the coagulated blood and partly no doubt was present in oedematous spaces in the retina. It will be remembered that in normal retina the nuclei of the outer nuclear layer are mostly close to the membrana limitans externa, leaving a space between them and the outer reticular layer crossed by the supporting and conducting fibres of the retina. The position of these bodies is about the position of this space. The rods and cones are greatly distorted and no details of their structure are to be made out. Deutschmann's appearance of the cone nuclei passing into the cones, is not to be seen in these sections. The pigment epithelium is normal. The

retinal vessels show very marked evidence of disease in the form of a great thickening of their internal coats. (figs 38,39) This has chiefly involved the veins. Where it is most advanced it has caused almost complete obliteration of the lumen of the vessel, and where it is slighter it has usually involved the lumen eccentrically, encroaching from one or other side. (fig 38, 39) The thickening is cellular, and there are present in it small new formed vessels. Frequently such diseased vessels are surrounded by profuse haemorrhage. (fig 39)

Nowhere in the retina are there any collections of round cells or other evidences of inflammation.

The sclera shows no abnormality; the chorioid is perhaps somewhat engorged though this is a matter not easily judged of. There are no signs of inflammation in either tunic.

## Critique.

Evidently then, although there are points of difference which I shall discuss presently, this case shows a considerable resemblance to the last. We have a glaucoma unamenable to treatment by operation, and the eye on enucleation shows on the whole similar changes to those already described viz. (1) Adhesion of the root of the iris to the back of the cornea. (2) Profuse retinal and vitreous

haemorrhages. (3) Atrophy of the nerve elements of the retina. The latter may be at once set aside as secondary to the retinal haemorrhages and the glaucoma, leaving the mutual relations of the first and second for discussion. Although the radial arteries in this case were not markedly rigid, yet there was evidence of some general vascular disease, if we may believe Marcus Gunn (see foot-note 4. in the ophthalmoscopic appearances of the right eye. At any rate we should not expect to find very healthy vessels in a man of 52, the subject of albuminuria, with high tension pulse and forcibly acting heart, and suffering from dyspnoea. Hence in any hypothesis dealing with the occurrence of the retinal haemorrhages we may take account of the probable existence of general arteriosclerosis.

As before two hypotheses are open for discussion which take account of this. (1) That there had occurred a haemorrhagic retinitis secondarily to which a glaucoma developed. (2) That a primary glaucoma had occurred in the course of which, probably at the time of operation profuse retinal haemorrhage had taken place. In case 7 as we have seen the second of these hypotheses seemed to be the true one; in the present instance I think the evidence is in favour of the former, and therefore the case would be similar to those described in the literature. The point cannot be settled by the ophthalmoscotic examination since glaucoma was already present at his

first visit, and the media too opaque to allow of examination. The history of onset, however, does not suggest primary glaucoma but is typical of haemorrhagic retinitis viz. on awakening one morning there was a general mistiness before the sight of the eye (C.f. the clinical histories of the cases already reported), and for five days thereafter there was no pain. Then follows the periorbital pain, which practically continued up to the time of enucleation. This evidently sounds like the history of a haemorrhagic retinitis followed by glaucoma. In the last case the occurrence of haemorrhage after the operation was actually observed, and the pathological appearances were consistent with the origin of all the haemorrhages at that time. In the present instance there was no such observation of the occurrence of intraocular haemorrhages, and the pathological appearances were against the idea of its having occurred at the time of operation. Thus the haemorrhages were confined to the retina and adjacent vitreous; none were present in the iris or ciliary body where we might have expected them had they been due to operative interference, and where they were abundantly present in the last case. The most essential difference between the two cases is perhaps that in the former there was much evidence of inflammatory processes in the distortion of the iris, whereas in the present instance, apart from its adhesion to the back of the cornea, the iris was normal. I am not sure what the

significance of this difference may be. Perhaps, as I have before stated, in case 7, the glaucoma was dependent upon a very chronic process of iritis, but at anyrate this was not the case in the present instance, and we must probably therefore look to the haemorrhagic retinitis as the primary factor in the case. For the local cause of this haemorrhagic retinitis we must look to the retinal vessels. The central vein was unfortunately not available for examination, but the retinal veins showed changes of very high degree, much higher than in the case 7. The changes were I think, much too great to be consistent with the supposition that they were secondary to a state of high tension, more especially as they were not in the form of endarteritis but of cellular and organized tissue, in all probability of organized thrombus.

In attempting to gain light on the significance of the pathological changes in these two instances a comparison of the duration of the different clinical stages is of importance. This may be set down as follows:-

| | Case 7. | Case 8. |
|---|---|---|
| From the date of the first symptoms to the operative interference. | about 60 days. | about 18 days. |
| From the date of operative interference to the date of enucleation. | 14 days. | 35 days. |
| Totals | 74 days | 53 days |

Hence the glaucoma had lasted considerably longer in the first case than in the second, and therefore, had the vessel changes been dependent on this alone they should have been less in case 8 instead of much greater. The more marked retinal changes in the present case, since they cannot be due to a longer continuance of the glaucoma, must be due either to the more prolonged presence of blood in the tissues, or to the greater interference with the blood supply, or to both. From the higher degree of the changes alone however, it cannot be argued that the haemorrhages took place in case 7 at the time of operation and in the present instance before that time, since even if in both instance the haemorrhages had taken place at the time of operation, it would have been present in the latter case 35 days as against 14 days in the former, and would therefore have produced greater changes. Nevertheless as before said the bulk of the evidence, and especially the clinical history of the mode of onset, favours the view that the haemorrhagic retinitis did indeed occur before the glaucoma.

Let us now try to trace the sequence of events in this eye according to the theory above stated. We should say that there was probably in the first place a diseased condition of the vessels of both eyes as a part of a general arteriosclerosis. To this was added in the left some local lesion probably, in view of the condition of the vessels in the sections,

a thrombosis of the retinal veins with perhaps also a thrombosis in the central vein. This determined profuse retinal haemorrhage and brought on the first dimness of vision. Next the blood broke through into the vitreous, and partly by its mere bulk, partly by driving forward the lens and with it the iris, and partly by an alteration in the intraocular fluids which made them more colloidal and less easily filtered off, (according to the theory of Stölting) set up a glaucoma. The iris probably took only a passive part in this at first, since it shows no signs of inflammation in its general substance, but after some time, the protective endothelium on its anterior surface and on the ligamentum pectinatum and membrane of Descemet having been no doubt destroyed by the pressure, an adhesion formed between the iris and the back of the cornea which would perpetuate the glaucoma. As a result of the primary interference with its blood supply, of the destruction caused by the pouring out of blood in its substance, and of the continued glaucomatous tension, the intense changes in the retina described above were brought about.

But shortly then the essential differences between this case and the last might be stated as follows. (1) In the first case the onset was with periorbital pain and redness of the eye; in the second with sudden mistiness but without pain

for five days. (2) In the first case profuse haemorrhage was observed clinically after operation; in the second there was no such occurrence. (3) Pathologically, in the first case there was profuse haemorrhage in the iris; in the second there was none. (4) In the first case there were marked signs of chronic inflammation of the iris; in the second there were no such signs. (5) In the first case the vascular disease in the retina was slight and in the form of endarteritis; in the second it was much greater, and there was evidence of organization. Other differences are probably of minor importance or secondary to these. The chief points of resemblance may be summarized as follows. (1) In both cases there was evidence of general vascular disease in the patients. (2) Both showed a severe form of glaucoma with much periorbital pain and opacity of the media. This glaucoma was uninfluenced permanently by operation, and on this account came to enucleation. (3) Both were found pathologically to be of the class of "haemorrhagic" glaucomas.

## Conclusion.

We have seen then that the clinical picture of severe unilateral haemorrhagic retinitis has been several times proved pathologically to depend upon a thrombosis of the central vein of the retina. That in other cases, although this was not excluded since the whole length of the vein was not available for examination, yet the only lesion found was certain changes in the retinal vessels apparently in the form either of organized emboli or thrombi. In the two cases of Deutschmann alone the lesion was a haemorrhage within the optic nerve which probably acted in the same way as a thrombus by obstructing the vein. It has further been shown that there may follow upon such a haemorrhagic retinitis a condition of glaucoma, sometimes preceded by an iritis but apparently more often not.

