T H E S I S

Presented for the Degree of M.D., (Univ. Glasgow).

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On

Primary Glaucoma

with an Analysis of 34 Cases.

P R E F A C E.

In presenting the following essay as a Thesis for the Degree of Doctor of Medicine, I have been actuated mainly by the impressions of the disease which I received while acting as House Surgeon in the Glasgow Eye Infirmary. Very soon I came to look upon Glaucoma as one of the most formidable of diseases of the eye. My rather pessimistic mood may be accounted for by the fact that very few of the cases which I saw were of the true acute primary type, the type most amenable to treatment. They were cases mostly of subacute and chronic congestive glaucoma and glaucoma simplex.

I was taught that the increased intra-ocular tension, the main factor in glaucoma, was due to the obstruction of the filtration angle and the treatment which I saw directed to the cure or relief of the disease was almost invariably directed to removing so far as possible that obstruction by iridectomy. The apparent lack of uniform results by this method of treatment led me to make the following investigation of the opinions

held as to the etiology, the main symptoms and course of the disease and its treatment.

I have made an analysis of my own cases and have stated where, from consideration of these cases, my opinions differ from those generally accepted. I regret that the number of cases I have had the opportunity of studying is too small to make my deductions from them of much scientific value as compared with the large numbers investigated by some of the authorities I quote.

I am compelled to acknowledge that I am still in doubt as to the exact etiology of the disease, and that I cannot suggest any alternative treatment for the cases in which iridectomy and the other methods already in use have proved unsatisfactory.

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PRIMARY GLAUCOMA.

Brief Historical Sketch of the Opinions held regarding the Nature and Symptoms of Glaucoma.

The earliest article on glaucoma to which I have been able to refer is that by Mackenzie[1] in the 1834 edition of his renowned work on ophthalmology. In it he traces the history of the term glaucoma to the days of Hippocrates who used the word "γλαυκώσεις " to include all the opacities seen behind the pupil irrespective of their exact nature. The later Greeks, he points out, used the term "γλαυκώματα " to include all opacities behind the pupil which were not amenable to treatment. They were of the opinion that γλαυκώματα were due to changes in the colour and consistence of the crystalline lens and that cataract was due to a filmy suffusion between the iris and lens and was designated by them "ὑπόχυματα".

Maitre-Jan also held that glaucoma was a disease of the lens.

Brisseau in 1709 demonstrated that cataract was a disease of the lens and concluded that glaucoma was a disease of the vitreous body.

Lippincott ascribes to Brisseau the discovery that the glaucomatous eye is harder than normal. (Diseases of Eye etc., De Schweinitz and Randal, Vol. 1.)

Mackenzie, in his 1834 edition describes the condition of a glaucomatous eye as follows: "appearances lead observers to think that they are looking through the transparent lens at an opaque vitreous humour. At the commencement of the diaease the opacity appears to be more deeply seated; as the disease advances however the apparent opacity, always of a green colour, is seen as if occupying the centre of the vitreous humour, and at last appears to be immediately behind the lens. Limited and sluggish motion of the pupil with other amaurotic symptoms always attends glaucoma. Ultimately the pupil becomes dilated and the retina insensible to light. Loss of sight is very gradual and attended sometimes with diminution of the size of the pupil."

Mackenzie laid great stress on the green colour as the diagnostic point between the opacity in glaucoma and that of lenticular cataract for he says - "opacity of the lens alone is never green." He says that "on dilating the pupil with belladonna the green appearance seems to retire to a greater depth behind the iris and becomes more circumscribed". Now-a-days the use of belladonna might

lead to an action for criminal negligence. I have seen in private practice a case which was evidently taken to be (syphilitic) iritis by an eye specialist, who gave his patient a prescription for atropine drops and who had promptly to perform a wide iridectomy as the result. His subsequent prescriptions contained eserine.

In giving the differential diagnosis between cataract and glaucoma, Mackenzie[2] as early as 1830 laid some weight on the increased tension; he says - "the eyeball in glaucomatous amaurosis always feels firmer than natural while in cataract it presents its usual degree of resistance to the pressure of the finger".

That is probably the first mention of the symptom which we now hold as the distinguishing feature of glaucoma although von Graefe[4] says that increased tension was known in very early times. Von Graefe however, does not state any authority. Fuchs[5] amongst others gives the credit of the discovery to Mackenzie, so also does De Wecker.[6] In talking of acute glaucoma, Mackenzie says that it is often associated with arthritic inflammation, "but if", he adds, "ophthalmia arthritica be really gouty then that disease is much more frequent than practitioners in Scotland will admit, occurring as it does among the poor and ill fed."

It is easy to see from Mackenzie's description of the symptomatology of ophthalmia arthritica that the disease

was no other than acute glaucoma running an unchecked course.

He describes as a terminal phase in ophthalmia arthritica, ulceration of the cornea, with spontaneous expulsion of the lens. We never see now-a-days the ulceration of the cornea with spontaneous expulsion of the lens although cases in which the tension has risen rapidly in eyes the seat of chronic congestive glaucoma and where a corneal section is being made with a view to performing an iridectomy, may then spontaneously expel the lens. I have seen two such cases; in one the lens was immediately followed by vitreous and then copious haemorrhage.

Mackenzie[2] as late as 1854 states the morbid condition of glaucomatous eyes to be -

1. Lens, yellow amber or reddish brown.
2. Vitreous humour, fluid, pellucid, colourless or slightly yellow. No trace of hyaloid membrane.
3. Choroid coat light brown, little or no appearance of pigmentum nigrum.
4. In retina no limbus luteus or foramen centrale.

From the foregoing historical sketch it will be seen that down to the year 1854 although the symptomatology of glaucoma had been carefully and accurately observed, no conception of its true pathologico-anatomical condition had been arrived at and as a result only the vaguest ideas of

its etiology were entertained. Mackenzie noted the occurrence of the disease as being most frequent at or over the age of 50 years and certainly held grave doubts respecting the gouty element in its nature although he continued to use the term "ophthalmia arthritica".

More Recent Theories.

I. Hyper-secretion Theory with special reference to von Graefe's work.

Helmholtz's discovery of the ophthalmoscope (1851) (augen-spiegel) raised the hopes of investigators that the light would shine on the fons et origo mali, but its value proved to be negative. It demonstrated that in true glaucoma those effusions supposed to lie between the retina and choroid did not exist. Jäger's idea that a change in the form of the optic papilla resulting in its projection forward was corrected by von Graefe and Prof. Heinrich Müller in 1856 proved by anatomical demonstration the cupping of the disc.

Ed. Jäger was the first to describe the arching of the retinal vessels within the limit of the papilla and the pulsation of the retinal arteries and von Graefe was the first to look upon these signs as pathognomonic of glaucoma.

That the change in the optic nerve was not a causal one with reference to the other symptoms of glaucoma von Graefe[4] deduced by careful arguments, but he admitted - "that there is a small but incontestable category of cases in which the affection of the optic nerve alone exists and continues unaccompanied by any other symptom".

Schroeder van der Kolk and Arlt were the principal supporters of the view that glaucoma was due to an inflammation of the choroid with effusion between that membrane and the retina and although they advanced pathologico-anatomical facts in its support it was relinquished on these effusions being non-demonstrable by the ophthalmoscope.

Von Graefe believed the hardness of the globe to be due to an overfilling of it with fluid the result of an inflammatory hyper-secretion.

He was aware that the mydriasis was not due to the blindness but to the pressure on the ciliary nerves.

The anaesthesia of the cornea was recognised as being analogous to the mydriasis. Von Graefe believed that the flattening of the anterior chamber was due to diminished curvature of the cornea, and to arching forward of the iris. He held the flattening of the cornea to be an invaluable argument in favour of increased intra-ocular pressure, and he hoped to found on it an accurate method of

measuring the amount of increased pressure, "for", as he says, "the curvature is subject to mathematical estimation by means of Helmholtz's ophthalmometer".

Von Graefe, after careful observation of acute cases in which he had lessened the pressure by paraceutesis and the media becoming clear took the opportunity of examining the optic papilla, concluded that in some groups of the disease at least, the cupping was a secondary effect of the increased pressure and not a cause of it.