Six clinical examples of such a haemorrhagic retinitis have been presented, of which four (Cases 1, 2, 5, 6) were in middle or advanced life (40 to 62), and two were young. Of the four cases two showed some degree of arteriosclerosis. (cases 1 & 5) and in the other two the signs were more indefinite. In cases 1 and 5 there was a trace of albumen in the urine. Of the two younger patients one (case 3) gave no clear clue to the etiology, the other (case 4) showed evidence of mitral stenosis with

cardiac embarassment; in neither of these two was the urine examined. Cases 1, 2 and 3 each gave a history of an attack of influenza; in case 1 the ocular lesion occurred during the attack; in case 2 a fortnight later; and in case 3 a month later. Except for case 4 none of the patients showed signs of valvular cardiac disease or other definite illness. As regards the subsequent course of the disease cases 2 and 4 were not traced. Case 1 showed no improvement after a month; Case 3 showed a rise of visual acuity from less than 20/200 to 20/20, and the haemorrhages disappeared entirely in ten months. Case 5 showed a rise of the visual acuity in two months from 20/200 to 20/70 and the haemorrhages had almost disappeared, but there was some degree of optic atrophy left. Case 6 showed two months later no great change in the ophthalmoscopic picture, and vision had sunk a little - from 10/200 to 6/200. Thus of the two cases which showed improvement one was not a severe case (Case 5), and one occurred in a young person (Case 3), so that it would seem that in elderly people and with severe retinal haemorrhage the prognosis is bad. This is the same as Michel's finding except that Michel does not mention a good prognosis in the young.

Of the treatment of the affection I have little to say, and indeed it is not one which is likely to be very directly affected by therapeutics. Some of the

patients were given potassium iodide in the hope of promoting absorption of the thrombus; but judging from the analogy of the effect of the drug in aneurysm it might be questioned whether it would not tend to increase the coagulability of the blood. It is noteworthy that the patient who showed the greatest improvement (Case 3) found herself unable to take potassium iodide and stopped it after a day or two. A tonic line of treatment might be undertaken if the general health were unsatisfactory, which however seems to be by no means always the case. The condition seems to be an important evidence of vascular disease, and might guide us in the prophylactic treatment of cerebral haemorrhage. The danger to the other eye does not seem to be great.

It has not been my lot to observe any case pass from the condition of haemorrhagic retinitis to that of glaucoma. Yet it seems to me, from the considerations mentioned in the text that case 8 was an example of this class in which glaucoma had already occurred before he came under observation. Case 7 I have placed in another category, believing it to be a primary glaucoma with intraocular haemorrhage following upon operation. If this be so, then it is proved that the condition known as "haemorrhagic glaucoma" is not a pathological entity but many arise in more ways than one, although probably arteriosclerosis underlies all the forms. Both of the cases reported

here showed both albuminuria and arteriosclerosis. Unfortunately in neither case could the central vessels be submitted to a full pathological examination, so that the question whether or not there was a thrombus in the central vein cannot be settled from actual observation. We can only say, judging chiefly from the clinical history and the pathological state of the retinal vessels, that in Case 7 there probably was not, while in Case 8 there may have been such a thrombus.

## Appendix.

The following is also a case of unilateral retinal haemorrhage but evidently belonging to a different category from any of the cases previously described.

Enormous subvitreous, with some smaller retinal Haemorrhages in the left Eye. Evidence of cardiac Disease and of Nephritis.

Marion Mc C. - aet. 50.

Presented herself at the dispensary department of the Glasgow Eye Infirmary on May 17th, 1900, complaining of dimness of vision of the left eye of about a month's duration.

Her hereditary history showed nothing remarkable. Her previous health had been

good, though she had suffered at times from indefinite pains in the back, and the right foot sometimes swelled after long walking. She had never had headache or sickness. Eight or nine months before she was first seen she had received a blow from the cork of a lemonade bottle on the left eye or eyebrow. There was some pain in the brow for a day or two afterwards, but not much, and neither at the time nor immediately afterwards was there any defect in the sight.

About 7 or 8 months later she was out of sorts and feeling rather weak though not confined to bed, and she believed that she had "influenza." At this time a mistiness came over the sight of the left eye, so that everything appeared blurred; 3 weeks later a sudden further increase in the amblyopia occurred, and in especial she found that she could see nothing straight in front of her with the left eye, but had to turn her head to the right so as to bring the rays on to the nasal half of the retina, when she could dimly see objects. This condition had remained unaltered since then.

Present Condition. - The patient's general health seemed fairly good, but she was a little breathless and she stated that she had sometimes slight precordial pain on exertion. Examination of the heart showed to percussion some evidence of enlargement, and the apex beat was forcible and displaced outwards beyond the nipple line.

Auscultation revealed in the mitral area a soft blowing systolic murmur. The lungs were normal to physical examination, and the urine contained a distinct amount of albumen, but no sugar. Microscopically there were scanty hyaline and granular casts.

Both eyes were normal to external appearance, the pupils equal and responding to light and convergence, and the tension not raised. She was unable to read any of Snellen's types with the left eye when she looked straight at them, but on turning the head to the right she distinguished letters of 20/100. The visual acuity of the right was 20/50. The right fundus showed no abnormality. O.E.L. The disc was practically normal, perhaps a little congested. The retinal vessels were normal in course and calibre. About one disc breadth to the temporal side of the disc there was an enormous haemorrhage which completely covered the macular region. This haemorrhage was of the submitreous type. All its edges were sharply defined and rounded, especially the edge towards the disc. The outer edge was not visible, though the dark colour of the haemorrhage seemed to be lightening off in this direction. Along the course of the upper temporal vessels there were two or three small flame-shaped haemorrhages, one of which, not far above the large haemorrhage lay on the top of the vein and obscured a portion of it.

There was, however, no connection between these flame-shaped haemorrhages and the large one. (fig 40)

Corresponding to this haemorrhage there was a large scotoma in the field of vision. There was an area of absolute scotoma engulfing the fixing point and surrounded by an area of relative scotoma (see perimeter chart) stretching out to the periphery of the field. (fig 41) The further history of the case I do not know.

In accounting for this large haemorrhage the most natural supposition is that it was bored out from the temporal vein above it at the place where the small haemorrhage lay on the top of it, and that the connection had afterwards been broken. It is not clear why so large a haemorrhage had not gravitated further down, unless indeed it were kept in position by some slight staphylomatous bulging of the macular region (the disc itself was emmetropic.) The lesions of the vessels were probably dependent on nephritis, since there were albumen and casts in the urine. The blow from the cork seven or eight months previously, and not followed at the time by any dimness of vision, may almost I think be excluded from the etiology.

A case almost exactly the same as this is given by Liebreich. (19) It occurred in a woman of 45, and Liebreich associates it with the climacteric and says that he has seen other similar cases at the same

time of life. He does not mention the state of the urine. There was in this case also a large scotoma, and vision was better eccentrically than centrally. Several small fan-shaped haemorrhages were present. Vision rose to normal in five months, the small haemorrhages soon disappearing and the large very gradually. In my own case the woman being aged 50 should have been beyond the climacteric, but I did not make any inquiries on the subject. Another curious case reported by Latham Thompson (20) occurred in a widow aged 51. It is stated that there was no history of cardiac or renal disease, but it is not clear if the heart or urine were examined. The haemorrhage had two prongs stretching downwards and outwards temporally, so that the scotoma had to the woman the appearance of a "goat's head with the horns curving backwards", an appearance well shown in a perimeter tracing which is given.

Finch after historical resumé in which he states that Michel having had opportunity to examine Angelucci's preparations denied that there was any thrombus, only a coagulation lying outside the vein, gives it as his opinion that where glaucoma follows upon hyperextinction there is no proof of cause and effect but only of dependence on a common cause i.e. vascular disease.

Woman set 67 had sudden unilateral eight in left eye 4 weeks before he was first seen. Irregular heart action and slight emphysema no glycosuria or albuminuria. Right normal. Left VA = movement of hand. Pupil immobile. Disc red, hazy edged; innumerable irregular not very large hges in fundus; in central part many white flecks. Veins not enlarged + tortuous just at papilla but somewhat so more peripherally. Arteries invisible. Tn - Died of pneumonia 9 days later. PM also endocarditis.

Thrombus in central vein from lamina Cribrosa

about .5 mm backwards. Anteriorly organized & fully stopping vein. Posteriorly not so fully & still cleft between it & vein wall. No pathological changes in either vein or artery wall. Retina destroyed & distorted by haemorrhages and oedema with homogeneous coagula & prominence of fibres of Müller. No thickening of intima of retinal vessels.

## Wündemann.

Boy 8 yrs old became suddenly blind in the left eye 4 days before. No general disease present. Right eye normal

VAL = 0. pupil immobile, reacts consensually. Enlarged, tortuous veins very many haemorrhages. Oedematous appearances in macula with also haemorrhages there. Arteries very small. About 6 weeks later white flecks in place of hge. Papilla white, slightly excavated. Vessels hidden in place

& remains of former hges. A year later was having acute glaucomatous attacks in eye. (had begun some months before). White fibrous bands in vitreous no details of fundus Enucleation. Iris angle blocked. Connective tissue in vitreous. Blood in retina. Atrophy of nerve. Artery totally blocked & clot containing degenerated red & white corpuscles for about 5 mm beginning 1 mm behind lamina. Adventitia thick but not intima. Similar changes in retinal vessels. Small "clot" in vein.

## Michel

(Zeitschrift für Augenheilkunde 1899 Band II 5 i) says that retinitis haemorrhagica may be caused & ① a marasmic thrombosis of the central vein ② & a Phlebitis Proliferans shutting of its lumen ③ & wide spread disease of the retinal veins (Phlebitis proliferans with narrowing & occlusion of the lumen either by proliferation or thrombosis.

In ① he says the vein will be enlarged & tortuous in ② & ③ more or less diminished in size & little filled. It is here that M. says that having examined one of Angelucci's preparations he believes the so-called thrombus to have been a concretion (like a Prammonia) lying outside the vessel and possibly compressing the artery.

Schmidt Arch f. Augenheilk. XXIV 1892

Das glaucomatöse Sehnervenbecken. p 273

"In an eye enucleated 3 weeks after the outbreak of acute secondary Glaucoma I found a marked cellular infiltration around the central vein and a thrombosis of it, which I specially mention because the finding appears to me of importance for the understanding of haemorrhagic glaucoma.

(see p. 283)

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(33) Michel. "In 1898 reviews pathological findings in retinitis haemorrhagica and gives two cases due to phlebitis proliferons of the central vein) Zeitschrift f. Augenheilk, Vol 2, 1898.

(34) Reimar.

Versammlung der Ophth.
Gesellsch. zu Heidelberg.
1898, p. 331.

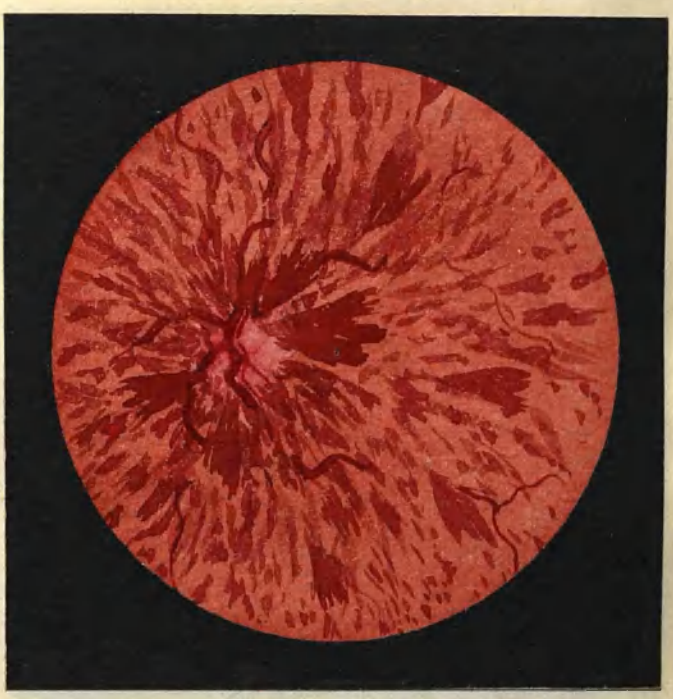

Fig. 1. Ophthalmoscopic appearances in Case 1.

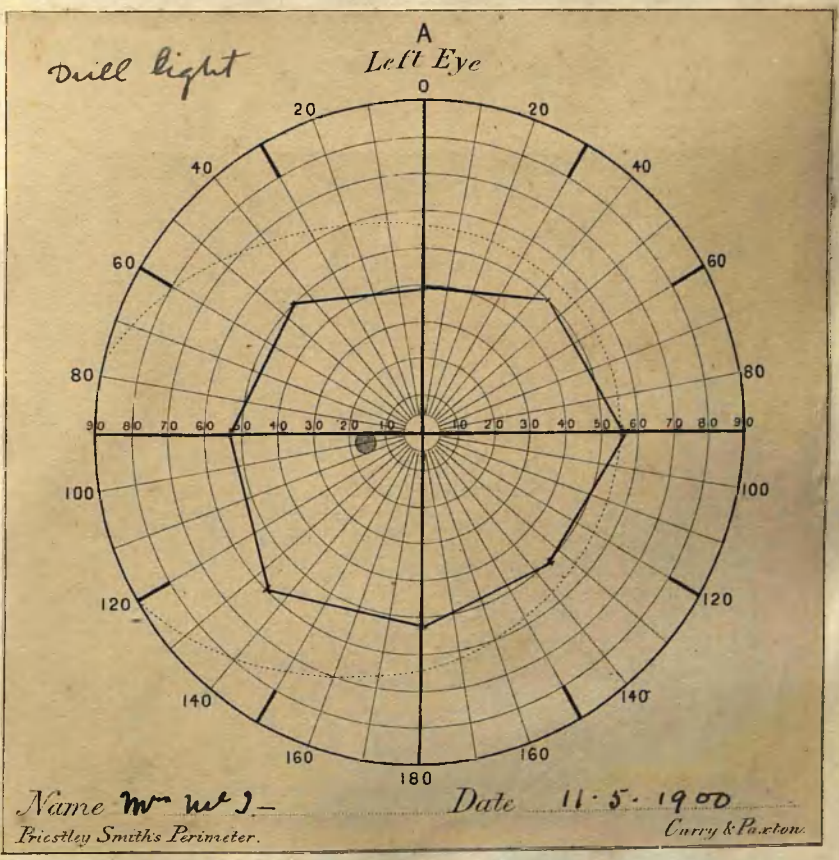


Fig. 2. Field of Vision of Case 2.

Fig. 3. Ophthalmoscopic appearances in Case 2.

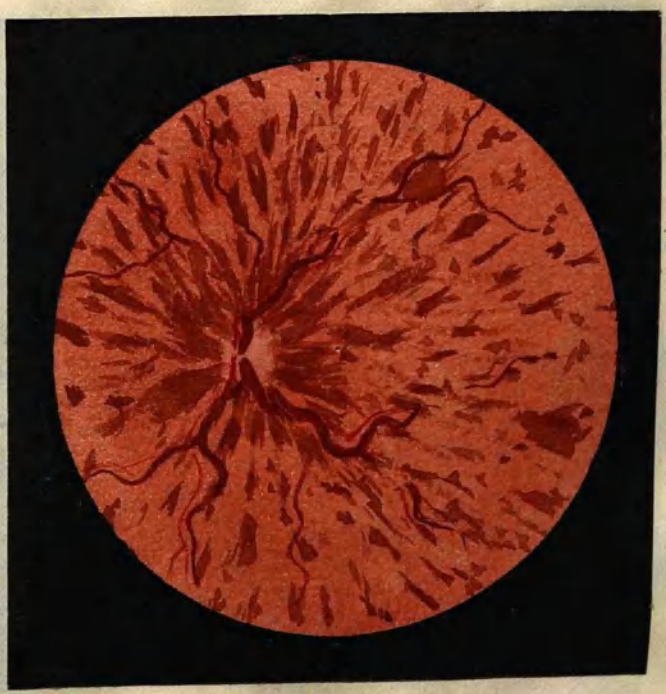

Fig. 4. Ophthalmoscopic appearances in Case 3.

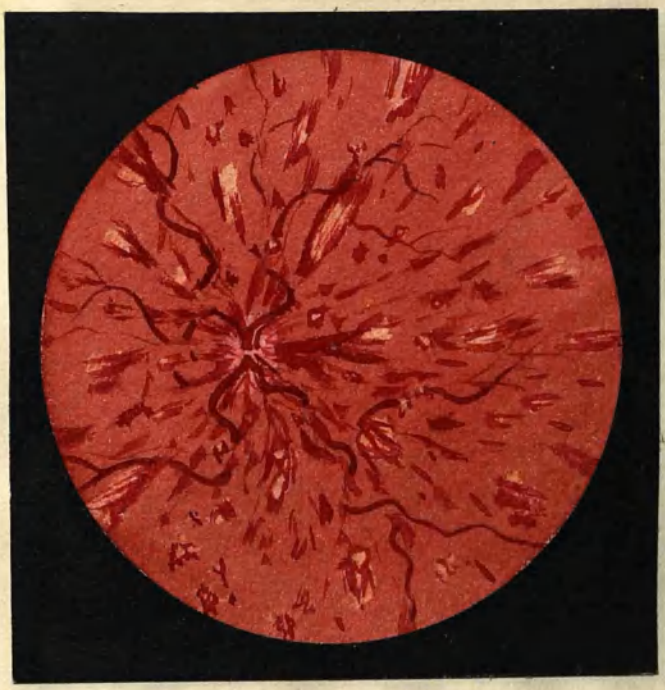

Fig. 5. Ophthalmoscopic appearances in Case 4.