In his essay of 1857 von Graefe sums up acute glaucoma as "a choroiditis or irido-choroiditis with diffuse imbibition of the vitreous body and aqueous humour and in which increase of the intra-ocular pressure, compression of the retinal structure and the well known series of secondary symptoms are produced by the increased volume of the vitreous fluid".

In reviewing the effect of iridectomy on the glaucomatous process he observes that "the results are at first apt to make one give up the idea that the disease is not due to a vascular affection of the globe generally but to a purely local disturbance". He adds, however, that "such an unconditional inference, for the present, must be avoided, for," he says, "it may readily be imagined that by changes in the vessels a definite anomaly of the circulation is induced which per se does not abolish the functions

of the internal parts of the eye but only when a local factor is added to it after the required change of the local circulation the original cause might possibly continue without producing the former effect".

While he makes no further suggestion as to the possible nature of the local factor, he drew attention to the frequency of secondary glaucoma after needling operations for cataract and he employed in his own practice iridectomy as a cure for such, where he was unable to extract the lens nucleus by linear extraction. He also noted the fact that the blindness following reclination of the lens by older surgeons and pronounced by them to be due to the onset of "gouty ophthalmia" was a sequel to the increased pressure and cupping of the disc - due to a hypersecretion of the vitreous fluid and increase of the intraocular pressure excited by the irritation of the depressed lens. "The result", he adds, "is an amaurosis with contraction of the field of vision similar to that occurring in the very chronic cases of glaucoma which are attended with few external symptoms".

He also points out that increased intraocular pressure may result from the passage of a foreign body into the interior of the eye and that by hypersecretion of its fluids without any diffuse inflammation.

Donders also believed that glaucoma was the result of a hypersecretion of intraocular fluid but he was of the opinion that such was due to nervous irritation.

The hypersecretion theories of von Graefe and Donders were generally accepted until the retention theory of Knies and Weber was formulated.

II. The Retention Theories. The work of Knies and Weber and the researches of Brailey and Priestley Smith.

It was Leber's researches, however, on the direction of the current of intraocular fluid which showed that the principal outlet for the effete fluid was through the zonula and pupil into the anterior chamber and thence through the pectinate ligament into the canal of Schlemm and from there into the venous system.

Knies and Weber first described the obstruction to this efferent flow of intraocular fluid in glaucomatous eyes as due to the apposition of the iris periphery to the sclero-cornea in the region of the canal of Schlemm with resulting blocking of the "filtration angle".

The crowding forward of the iris base was supposed to be due to the hyperaemic engorgement of the ciliary processes.

Knies considered the adhesion of theiris to the sclero-cornea to be an inflammatory one while Weber regarded

it as due to pressure.

I am not aware, however, of pressure giving rise to adhesions in any other parts of the body, and I think such a result could only take place in the case of the cornea and iris if their endothelial linings became damaged, but there is no evidence to show that such is the case. On the other hand adhesion is found most frequently and much sooner in the acute or sub-acute cases (inflammatory) than in the chronic cases where indeed it may not exist to the slightest degree and where the filtration angle is simply compressed.

In the microphotograph (page 38) of the subacute case detailed, the angle is seen to be closed by the adhesion of the iris, and cornea, but the endothelial lining of the iris at least is seen to be little altered. Were the adhesion due simply to pressure one would expect to find it most frequently in the cases where that force had been acting for the longest periods, namely, in cases of chronic glaucoma, but as already stated it is by no means the rule in these cases. I therefore agree with Knies in believing the adhesion to be an inflammatory one.

Knies now holds that true glaucoma in an iridocyclitis anterior and that glaucoma simplex is an optic nerve atrophy with excavation. Some ophthalmologists agreeing with Knies, refer to the latter as posterior

glaucoma and to the former as anterior glaucoma.

It may be asked if the adhesion of the iris to the sclero-cornea takes place as a result of inflammation, why it is that glaucoma does not take place when there is an acute iritis and when there is hypopyon. In iritis we do see adhesion take place both to the cornea and to the anterior surface of the lens, but only in very rare instances does the adhesion affect more than a few points of the iris surface. Where there is complete adhesion of the iris ~~periphery~~ pupillary margin to the anterior surface of the lens, secondary glaucoma results. This is a rare occurrence in iritis. It is necessary for so-called primary glaucoma resulting from closure of the filtration angle that the whole of the circumference of the corneo-iritic angle be closed. It is possible that in many cases of iritis the corneo-iritic angle is closed to a greater or less extent by adhesions but ~~that~~ as the closure is not complete, filtration of the aqueous humour is still possible and glaucoma does not result. It is impossible to see the corneo-iritic angle in an eye in situ, hence I think it possible that partial adhesion between the iris base and cornea may be of frequent occurrence. We sometimes see glaucoma develop in eyes the seat of old iritis and I am inclined to think that in these cases there may have been adhesion of the iris base to the corneal surface dating from the attack of

iritis which, although only partial, would render a sudden complete blockage a matter of comparative ease.

In order that the adhesion may take place between the iris base and corneal surface there must be a factor at work which results in the pushing forward of the iris base. This is generally admitted to be the turgescence of the ciliary processes.

Brailey[10] made a careful examination of 80 cases, and found in two only, sclerosis of the optic nerve as the sole pathological change, but in the majority of the cases there was present neuritis or sclerosis of the nerve in addition to pathological changes in the iris and ciliary body with blocking of the canal of Schlemm.

In summing up his views on the pathology, Brailey says - "It is certain that inflammation of the ciliary body, iris and optic nerve is always present, and that it is one of the earliest symptoms of glaucoma, being developed previous to the tension".

He thinks that the iris periphery is nearly always applied to the cornea, its advanced position in the first instance resulting from vascular turgescence of the ciliary folds. "Subsequently there arises very rapid atrophy of the ciliary muscle fibres with very commonly dense connective tissue formation between these and in the optic nerve".

Brailey thinks the increased intraocular fluid must be attributed to an inflammatory hypersecretion from the ciliary body and iris, but when the iris has once become applied to the cornea and the natural outflow through Schlemm's canal obstructed, there is established another and a very serious factor in the production of the high tension.

Priestley Smith[11] made a careful examination of a large number of glaucomatous eyes and found that

(a) haemorrhage had been present in many cases, and in some had probably been the starting point;

(b) the filtration angle had been closed in the majority of the cases. He found that the tendency to adhesion of the iris base to the sclerocornea varied with the vascular congestion;

(c) the ciliary processes in the congestive type showed enlargement, the apices directed forward and increased in thickness. Where the glaucoma had been of long standing the processes were sometimes shrunken and retracted;

(d) the lens sometimes in close contact with the processes and iris, more often a little separated from them. He found also a disproportion between the size of the lens and the eye.—The lens being

larger than usual and causing a diminution of the circumlental space in most of his cases, led Priestley Smith to the conclusion that therein lies the real predisposing cause of glaucoma.

He made a series of careful calculations and proved that there is a progressive increase in the size of the lens during life, and that in glaucomatous eyes this increase is more out of proportion to the eye about and after the age of 50 years, and he accounted in that manner for the increased frequency of glaucoma about that age.

Priestly Smith[11] agrees with Adolph Weber that the iris base is pushed forward by the ciliary processes and adheres sooner or later to the opposing surface of the cornea and ligamentum pectinatum, according to the degree of vascular disturbance and inflammatory reaction.

In enquiring into the occurrence of phlebitis of the vortex veins in cases of glaucoma, Priestley Smith examined 13 eyes lost by glaucoma (10 by primary and 3 by secondary), and found in one of subacute primary and one of secondary glaucoma, partial obstruction by formations in their interior such as have been described by Czermak and Birnbacher. In some the vein wall was thickened, and in some the perivascular space was abolished by the close contact of the vein with the wall of the scleral channel.

This he found also in a healthy eye.

Priestley Smith asks if this phlebitis or peri-phlebitis is not rather a result than a cause of glaucoma. He adds "if glaucoma originated in blockage of the veins it would hardly be curable by eserine or iridectomy". In my experience of the disease, however, it was just in cases whose vascular systems showed degenerative changes that the results of iridectomy or eserine were so disappointing, and on account of the comparative frequency of the failure of operation or eserine to stay the progress of the disease in patients with manifestly degenerate vascular systems, I am inclined to believe that such degeneration may be, in them, the primary cause of the increased intraocular fluid, either by permitting a more free exit of the fluid part of the bloodfrom the vessels, under ordinary tension, or by obstruction to thereturn of the venous blood by the blocking of the veins, with increased transudation of serum from the capillaries as a result. Where obstruction to the return of lymph through the perivascular lymph spaces existed along with obstruction to the venous flow, one would naturally expect a more pronounced increase in the tension.

The increased tension in the posterior part of the posterior chamber would result in the pushing forward of the lens and iris until the latter was pressed against the

corneo-sclera so long as the fluid could escape from the anterior chamber. If the fluid occupying the anterior chamber were unable to escape, I think that the advance of the lens and iris could not take place, and this forms an argument in favour of the view that closure of the filtration angle is not the first step in the production of glaucoma.

It is known that experimental rise of pressure in the vitreous chamber will result in forward displacement of the lens and ciliary processes. "In the normal eye the fluid can easily pass from the vitreous to the aqueous chamber but changes in the hyaloid membrane, in the stroma of the vitreous, or in the constitution of the vitreous fluid, may hinder such physiological process, and as a matter of fact the vitreous often shows a more membranous consistence or its septa are found to be coated with albuminous coagula". (Priestley Smith, [12])

The fluid secreted by the ciliary bodies has sometimes been found to collect between the hyaloid membrane and the lens and thereby to cause displacement forward of the latter, which again results in jamming of the iris periphery against the cornea.

Brailey does not think that the lens can by its advancement jam the iris periphery against the cornea but that it would rather jam the free margin against it.

Personally, I think more accurate and extensive demonstration is required to settle this point. I do not think Priestley Smith's[11c.] photographs of sections of eyeballs showing enlargement of the lenses bear out his views on this point.

Snellen[3] believes that an abnormal slackness of the zonula is a factor in the forward displacement of the lens, and he holds this to be more likely to result in old people as a loss of the normal elasticity which accompanies the degenerative processes leading to senile cataract, hence the occurrence of glaucoma seen in connection with cataractous lenses.

Priestley Smith[11c.] sums up the causes of glaucoma as "various and complex, but they are all alike in that they lead to compression of the filtration angle; with that compression the glaucomatous process begins".

The Opinions of Terson, Gasparinni and Abadie, and Laquer.

Mons. A. Terson in the Annales d'Oculistique of July 1901 states that the majority of cases of acute primary glaucoma bear a close resemblance to the acute idiopathic oedema which occurs in various organs of the body but especially in the lungs. He holds that there is always present a diffuse toxaemic condition over the whole

body, which may be either a general infection, such as erysipelas, influenza, etc , or some pronounced diathesis, as rheumatism or gout. He considers hypermetropia as having some influence in predisposing to the acute form, and as the hypermetropia as well as the diathesis may be hereditary, he seeks on that account to explain the occurrence of acute primary glaucoma in successive generations. He holds as necessary to the production of the local hypersecretion a third factor, namely, some transient excitement of a nervous or reflex emotional nature.

He has observed that in the acute forms the blood tension remains normal but that in the subacute and chronic forms it is increased, due to the presence in the latter of a general arterial sclerosis.

In the Ophth: Rev: October 1901, there is an extract from Gasparinni's paper in the Annali di Ottalmologia XXX, iv. 1901, where the existence of intermediate types connecting the two varieties, anterior or acute and posterior or glaucoma simplex, is pointed out and where it is demonstrated that the disease may originally appear in one form and merge into the other. Gasparinni illustrates this by the case of a man who lost one eye by acute glaucoma and who came to him with glaucoma simplex in the other

which later developed acute symptoms. (as a parallel case (see that of D.S. (reported at p.44)

He refers to the difficulty of diagnosis between glaucoma simplex and optic nerve atrophy, and states that it is sometimes impossible until more acute symptoms develop. Gasparinni relates three cases where the onset of glaucoma simplex appeared after and in consequence of retrobulbar neuritis. The patients were aged respectively 70, 58 and 15 years. The onset of the cupping appeared a few weeks after the subsidence of the more acute symptoms of the neuritis.

He believes that the exudation in the course of the neuritis in his cases had the effect of closing the lymphatic canals in the sheath of the optic nerve, thus favouring, if not directly causing the glaucoma by resulting in the cessation of removal of effete matter, which takes place to a slight extent by the nerve sheath. The type of glaucoma most likely to originate under these conditions would, Gasparinni thinks, be posterior glaucoma.

In favour of his contention, Gasparinni points out that no case is known to him where glaucoma followed that type of optic neuritis producing a true amblyopia where only the macular fibres and probably the lymph vessels incorporated with them are compressed.

Against Gasparinni's view however, it may be stated that the function of the lymph spaces in the fibrous stroma of the optic nerve and the sheaths of the retinal vessels as an excretory one for the intraocular fluids, has not been proved, although it is not altogether unlikely. And further, the cupping of the disc is held to be a secondary result of the increased intraocular pressure acting on a weak portion of the globe, so that the occurrence of cupping cannot at present be held as a proof of a true posterior glaucoma as opposed to an anterior glaucoma. Gasparinni does not state in his paper however, that the intraocular tension was abnormally high before the onset of the cupping, but simply that the latter appeared a few weeks after the subsidence of the more acute symptoms of the neuritis. If Gasparinni's observations have been correct and are confirmed by other observers, the theory of the causation of cupped discs will require some revision.

It is generally admitted that cupping of the disc is not found in acute cases which are operated on early and as already mentioned, (page 7), von Graefe concluded that in some groups of the disease at least, cupping of the disc was secondary to the increased pressure. It is worth noting however, in this connection, that Brailey found two cases with sclerosis of the nerve as the sole lesion; and that it was present in the majority of the cases in addition to other lesions probably of a more causal nature.

It is possible that the changes in the nerve which ultimately lead to sclerosis may primarily induce a softening of the tissues which renders them unfit to withstand even the normal intraocular tension.

Laquer was of the opinion that changes in the sclerotic rendering it more rigid and causing probably some shrinking in the size of the globe, produce obstruction of the posterior intraocular lymphatics which find their exits with the venae vorticosae, and so give rise to increased tension.

Mons. Abadie, at the meeting of the French Society of Ophthalmologists in October 1900, stated that he had shown that primary glaucoma was not a disease of the eye but of the sympathetic which innervated the vessels of the globe and that the good resulting from iridectomy really resulted from the section of the nerve fibres in the iris tissue. The results of sympathotomy and sympathectomy, so far, are not uniform enough to make Abadie's opinion applicable to all cases of primary glaucoma, to say the least of it.

ANALYSIS of CASES.

The following analysis I have made from the cases of primary glaucoma of congestive and non-congestive types, which I observed in the wards and out-patients' department of the Glasgow Eye Infirmary, and in the Rochdale Infirmary during my terms of residence there, as well as several cases seen in private practice. In all I have notes on thirty-four cases of primary glaucoma. I have excluded two cases which were doubtfully primary as each had a history of old iritis. Under the term "congestive glaucoma" I include cases of acute, subacute and chronic congestive, while under the term "non-congestive glaucoma" I include that type of glaucoma which, so far as observation and clinical history can determine, has at no period of its course shown any vascular congestive disturbance, in short, glaucoma simplex.

The Influence of Sex.

Of my total of 34 cases, 23, or 67.6% were males, and 11, or 32.4% were females. From this it will be seen that my experience does not coincide with the generally accepted statement that "slightly more men than women suffer from primary glaucoma." I am well aware, however,

that with such a limited number of cases it would be very unwise to draw the inference that such is the general proportion.

(11 c.)

In P. Smith's collection of 1,000 cases the liability to simple non-congestive glaucoma was found to be about equal in both sexes, whereas women were more prone than men to congestive exacerbations.

On further analysis my cases differ from those in P. Smith's collection, for I find that of the total of 34 cases, 11, or 32.3% were those of the congestive type in males, as compared with 9 or 26.5% in females, while further, the disproportion in the non-congestive group is still more striking, there being a very large preponderance of males, the figures being 12, or 35.2% males and only 6% females.

The Influence of Age.

As regards age my cases seem to agree with the analysis of others, in that the greatest proportion occurred in the fifth and sixth decades of life; namely 22 cases, or 64.6% of the total.