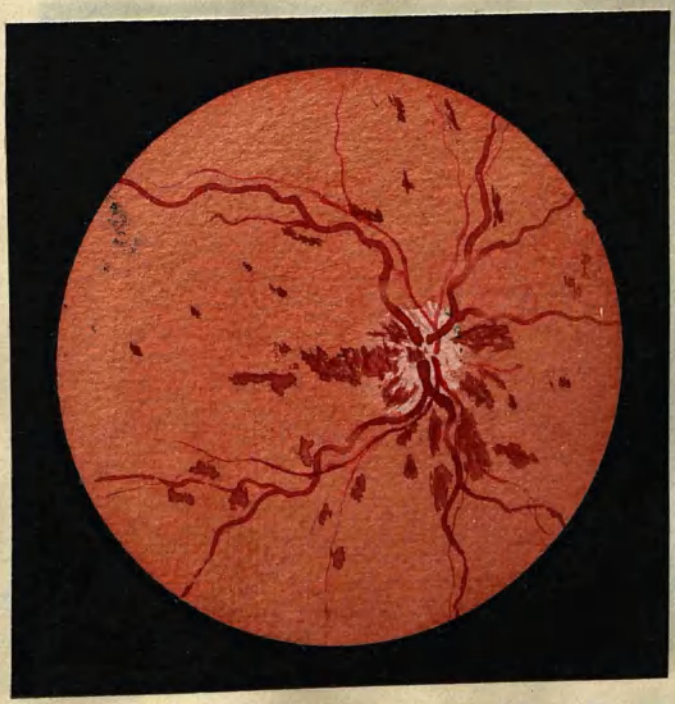

Fig. 6. Ophthalmoscopic appearances in Case 5.

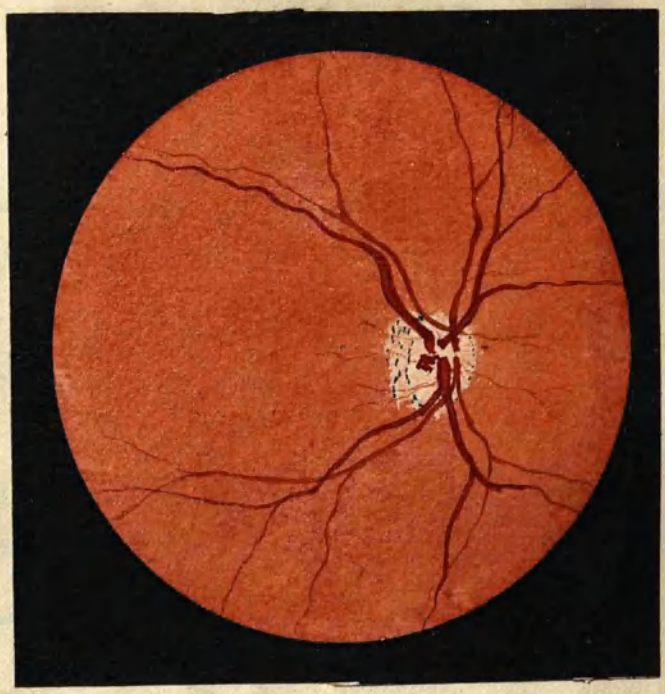

Fig. 7. The same 48 days later.

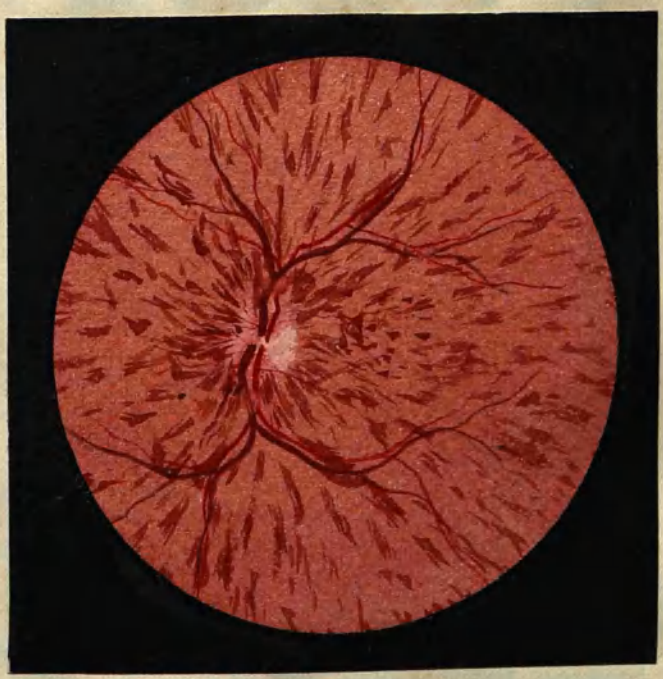

Fig. 8. Ophthalmoscopic appearances in Case 6.

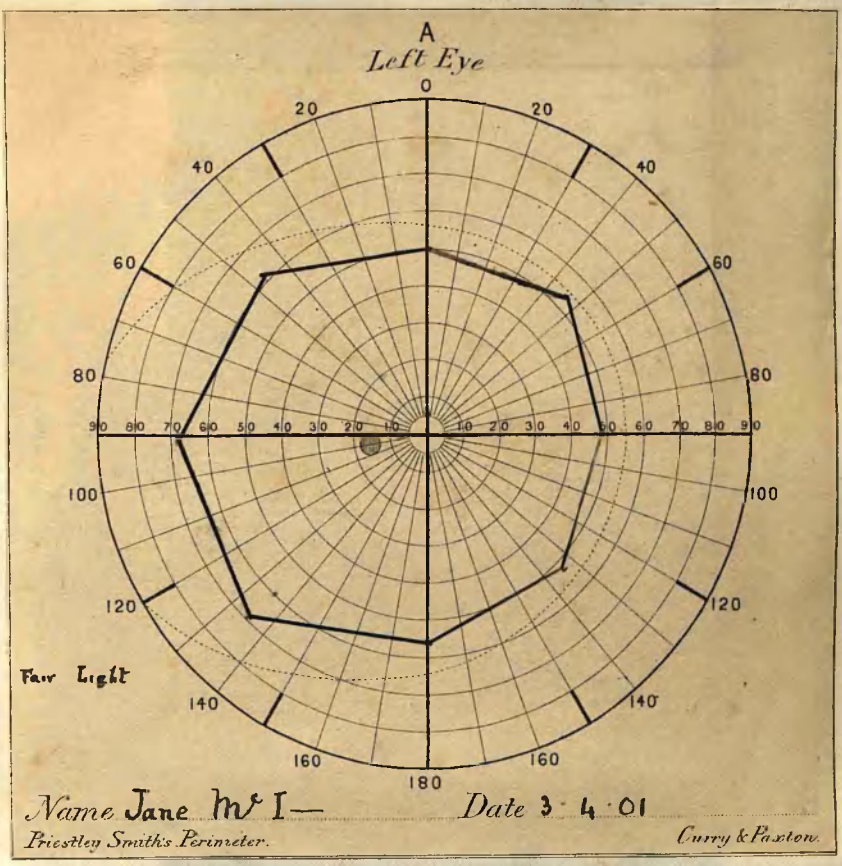


Fig. 9. Field of Vision in Case 7.