My youngest case was aged 32 years, a male suffering from congestive glaucoma of both eyes, (J. H. see p. 92 for Fields of Vision etc.) and my oldest case was also in a male (P. C. aged 77 years.). In the latter's case the increased tension was associated with cataract. The vision in the Rt. E. = fingers at 3 ft., and the field was contracted; T.=n. In the L. E. the vision amounted only to light and shade, and the light projection was very deficient while the T. was fully + 1.

54.43
58.54

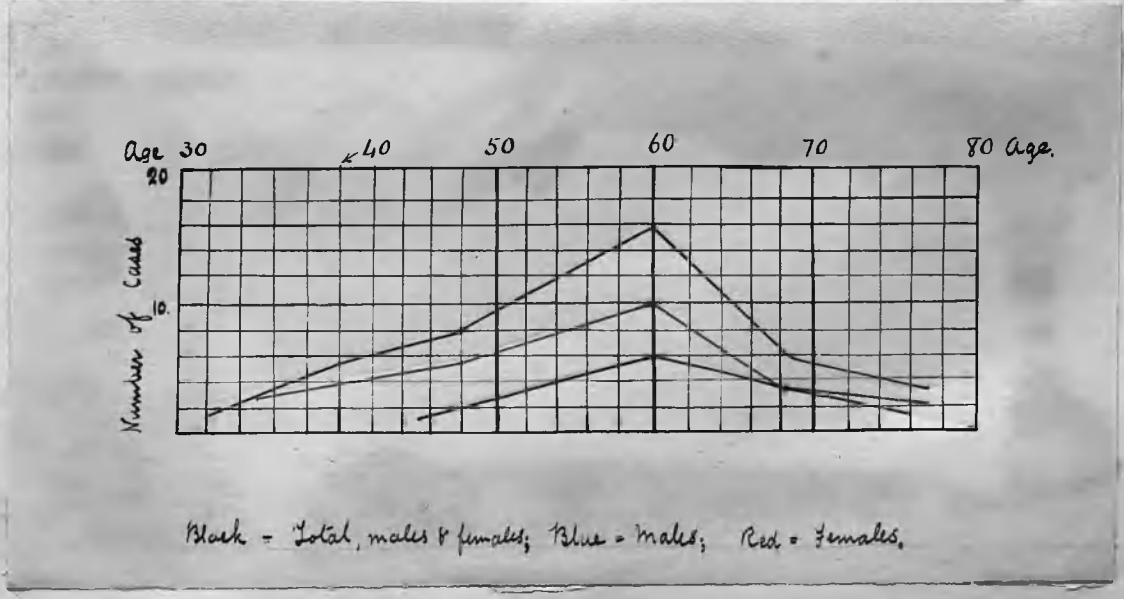
The average ages were males 54.43 years; females 58.54 years; this also is a reversal of the generally accepted figures.

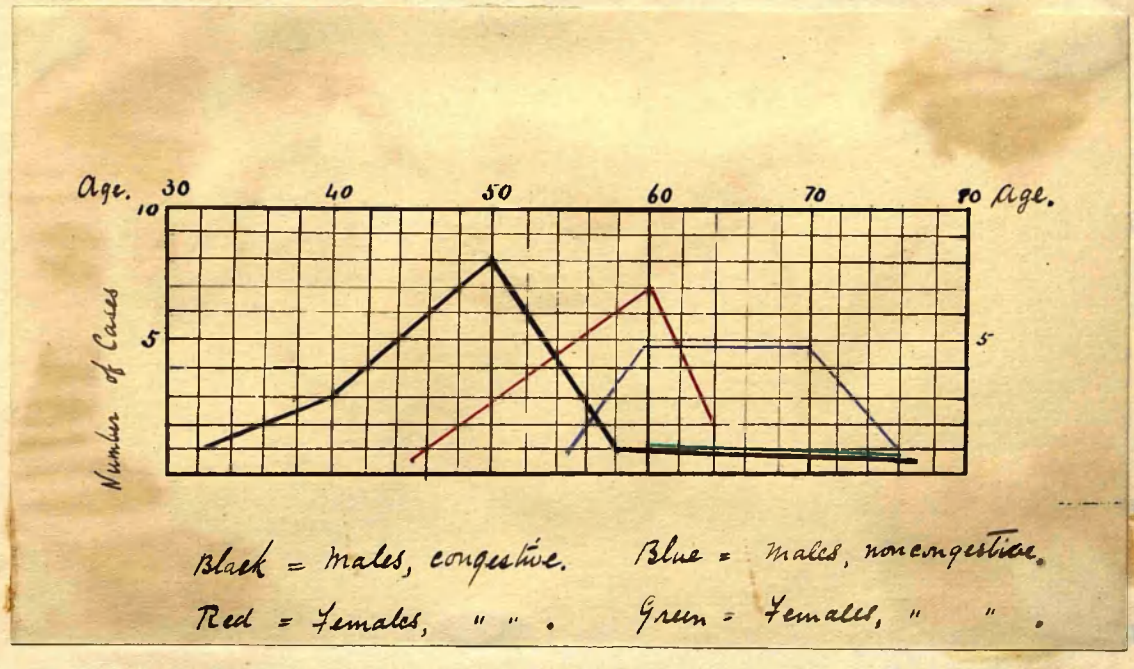
Examining the ages with reference to the sex, and type of glaucoma, I find that, of the males in the congestive group, 8 or 23.5% of the total cases occurred under 50 years, as contrasted with one female, or 2.94% of the total; while 8.8% of the total were males over 50 as against 23.5% for females. Of that percentage 17.64 occurred in females between the ages of 50 and 60 that is at the age at which females are most susceptible to vaso-motor disturbance. Turning to the group of non-congestive glaucoma, I find no case in a male under 50. The proportion is equal between the ages of 50 and 60

and 60 and 70 namely 14.7% while between the years 70 and 80 there are only 2 cases or 5.8%. Amongst the females there was no case of non-congestive glaucoma under 60 years; and there was the same percentage between the years 60 to 70 and 70 to 80, namely 2.94%.

These proportions may be more readily grasped by consulting the subjoined table and diagrams:-

	Congestive.		Non-congestive.	
AGE	MALES	FEMALES	MALES	FEMALES
Between 30 & 40	3 - 8.8%			
40 & 50	5 - 14.7%	1 - 2.94%		
50 & 60	1 - 2.94%	6 - 17.64%	5 - 14.7%	
60 & 70	1 - 2.94%	2 - 5.88%	5 - 14.7%	1 - 2.94%
70 & 80	1 - 2.94%	..	2 - 5.88%	1 - 2.94%
TOTAL	11-32.3%	9 - 26.5%	12 - 35.2%	2 - 5.88%





Influence of Race.

As regards Race, my cases were all amongst Britishers, the very large majority being Scotch, so that I have not had an opportunity of studying any racial peculiarity.

Knapp(3) and Benedict of Breslau state that Jews are disproportionately liable, and Brugsch Bey's (3) observations led him to believe that Egyptians are more liable to the disease than Europeans and that this was due to the smallness of the eyes as measured by the size of the cornea. Egypt is well known as a hotbed of ophthalmia not only amongst children but amongst adults,

and one would naturally expect to find a larger proportion of the adult population suffering from congestive or inflammatory disease of the uveal tract than among other races.

The Influence of Heredity.

De Wecker (6) states that there is a tendency to hereditary transmission, but I was unable to obtain any satisfactory evidence of this among my cases, probably because in the class of patients to which one is accustomed to deal with in hospital there is always a difficulty in eliciting family history where it even concerns only a question of general medical or surgical disease. In enquiries as to previous cases in the family one is met with such indefinite answers as "My mother had inflammation in her eyes"; and attempts at narrowing down the meaning of inflammation usually lead to no definite notion of the disease which did exist. "Inflammation" is a term which is too readily accepted by the uneducated as covering almost every pathological condition. "Cataract" too, is used by the same class to include any and every opacity affecting the cornea or lens.

The Influence of Gout and Rheumatism.

Gouty and Rheumatic diathesis are believed by most observers to predispose to the congestive form of glaucoma, but it is difficult to say whether or not the predisposition is the result of an iritis having been set up, especially in rheumatic individuals, at some former time. Rheumatism is common enough in both England and Scotland, while gout among the middle and lower classes of Scotland is practically unknown, and in my experience of nearly five years English practice I have found it much rarer than I was led to believe during my student days. In a large practice in Durham (Sunderland) during six months I saw no case. In a large general practice in Derby, during two years I saw only one case and in two and a half years in hospital (15 months) and general practice in Lancashire I have only seen one case of gout among the working class.