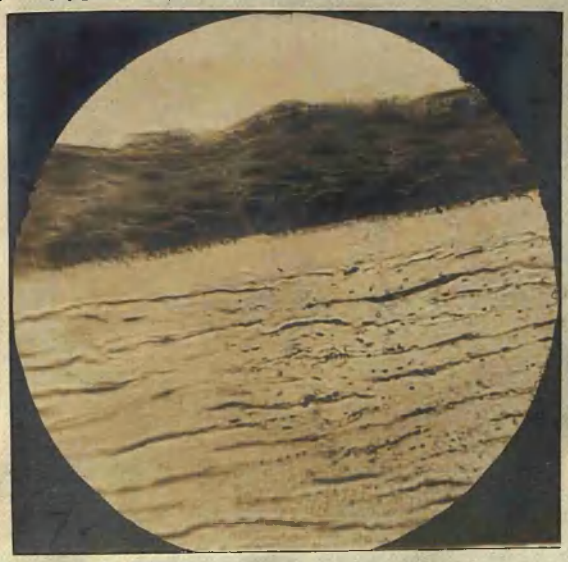

Fig. 10. Shows the flattened appearance of the corneal epithelium and the granular appearance under it and in the superficial corneal layers. The membrane of Bowman shows no such appearance.

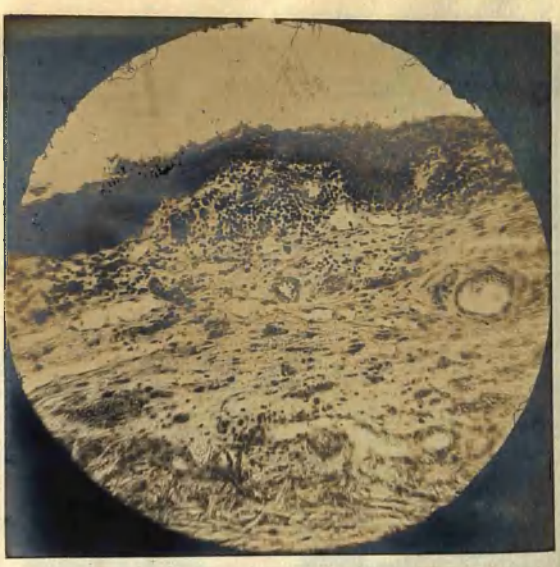

Fig. 11. A small round cell infiltration near the corneal limbus.

Fig. 12. The filtration angle shut off by fibrous tissue so that the Canal of Schlemm is some distance behind it. A little blood in the angle of the anterior chamber.

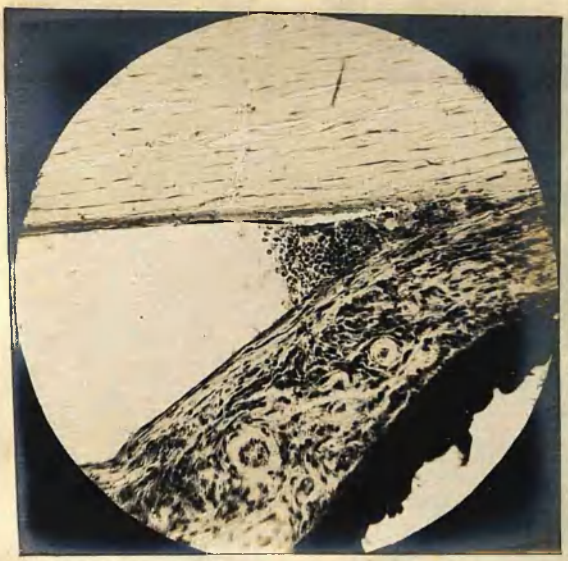

Fig. 13. Blood-corpuscles in the anterior chamber. The dense structure of the iris and the thickening of the vessel walls in it.

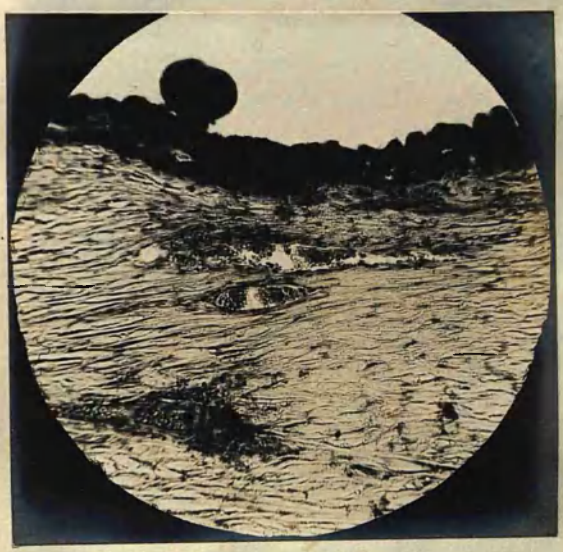

Fig. 14. The Canal of Schlemm with a large vein in close connection with it.

Fig. 15. The Canal of Schlemm with a large vein in close connection with it.

Fig. 16. Low power view of the iris showing the large haemorrhage on the anterior surface and the ectropium uveae (Haematoxylin and eosin).

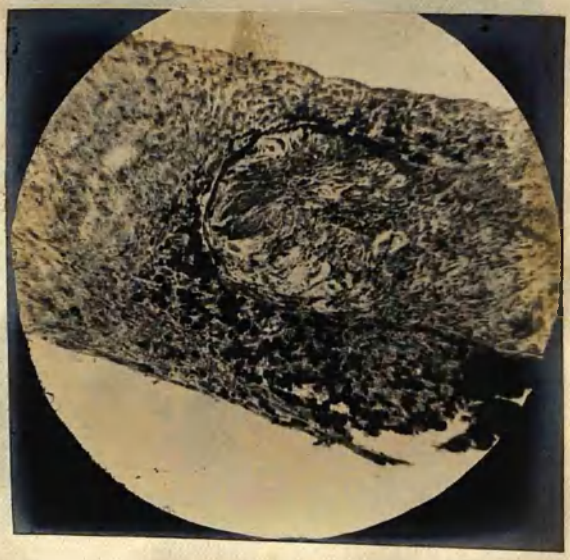

Fig. 17. Another part of the iris showing the distorted sphincter, the ectropium uveae, the ball-like pigment masses and the badly staining mass of blood within its pupillary margin. The anterior surface of the iris is to the upper side.

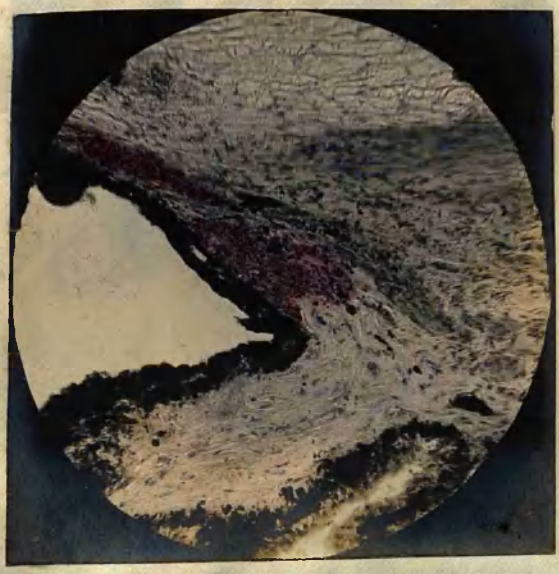

Fig. 18. A small extravasation in the anterior part of the ciliary body (Haematoxylin and eosin).

Fig. 19. The ora serrata showing the great disorganization and the adherent hyaloid membrane.

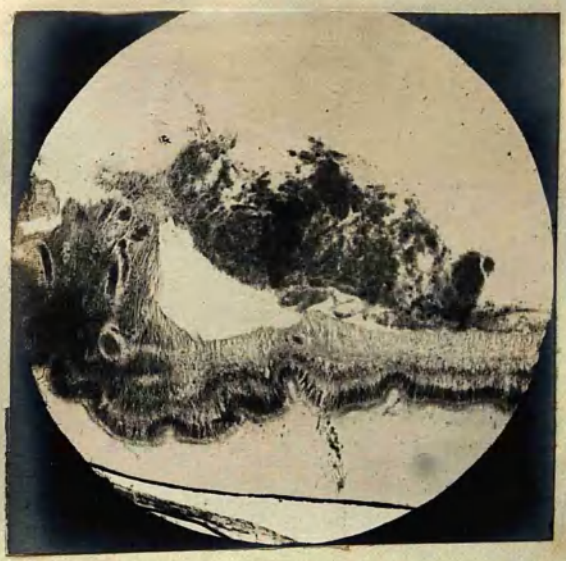

Fig. 20. A large haemorrhage in front of the retina which probably arose from the engorged vessels in the thickened part to the left.

Fig. 21. A subretinal haemorrhage lying in a homogeneous coagulum. The retina somewhat thickened and much folded; its layers not very distinctly separable.

Fig. 22. An enormous thickening of the retina much infiltrated with blood.