The Lancashire case was a man aged 35 years who had been a total abstainer for life, but whose father had suffered from gout. So that so far as gout is concerned in this country at least (Britain), it may be neglected as an etiological factor in the production of glaucoma among hospital patients.

A fair proportion of my patients gave a history of rheumatism - mostly chronic. One case had a pretty clear history of sub-acute rheumatism, and the glaucoma was not a direct result of a former iritis, but how much of etiological import could be attributed to the rheumatic tendency is problematical.

The Neurotic Element.

Among the more acute congestive cases there was a distinct neurotic element. The patients showed unmistakeable signs of a highly strung nervous condition. They were excitable and anxious and of what might be called "a worrying disposition." Of that type D. McI. as a male and C. F. as a female were good examples, and in a less degree also my youngest case J. H.

The Influence of Cardiac and Arterial Degeneration.

In the non-congestive and in the chronic congestive groups cardiac and arterial degeneration and chronic bronchitis (probably due to pulmonary vascular degeneration) were marked features of the general condition. These cases were of course most common in the

later decades, and proved more serious so far as the hope of checking the progress of the disease was concerned, probably because in them the mechanical blocking of the filtration angle, which is to some extent removable by operation, is only a comparatively small factor in the causation. In patients with evidence of cardio vascular degeneration one usually finds evidence of impaired functional activity in the absorbent system generally, for example, oedema of the lungs and in the dependent portions of the body, and it is natural to conclude that the absorbent channels for intraocular fluid will also be of less than normal functional activity. In such patients we know that the restitutio ad integrum is seldom attained in any organ which may have been attacked by disease, and on that account alone I think the prognosis is rendered much less hopeful in glaucoma. A striking illustration of this arterial degeneration was afforded by one of my cases of non-congestive glaucoma -in a female aged 64. On coming under observation she had chronic glaucoma of both eyes, which however, in the right had resulted in total blindness. In the left there was slight cupping of the disc with a somewhat restricted field of vision. During the two months previcus to being admitted to the Rochdale Infirmary she had suffered great pain in the right eye.

I advised enucleation, but this was refused. The surgeon in charge of the case then tried to do a sclerotomy, but unfortunately injured the lens capsule with the von Graefe knife, and in less than 36 hours the lens had swollen up and the increased tension and consequently very much increased pain necessitated an attempt at removing the lens. Holocaine (1% solution) was instilled and the usual corneo-scleral incision made; as soon as this was completed the lens was spontaneously expelled, and followed immediately by vitreous and then profuse haemorrhage; the eye subsequently shrivelled. The left eye became rapidly worse and took on a congestive character and the vision became rapidly deteriorated.

The Size and Shape of the Eyeball.

It is stated in most text books that the greatest proportion of cases occurs in hypermetropes, and that this is accounted for by the smallness of hypermetropic eyes, with a consequent diminution in the circumlental space, which again is held to be partly a result of a hypertrophied ciliary muscle and partly (P. Smith(11)) a result of enlargement of the lens.

The measurements of the corneæ have been taken as the basis of the estimation of the size of the eyes in glaucoma and it has been found that the average size of the corneæ in glaucoma is less than normal, the proportion being 11.6 m. m. in normal eyes, and 11.1 m. m. in glaucomatous eyes. Exceptions, however, are by no means infrequent, and the case of D. McI. is an example.

Exciting Causes.

1. Cold.

The exciting cause in most of my acute cases was put down to "cold", while in the case of one female I considered it highly probable that over indulgence in

2. Alcoholic Excess.

alcoholic drinks had precipitated the attack which was equally severe in both eyes. Priestley Smith (11 c.) also considers that drinking bouts may in the predisposed bring on an attack.

3. Mental Disturbance

Anxiety, worry or sorrow are also held to be exciting causes, and as I have already stated, the neurotic element was marked in my acute cases, and I have no doubt that in them any sudden explosion of nerve energy might lead to a disturbance of the balance of control over the vascular system of the eyeball with a resulting active or passive congestion and consequently hypersecretion of the intraocular fluids.

4.Mydriatics. The use of atropine has resulted in attacks of glaucoma in the predisposed and I know of several examples of this, one I have already mentioned. In a case where the diagnosis was uncertain and the line of treatment also difficult to decide, a friend of mine instilled a drop of a sol. of atropine and in 12 hours there was no longer doubt as to the disease or its appropriate treatment.

Cocaine has on several occasions had the same effect. Cases have been recorded by Dr. Hinshelwood[15] and Dr. Snell.[14]

SELECTED CASE RECORDS.

I have selected the following cases as fairly typical examples of the different types of glaucoma which I have observed.

SUB-ACUTE GLAUCOMA.

Case Record 1. D. McI. Age 41. May 6th.

For the last 13 years has had to wear glasses for reading, but has not hitherto had any inflammatory mischief in eyes. There is no history of injury. Present illness began about Jan. 19th. when he "caught cold". On Jan. 20th. patient had rigors (?) and on the 24th he felt a tightness over the eyes, and on looking at the gas light he saw with his left eye only a rainbow of red, green and blue. His eye was poulticed and "eye drops" were put in. Five weeks later the vision amounted only to light and shadows. Any sudden noise at that time, such as the slamming of a door, caused a sensation of flashes of light in the affected eye. Patient states that about six weeks later the vision had improved until he could see, with the aid of a glass

held before his eye, letters of about ½ inch at a distance of about 20 ft. but only on moving the head from side to side and not when looking directly forward (eccentric vision) On April 29th. the pain recurred and the vision again diminished. He then sought advice at the G. E. I.

On examining the patient on May 5th. I noted;— General appearance, rather under average height, slight build, dark complexion, patient of a decidedly nervous temperament, jerky in his movements and to some extent in his speech, but mentally alert and willingly gives full details of previous history etc. No evidence of any general disease.

Eyes: V. A. R. $= \frac{20}{50}$, V. A. L. $= \frac{20}{100}$.

Nothing abnormal to note in external appearance of the right eye. Ophthalmoscopic examination of right eye shows nerve head red, no cup and just below and to nasal side bundles of persistent nerve sheaths. Hypermetropia + 2D. Externally, the left eye shows some congestion of conjunctiva, dilatation of the episcleral veins, dilatation of pupil and shallowness of anter. chamber. There is a very slight haziness at one point of the corneal surface external to the centre.

Ophthalmoscopic examination shows cupping of the disc of about 1 m.m. in depth (fundus generally = + 3D, floor of cup = -.5D) Around disc are a few atrophic spots, while scattered here and there in the course of the vessels are haemorrhages of moderate size, dark brown in appearances. Macula looks free from disease but cannot be very clearly seen; near macula the vessels are focussed with + 2.5D while at disc with +3D. No pulsation visible in arteries. Veins slightly dilated. Tension + 2 in Left Eye, normal in Rt. E. In external examination of left eye, there is also noted to the outer side a bulging outwards of the sclera of a bluish colour such as might suggest a melanosarcoma, but there is no evidence of such on O.E.. Bulging is a simple ectasia from increased intraocular tension.

May 14th. Sclerotomy was performed and eserine drops instilled.

May 17th. Tension nearly normal but V = $\frac{6}{200}$.

May 19th. Tension normal and V.A. improved to $\frac{12}{200}$. ectasia still prominent.

May 21st. Pain has returned and enucleation was performed.

May 25th. V.A.R. $\frac{20}{40}$ and Visual Field as on admission (see chart of perimetric tracings) The great contraction of the visual field of the left is to be noted for white light and also for colour. There is also some contraction of the visual field of right.

July 26th. Patient again examined.
V.A.R. = $\frac{20}{40}$ Marked improvement of V. F. see chart. pg 78.
And O.E. shows disc to be of normal colour.

Examination of the enucleated eye shows it to be of normal size, measuring 24.2 m.m. in antero posterior diameter and 24 m.m. in vert. equator, diameter.

Lens is of large size measuring 10.5 m.m. in equatorial diameter and 4.25 m.m. in anter. poster,. The cornea-iritic angle is apparently obstructed by pressure forward of large lens which entirely fills the interciliary space.