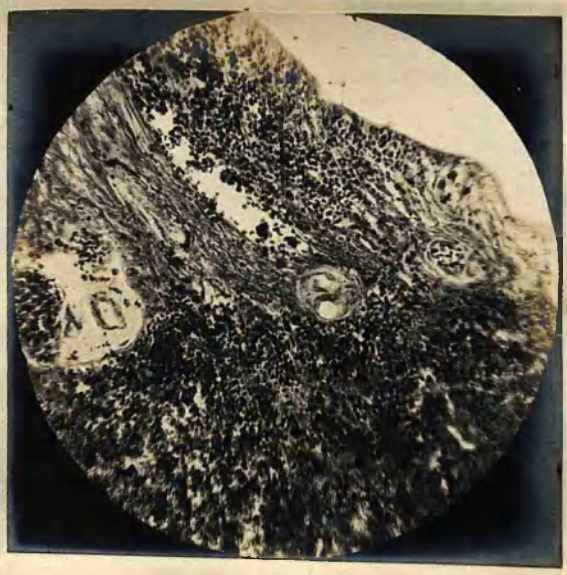

Fig. 23. Structure of the same. Tissue resembling the supporting structure of the retina, crammed with blood.

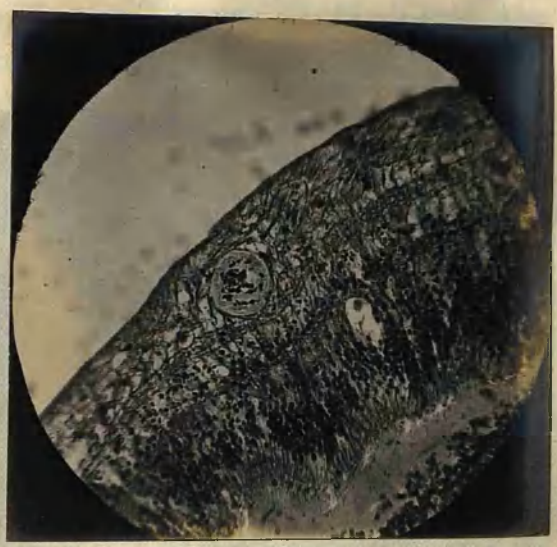

Fig. 24. View of the retina. Great atrophy and vacuolation of the inner layers, the outer nuclear layer being the best preserved though a large vacuole is seen between it and the outer reticular layer. A vessel almost obliterated by hyaline thickening of its walls is seen.

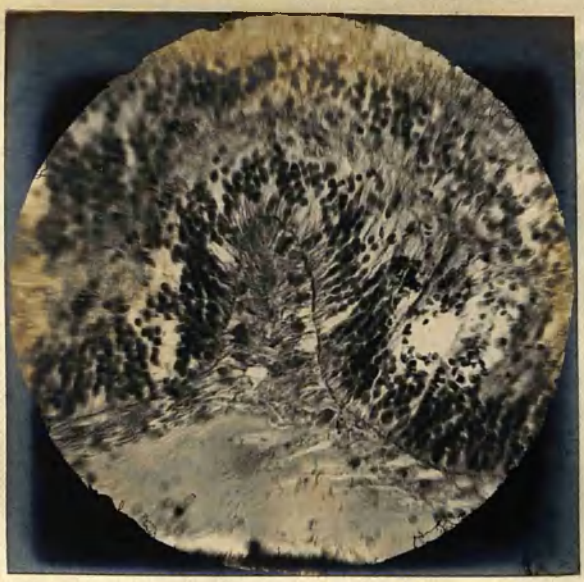

Fig. 25. Deutschmann's appearance of the cone nuclei passing through the limitans externa into the cone bodies. Note the bad preservation of the rods and cones, the subretinal coagulum and to the right the space caused by a small haemorrhage.

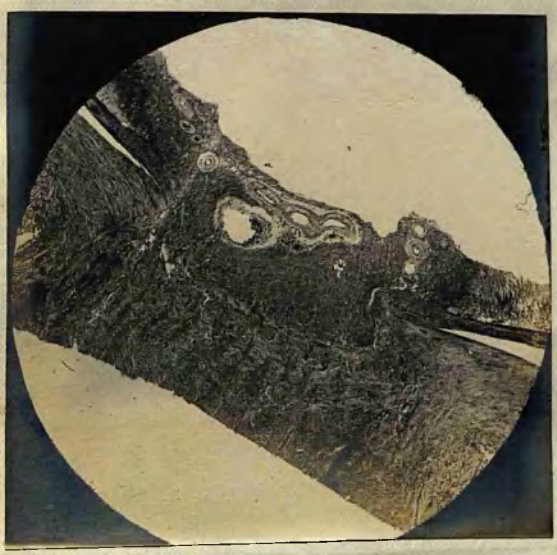

Fig. 26. The papilla cut not quite through the centre showing fairly normal vessels but much atrophy. The lamina cribrosa somewhat excavated.

Fig. 27. A very thick walled vessel on the papilla.

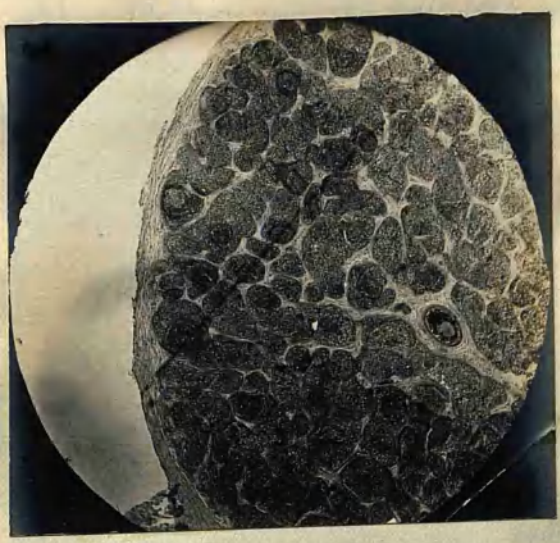

Fig. 28. Normal optic nerve to compare with Fig. 29.

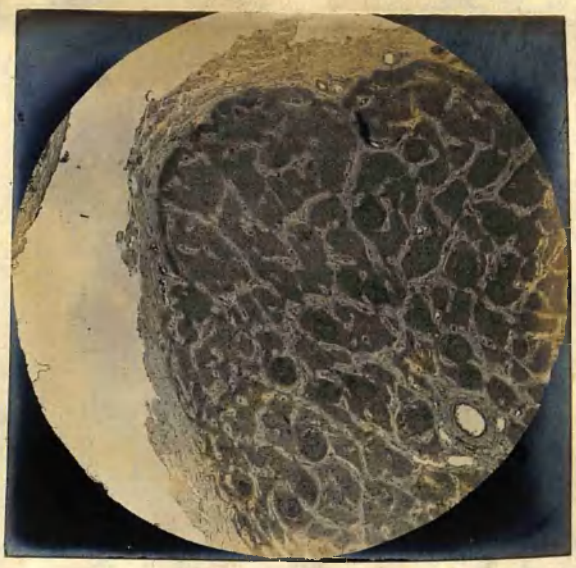

Fig. 29. Optic nerve to show the ill-defined and badly stained appearance of the nerve bundles, the otrophic condition of the nerve and the increase in the connective tissue septa. This specimen and the last are stained in the same way and photographed under the same power.

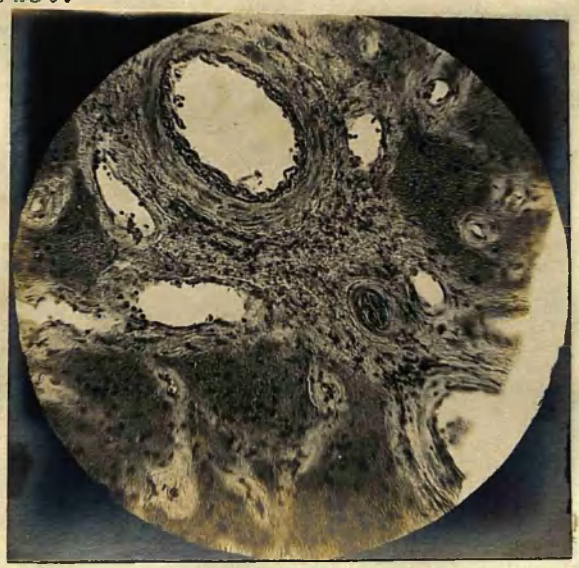

Fig. 30. The central vessels; the artery fairly normal the vein somewhat thick-walled but showing no endophlebitis; a small vessel between the two blocked. Cellular infiltration of the supporting connective tissue.

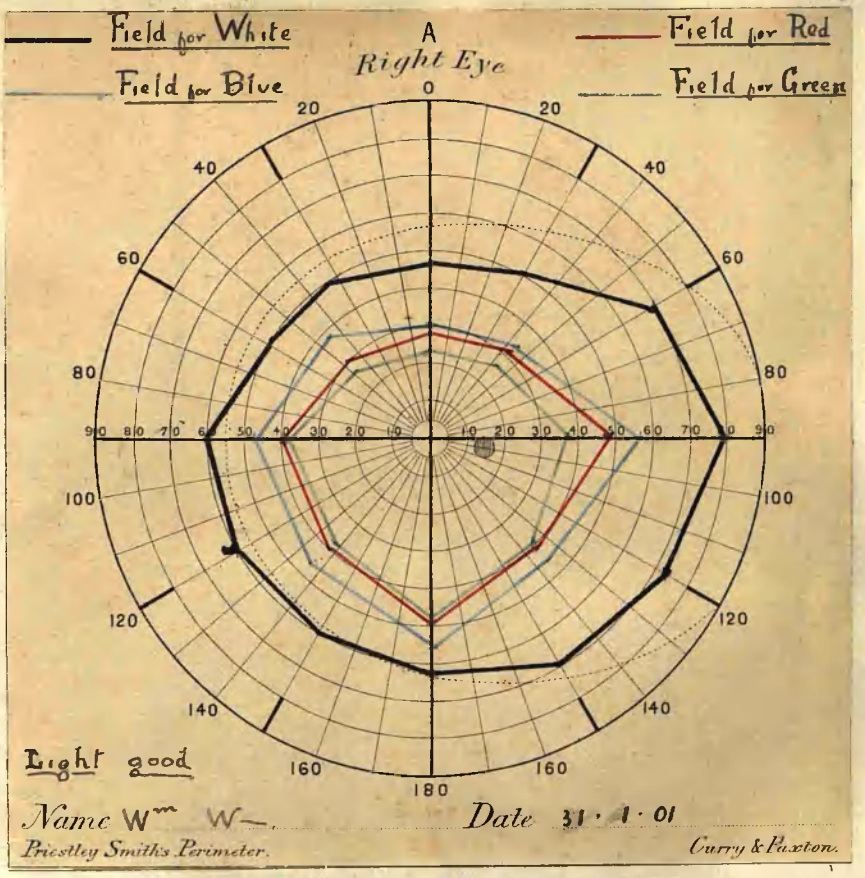


Fig. 31. Field of Vision of William W. –

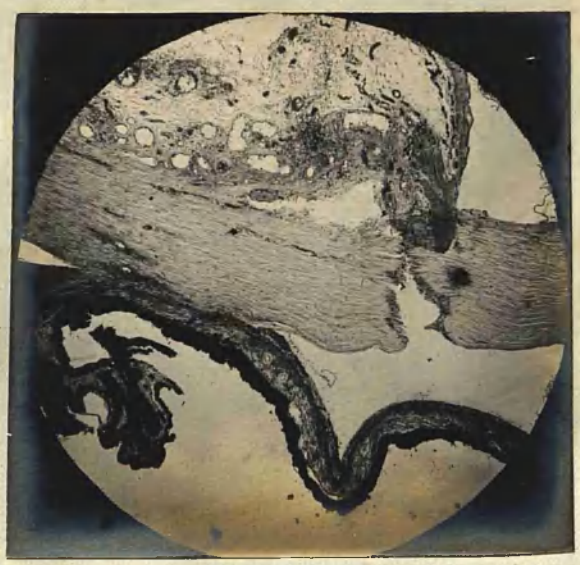

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Fig. 32. Corneo-iritic angle showing shutting off of the spaces of Fontana and Canal of Schlemen by fibrous tissue. The sclerotomy wound much in front of the angle. Engorgement of the episcleral vessels. The iris structure approximately normal.

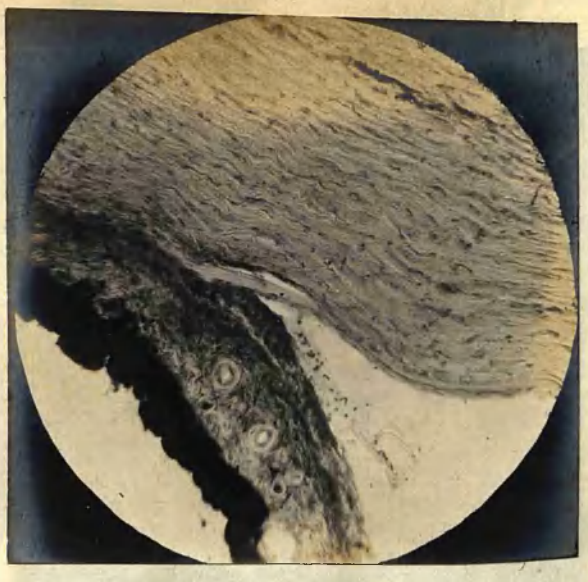

Fig. 33. A few blood corpuscles in the angle of the anterior chamber.

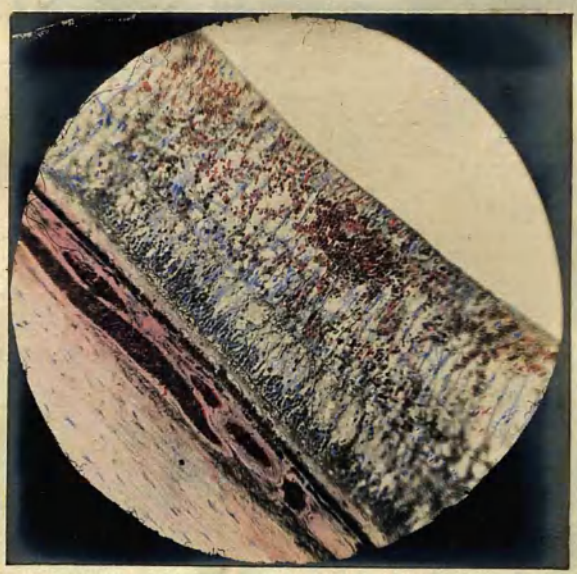

Fig. 34. General view of the retina showing a small haemorrhage and scattered blood corpuscles. Great atrophy of all the structures the outer nuclear layer being the best preserved. A thin layer of subretinal coagulum. The chorioidal vessels very large. (Haematoxylin and eosin)

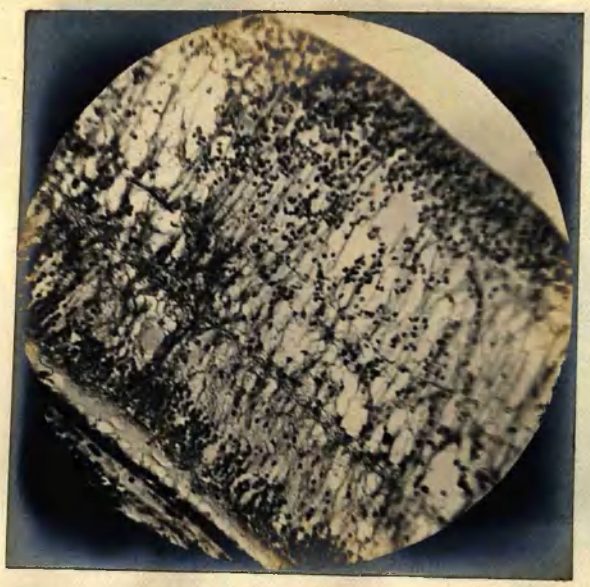

Fig. 35. A similar view more highly magnified.

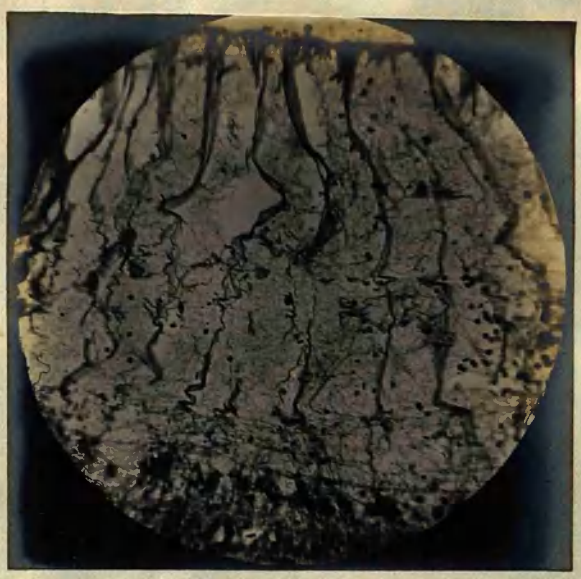

Fig. 36. Extreme atrophy of the inner layers of the retina in which the fibres of Müller show out very clearly. Fibrin is present between them.