No evidence of inflammatory deposit at any point. Iris free of sclerotomy wounds. Vitreous is perfectly free from opacity, being clear and bright.

Retina and choroid appear to be thinned. Numerous haemorrhages scattered over retina and following more or less closely the retinal vessels.

Vessels not tortuous and but little more than normal size. Macula clear and has no haemorrhage in it.

Ectatic region shows simple thinning of all the structures involved. No evidence of neoplasm.

The photograph above was kindly made for me by Dr. George Coats who was at the time my colleague in residence at the G. E. I.

The iris is seen closely adherent to the sclero-cornea, there is well marked rounding of the new cornea-iritic angle. Schlemm's canal is clearly shown. The ciliary processes are somewhat enlarged and directed forwards.

Case 2. Sub-acute Glaucoma C. F. Age 45, Female single.

Patient is of a decidedly nervous temperament, easily alarmed and very anxious about her prospects of improvement in her vision in the diseased eye and makes anxious enquiries regarding the condition of the unaffected eye. Her anxiety in that respect is no doubt due in some measure to the fact that she obtains her living entirely by sewing.

Her previous history is one of poor general health, she "never has been strong." Menstruation did not commence until she was 19 years of age, and after being established for a short time ceased until she was 24, and then occurred irregularly until she was 36 years old and from that time until now has only appeared once. Bowels have been regular.

Present illness. Patient complains of severe pain in supra-orlital region following immediately a feeling of

swelling and tightness in the right eye. The attack usually lasts for about two hours and seems to be brought on by straining to see to sew or read, more especially at night, indeed the attacks mostly have come on after the patient had retired for the night.

The attacks have been accompanied by coloured halos, which patient says appeared to be close to her eye and not around the gas light. They have been attended by loss of vision, which has become marked also in the intervals between the more acute seizures. The symptoms have lasted for about one year.

7.6.00. Examination shows the Right Eye to be congested as regards the conjunctival and episcleral vessels; the anterior chamber is shallow, the pupil dilated and the iris immobile; the cornea is not steamy. The disc is seen with the ophthalmoscope to be distinctly cupped. Tension is almost +2. Vision amounts to counting fingers and the visual field is very much contracted (see chart page 41.)

The left eye presents no departure from the normal in external examination, but ophthalmoscopic examination reveals slight cupping of the optic disc and the

acuity of vision is found to amount only to $\frac{20}{40}$, while the perimetric tracing shows some contraction at both the nasal and temporal aspects. On critical examination the Tension is slightly +. No symptoms have ever been noticed by the patient which she could refer to the left eye.

11.6.00. Iridectomy was performed in the right eye with relief of the tension and pain, although the tension did not reach normal for several weeks. (Hyphoema had developed)

13.7.00. Tension normal in right, practically no improvement in the vision or in the visual field, but the visual field of the left shows a slight improvement.

18.7.00. Tension in operated eye remains normal or almost so. Vision counting fingers.

Iridectomy as a prophylaetic measure advised for left eye

During many of her attacks of pain in the eye patient suffered from polyuria with pain in the back, but examination of the urine reveals nothing abnormal.

Case Record 3. Chronic Congestive Glaucoma J. F. Male Age 44

(1897) Patient first came under observation three years ago with a history of gradually increasing dimness of vision over a period of 18 months affecting the left eye. This was unaccompanied by any visible congestion or pain until

a fortnight before seeking any advice. On examination at that time the eye was congested, pupil dilated, with a greenish reflex from behind it. Fundus hardly visible, vision barely light and shadow, and Tension + 1.

Iridectomy was performed, but 6 days later the T = +. Fourteen days later the T = n and eye quiet. On Feb. 27.00. patient was again seen and then complained of seeing halos round the gas flame with the right eye, attended by attacks of pain and dimness of vision. These symptoms had lasted for 3 months. The tension = + 2. The pupil was small and the fundus just visible. A cystoid cicatrix had formed at the site of the previous iridectomy in the left eye.

The V. A. in the left eye then equalled L. & S. and in the right eye $\frac{20}{50}$ (i.e. 3 months from the commencement of symptoms in it.)

The following note is a summary of the progress of the case during the succeeding seven months.

Feb.28th. 1st. operation, sclerotomy, 3 days later T = n.

10 " " T = + 1.

2nd. operation, 14 days after the 1st.

4 days later T = n.

July 3rd. 13 weeks later T = + 1.

and V.A.R. = $\frac{20}{70}$ V.A.L. = L + D.

3rd. operation, Iridectomy, conjunctival flap tucked into wound. 13 days later T = + ?

4 weeks later T = + 1

Aug.15th. V.A.R. = $\frac{20}{100}$. V.A.L. = Nil. R.V.F. much contracted

4th. operation, Sclerotomy 1 day later T = n

5 days later T = + ?

5th. operation, 7 days after 4th. Sclerotomy with ◇ shaped knife and conj. flap tucked into wound -

12 days later T = n

and V improved to $\frac{20}{70}$ and V.F. also improved.

(See charts p. 80.)

Remarks: This patient was of a quiet and rather melancholic temperament. He presented rather rigid arteries for his age and had a general appearance of premature senility. He was of a spare build. Although his visual acuity rose to $\frac{20}{70}$ he had great difficulty in steering himself about on account of the extreme contraction of his field of vision.

The repeated failures of the operations of sclerotomy and iridectomy to maintain a normal tension impressed me very much. Each operation was performed

with unimpeachable dexterity and I was led to the conclusion that t he treatment by iridectomy and sclerotomy was unsuccessful in maintaining the normal tension, because it did not affect the fous et origo mali. Had the rise of tension been due solely to closure of the canal of Schlemm, I feel convinced that a far different result would have been obtained.

Case Record 4. D. S. Male age 43. 23.3.00.

Patient about medium height and build generally, rather florid countenance, hair greyish and general appearance that of a man over 50 years. Gives a history of gradual increase in dimness of vision over a period of nine years without any inflammatory attacks in his eyes. About 7 weeks ago he says he "caught cold" He suffered acute pain in his left supraorbital region and very considerable diminution in his vision.

Left eye. On examination the conjunctiva is congested, the cornea hazy, anterior chamber shallow, pupil dilated T +3. Visual acuity = fingers. Ophthalmoscopic examination shows that the fundus reflex can be obtained but the details cannot be seen.

Right eye. Externally there is no congestion. Cornea is clear, pupil is contracted (by eserine), anterior chamber is shallow T+ ? Ophthalmoscope reveals cupping of the disc.

26.3.00. Iridectomy in left eye. 27th. left = T n.

Examination 13 days after the operation reveals cupping of the disc to about the same extent as in the right eye, and the perimetric tracings show a most remarkable similarity in the amount of contraction. (see p. 74)

It is quite evident that for a number of years both eyes have been the seat of glaucoma simplex, presenting to the patient no symptom except increasing dimness of vision, and that less than two months ago acute glaucoma has been implanted in the left eye on the chronic affection. Why it only attacked the left eye could not be determined clinically. The only exciting cause which the patient could imagine was "cold" but that was rather indefinite and would have been almost as likely to attack both eyes as one only, nevertheless one frequently sees acute conjunctivitis supposed to be due to cold affecting one eye only.

Case Record 5. Cataract with Glaucoma W. P. male age 60 yrs.

Patient is under middle height but presents no peculiarity. His arteries are not unusually hard for his age and he has no history of any past illness which might have any bearing on his eye condition.

Patient first came under observation five years ago when he was suffering from glaucomatous symptoms associated with cataract in the left eye. An iridectomy was at once performed and several months afterwards the lens was extracted in the usual way. It was noticed that on the first examination the right eye (non-glaucomatous) showed a very large physiological cup.

May 26th. Patient reappears with subacute glaucoma in the right eye, while the left presents normal tension.

V.A.R. = $\frac{20}{100}$ V.A.L. with glasses = $\frac{20}{100}$.

The tension which was about + 2 was reduced by sclerotomy to normal in the R.E., but in two months the symptoms returned and there was a marked lowering of the visual acuity of the left eye as well as the right (After the sclerotomy had lowered the tension the vision of the L. E. had improved to $\frac{20}{50}$ while that of the R. E. remained at $\frac{20}{100}$ with the aid of correction for 4 D of hypermetropia) Now, two months after the operation the V.A.R. = $\frac{10}{200}$

Iridectomy was performed with relief of all pain and lowering of the tension to slightly - minus. A fortnight later a perimeter tracing showed slight increase in the contraction of the nasal side with, however, improvement at the temporal side as compared with the chart taken after the first sclerotomy, 2½ months previously.

CONSIDERATION of the FIELDS OF VISION IN GLAUCOMA.

The generally accepted opinion concerning the field of vision in glaucoma is that it is contracted mostly on the nasal side.

Swanzy (13) states that "as a rule the contraction commences at the nasal side while at the same time central vision is lowered and later on the temporal portion of the field becomes contracted."

Lippincot (3) states that "the nasal side suffers most."

With a view to examining the accuracy of this statement I have made a critical examination of 20 fields of vision of eyes suffering from glaucoma of both congestive (subacute and chronic congestive) and non-congestive types, and my conclusions in the main differ from the generally accepted statements mentioned above. I have measured in degrees the actual contraction which has taken place in the various meridians as compared with the normal field, because I think that neglect of this has probably given rise to the impression that the contraction is mainly on the nasal side. Looking at a

perimeter tracing in a case of glaucoma it is true that one will in the majority of cases find that the smaller portion of the field is on the nasal side, but the same holds true of the normal visual field, and I therefore think that unless the contraction from the normal is greater, it is incorrect to state that the nasal side suffers most.

In the subjoined table I have given the amounts of contraction in degrees, (1) in the horizontal meridians, (2) in the meridians of greatest contraction, and these I have specified by ∠x. corresponding to those marked on McHardy's design of chart which I have used in every case.

Briefly it may be stated that out of the 20 eyes examined, in five only (4 of glaucoma simplex, 1 congestive glaucoma) do the visual fields show a greater amount of contraction in the horizontal meridians. In the meridians of maximum contraction again, five only show the greatest amount of contraction to be on the nasal side.

I think that these figures prove sufficiently that it is the exception and not the rule for the nasal side to suffer most.

That the contraction on the nasal side however is less amenable to improvement by operation is seen by

comparing the charts taken before and after operation. It will be observed in these cases almost without exception, that the improvement has been greater on the temporal aspect of the field; this improvement is only temporarily greater however in these cases whose progress is only made less rapid (that is in the majority), for on subsequent examination the balance of contraction appears to be readjusted.

TABLE showing CONTRACTION IN DEGREES.

Case	Type	Horizontal Meridian				Meridian of Maximum Contraction.							
		Left Eye		Right Eye		Left Eye				Right Eye			
		Nasal	Temp.	Nasal	Temp.	Nasal.		Temp.		Nasal.		Temp.	
H.C.	S	30°	33°	20°	30°	∠60	35°	∠135	25°	∠30	28°	∠135	32°
D.S.	C+S	5°	35°	5°	35°	∠45	30°	∠90	35°	∠60	25°	∠90/135	35°
R.F.	S	2°	30°	45°	18°	∠60	12°	∠90/120	30°	∠60	54°	∠120	40°
L.S.	C	30°	64°	10°	45°	∠60	42°	∠120	60°	∠30	30°	∠120	55°
H.M'K.	S	45°	50°	15°	30°	∠60	52°	∠120	52°	∠45	30°	∠135	40°
D.M'K.	C	35°	75°			∠60	52°	∠120	75°				
P.C.	S			50°	38°					∠60	60°	∠135	42°
L.M.	S			30°	15°					∠60	50°	∠120	50°
W.P.	S			8°	25°					∠60	42°	∠135	32°
C.F.	C	10°	10°	50°	75°	∠60	12°	∠120	23°	∠45	55°	∠135	82°
J.H.	C	10°	18°	45°	28°	∠75	15°	∠60°	35°	∠75	55°	∠135	82°
J.P.	S	(sector)		5°	5°	(sector only)				∠60°	20°	∠120	20°

As regards the colour fields, I have been unable to find any constant rule in their contraction. In many cases the contraction of the colour field seems not disproportionate to that for white light, while in some it seems to be altogether out of proportion. Nor does the contraction in the field for one colour bear any constant proportion to that for another colour.

Green appears to be the most constant, in that it is almost invariably the most limited.

Red appears to provide the greatest amount of variation, at times it is very much contracted and well within the field for blue colour, while in other cases it may overlap that for blue at several points and may even show a marked difference in its proportion on the nasal as compared with the temporal aspect of the visual field.

In the case of H. McK. after operation the field for red extended in the horizontal meridian on the temporal side beyond the limit for white light, indeed at 50° horizontal the white disc was only recognisable as red. (see p. 79)

After operation (iridectomy or sclerotomy) the colour fields show irregularities in their improvement and seem to bear no direct proportion to the improvement or otherwise in the field for white, nor indeed to their areas before operation. I append several charts which bring out these irregularities.

CENTRAL VISUAL ACUITY.

As a general rule there is a proportionate loss of central visual acuity directly with the diminution in peripheral visual acuity, but many and marked exceptions may be observed. It is more general however, especially in the more chronic congestive types, to find a tolerable amount of central acuity of vision existing with a considerable contraction of the visual field than vice versa.

In the acute congestive forms of course, central vision is rapidly reduced to counting fingers at a short distance or even to difficulty in distinguishing light and shadows. As examples for comparison may be cited the visual fields of C. F. (p. 91) with those of J. F. (p. 80) The contraction in the Rt. eyesof C. F. and J. F. being extreme while the visual acuity of C. F. = Fingers; that of J. F. $=\frac{20}{70}$ Again H. McK's (p. 79) right eye shows a well contracted V.F. and yet his V.A. $=\frac{6}{6}$ letters; compare the left V.F. of R. F. p. 87 with the contraction of the right V.F. of H. McK. and it will be seen to suffer less, considerably less indeed, and yet the visual acuity is equal to only $\frac{15}{200}$; a very striking difference.

I think it may be stated as a general rule that the longer the duration of the disease the less disproportion is there between the central visual acuity and the peripheral visual limit.

There is a further point to which I wish to draw attention and which in my opinion is of considerable importance. I have not seen it mentioned in any text book or monograph on the subject of glaucoma. It is this.

On examining the past histories of several cases of double glaucoma, I found that while one eye was described as suffering from all the symptoms of a congestive glaucoma, the other eye showed good central visual acuity, and was described as having no symptom of glaucoma, but as presenting a large physiological cup. No perimeter tracings were given however. These patients turned up several years afterwards with glaucoma, either congestive or chronic, in the eye formerly unaffected so far as was ascertained, and presented cupping of the disc of a character typical of glaucoma, and on taking a perimeter tracing the visual field was contracted.

In more than one case in which I diagnosed in the apparently healthy eye (the other suffering from glaucoma) a physiological cup, and the diagnosis being confirmed by others more competent to judge, I found the visual field contracted.

Examples of the first class are W. P. p5. 85 of the second J. H. p5 82 (see charts.)

Again, in quite a fair proportion of the congestive type of cases I found that, while the eye believed to be unaffected because it showed no congestion, no pupillary disturbance, little or no defect in central visual acuity, with no loss of accommodation and no rise of tension, on taking a perimeter tracing there was undoubted contraction of the field of vision, and moreover, after operation on the undoubtedly glaucomatous eye, this contraction in the quasi sound eye diminished or entirely disappeared with improvement also in the central vision where this had been impaired.

A very excellent example of this is the case of D. McI, already described in some detail. In his case the glaucomatous eye was enucleated, and a comparison of the charts of the field of vision of the remaining eye taken respectively before the operation and about a week

and again about two months after, presents this improvement in a notable degree. The field of vision a week after the enucleation of the glaucomatous eye, I have noted as being the same as before, but the visual acuity had improved from $\frac{20}{50}$ to $\frac{20}{40}$. The chart taken two months after shows a field even larger than normal, but the vision was still = $\frac{20}{40}$ (uncorrected for hypermetropia).

In the case of J. H. (p. 82) I unfortunately did not get a tracing of the left eye (physiolog. cup) after the iridectomy on the right eye, but the tracing taken after the iridectomy on the left, shows a significant improvement in the visual field, and yet this eye had no evidence of glaucoma other than a diminution of vision (known to the patient for a very long time) and a contracted field. The case already mentioned also shows that even with a central visual acuity of $\frac{6}{6}$ (nearly), a contracted field may be the only evidence of an affection of the eye which can scarcely be other than of a very early glaucomatous nature.