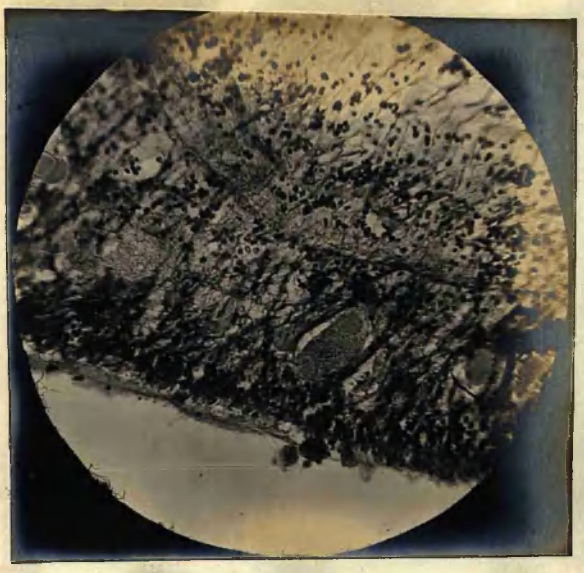

Fig. 37. The outer retinal layers showing some homogeneous masses. (Cf Haab's Atlas 1900 edition p 27, fig 27a.) Much distortion and over all a scattering of blood corpuscles.

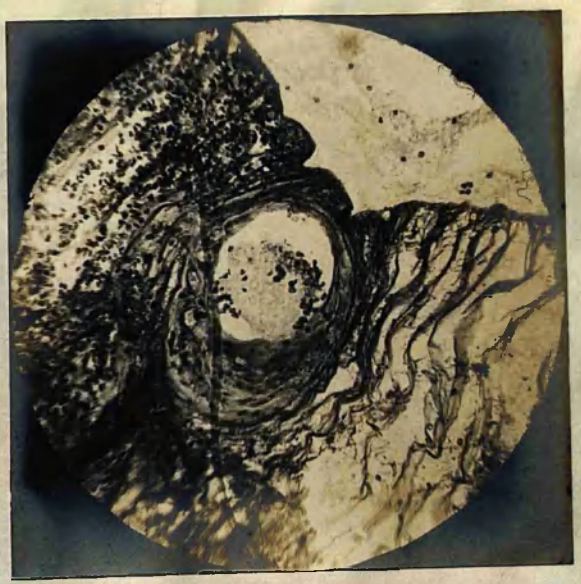

Fig. 38. A moderate degree of change in the wall of a retinal vein. The lumen is encroached on eccentrically. There is much blood in the surrounding retina.

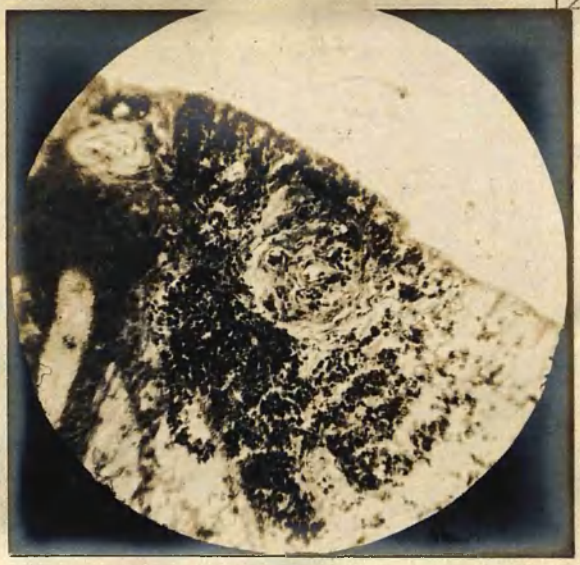

Fig. 39. A very high degree of change in a retinal vein amounting probably to obliteration. There are nuclei in the tissue and small new-formed vessels. The corresponding artery is seen out of focus to the left. A dense mass of blood corpuscles surrounds the obliterated vessel.

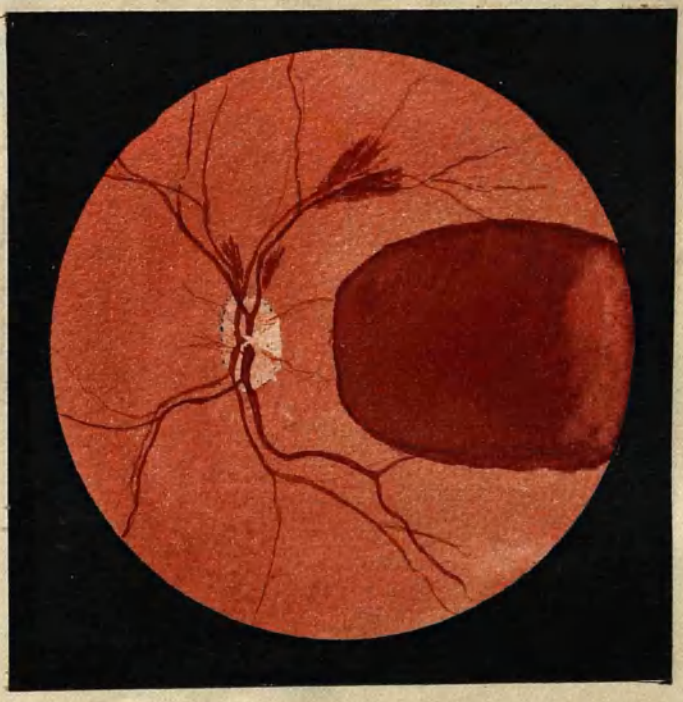

Fig. 40. Ophthalmoscopic appearance in the case of Marion Mc C.

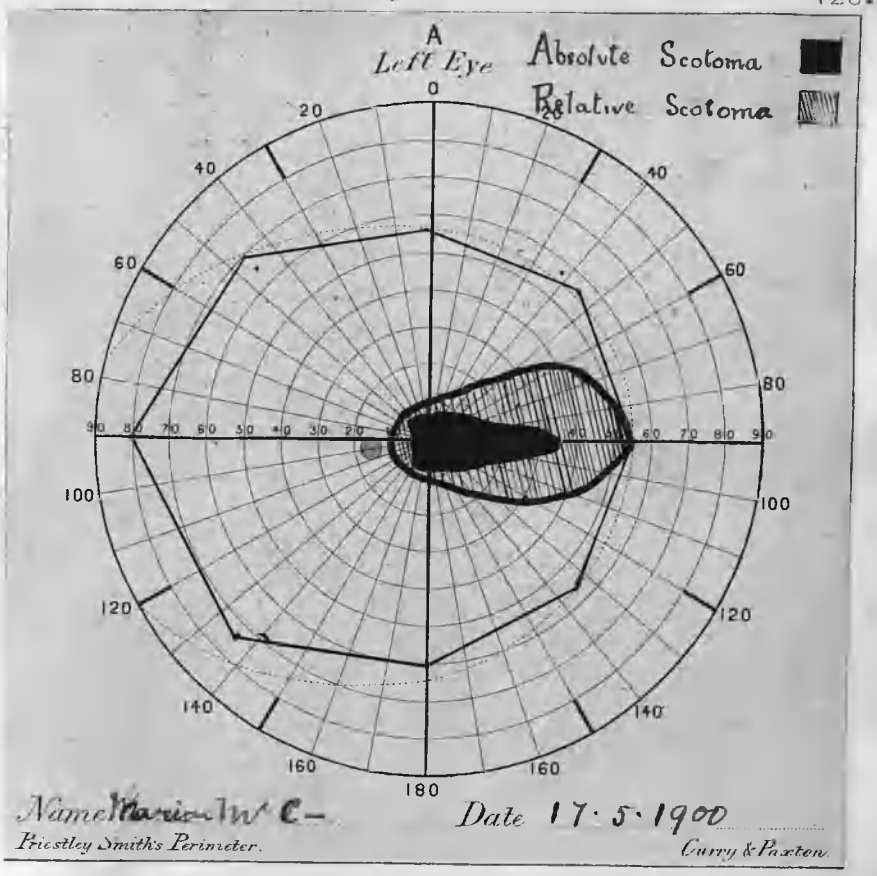


Fig. 41. Field of Vision in the case of Marion Mc C.