In D. M.'s case (p. 78) too, I think it is noteworthy that ophthalmoscopic examination showed the nerve in the right to be red.

To my mind the above observations have a further significance in view of what may be observed in cases of injuries to eyes. It was pointed out to me by a surgeon to the Glasgow Eye Infirmary, and in the accident ward I

(Dr F. Ferguson)

had ample opportunity of confirming the observation, that where an injury, of a nature which experience has shown to be liable to result in sympathetic ophthalmia, has been inflicted on one eye, careful watch of its fellow from the very commencement of the trouble, by ophthalmoscope and perimeter tracings, may reveal, in a red nerve and a commencing contraction of the field of vision, the very earliest appearance of sympathy, even where externally there is no appearance of any sympathetic trouble. Prompt enucleation of the injured eye resulted invariably in complete disappearance of the redness of the nerve and contraction of the visual field.

It was this which led me to look for any similar phenomenon in the case of glaucoma, and I think the cases which I have cited above show that such does occur.

While a surgeon has no hesitation, however, in enucleating the injured eye which is exciting sympathetic inflammation in its fellow, it is not thought justifiable to enucleate an eye in the early stage of glaucoma, unless the symptoms are very distressing and sclerotomy or iridectomy have failed in relieving the pain and checking the rapid deterioration of vision, and the signs generally indicate that recovery of good functional activity is not to be expected. Such was the case in D.Mc.I. and the improvement in the vision and visual field of the ? unaffected eye I think justified the operation.

Enucleation of one glaucomatous eye, after the other has advanced somewhat in the disease, is not attended by any improvement, and this is parallel to what is found in the case of sympathetic ophthalmitis where, after the condition has advanced in the sympathising eye, it is not considered good treatment to excise the original irritating eye.

The behaviour of the sympathising eye in cases of injury so closely resembles what takes place in cases of glaucoma that I am led to believe that in glaucoma of one eye there is a true sympathetic influence felt in the other.

I think that such sympathetic influence is excited more distinctly in cases of congestive glaucoma of acute an subacute character, but may possibly occur also in the more chronic affection, although in such it would be more difficult to prove, as cases do not come early enough under observation to let us see the effect of early operation.

In view of the fact that the sympathetie (?) contraction of the field may be present without either cupping of the disc or the slightest appreciable rise in tension, or indeed in some cases without any diminution of the central visual acuity, although this is present in most cases, as it is in the quasi-sympathetic neuritis in injury, and that it improved or disappears with operation on the glaucomatous

eye, I cannot accept von Graefe's statement "that typical glaucoma of one eye has no sympathetic influence on the other". (see von Graefe's second essay in Arch. f. Ophth. 1858)

Whatever may be the pathology of the early stage of sympathetic ophthalmitis already described as evidenced by redness of the optic nerve head with contraction of the field of vision, when fully established it is usually a condition of serous irido-cyclitis. As already stated in this essay, Knies now looks upon true glaucoma as an irido cyclitis.

CONCERNING TREATMENT.

In the earlier days treatment of glaucoma consisted in antiphlogistic measures more or less severe, and being attended by no very happy results, the disease came to be regarded as quite incurable.

Belladonna was given a fairly extensive trial and Mackenzie[1] recommended its use in his early edition of his work, but added, "sometimes its use is attended by an aggravation of the symptoms and it should then be discontinued".

Mackenzie was probably the first to draw attention to the increase of intraocular tension as a constant sign in glaucoma, and he was certainly the first to attempt a reduction of the abnormal tension by puncture of the globe.

The results of paracentesis, however, were of a somewhat disappointing nature, for although they showed an immediate benefit in lowering the tension and removing the steaminess of the cornea, they very seldom were of a permanently beneficial nature, nevertheless, they pointed out the direction in which rational treatment ought to be carried out.

To von Graefe[4] belongs the honour of introducing Iridectomy as a curative measure in glaucoma.

He had already used iridectomy in iritis and iridochoroiditis but it was not until he had used it as a therapeutical agent in cases of ulcer of the cornea that he thought it might have a beneficial action in glaucoma.

In describing his success in cases of idiopathic ulcer of the cornea he says, "the value of iridectomy in such a case does not depend simply on the paracentesis of the anterior chamber, as the wound being through healthy tissue rapidly closes - the result is a diminution of the intraocular tension due to the lessened secreting surface of the iris" and adds, "besides I have often found, weeks after unilateral operations, that the eye on which I had operated was softer to the touch than the healthy one".

Hehad also observed its beneficial action in anterior staphylomata and in leucoma adherens. He tried it in animals' eyes and noted a slight diminution of the physiological tension. In consideration of these results he thought he was justified in trying the effect of iridectomy for glaucoma.

Von Graefe performed iridectomy as a cure for glaucoma first in June 1856.

In his paper on Iridectomy in Glaucoma and on the Glaucomatous Process in the Archiv. f. Ophthalm. Berlin,

1857 he quotes in detail 9 cases operated on for acute and subacute glaucoma and 4 for chronic glaucoma.

In describing the operation he lays stress on the fact, borne out by subsequent years of experience of others and more recently by our knowledge of the morbid anatomy, that it is necessary for the best success to make the incision in the sclera so as to take away the portion of the iris as near to the ciliary processes as possible.

As regards the question of performing iridectomy in acute and subacute glaucoma, there are scarcely two opinions. Practically every ophthalmic surgeon advises it and most advise it also in the premonitory stage. (Mr. Treacher Collins is one exception I have met, he does not operate so long as eserine can relieve the symptoms).

Although there is such a concensus of opinion regarding the value of iridectomy in acute glaucoma, there is nevertheless considerable difference of opinion regarding its mode of action in relieving or curing the disease. Many surgeons believe that by iridectomy the filtration angle is re-opened and that the escape of intraocular fluid is rendered easy again. De Wecker maintains that the happy result is due to the formation of a permeable cicatrix or to the establishment of communication with the veins. (Therapeutique oculaire, pt.1. p.381.)

The benefit noticed in cases where a cystoid cicatrix had formed induced Dr. T. S. Meighan of Glasgow, to try the effect of tucking into the sclerocorneal wound a fold of the conjunctiva, in order if possible to make a fistula, which, by being lined with epithelial cells, would have less tendency to close.

Major Herbert, I.M.S. published in June of 1903 his impressions of about two years' experience of this method and these were of rather a favourable trend.

Regarding the advisability of iridectomy in Glaucoma simplex the greatest diversity of opinion exists. The majority of surgeons, I think, perform iridectomy in such cases but from the results in the cases I have personally examined, I am fairly certain that in the great majority of the cases little more than the immediate lowering of the tension and the symptoms directly due to such raised tension, were attained. In a few weeks or months the conditions were in statu quo antea, so far as the tension was concerned, and a progressive diminution of the visual field showed the steady march of the disease. Some cases certainly showed improvement and of no unequivocal degree (e.g. L.McL. V.A.R. = $\frac{20}{100}$ before oper. $\frac{20}{70}$ after. p. 88. visual field markedly improved, see chart), but these are exceptions.

The benefit resulting in some cases, coupled with the small risk attached to the operation itself, justifies in the present state of our knowledge the performance of the operation, provided a guarded prognosis has been given to his friends or to the patient himself. De Wecker, in a paper read to the French Society of Ophthalmologists at their Congress in 1901, showed himself a zealous advocate of iridectomy in Glaucoma Simplex. His statements, however, excited a heated controversy on this point: "Ophthalmic Review" (p. 294, Oct. 1901)

The late Mr. David Little of Manchester in his presidential address to the Ophth. Soc. Un. Kingdom. Oct. 17. 1901 gave notes of 37 cases of glaucoma (mixed) which he had been able to trace. Of the 37 cases, 10 had become blind, one from atrophy of the globe, another from recurrence of the glaucoma and the remaining eight from atrophy of the optic nerve, the tension in these cases remaining normal. In 4 of the remaining 27, the vision had materially deteriorated, and they were still going down hill from atrophy of the nerve. In the remaining 23, the vision in some was better, in most about the same, and in a few others a little worse than at the time of the operation.

Sixty-two and a half per cent therefore, had held their ground, seven to 13 years after the operation. He mentioned two other cases which, 20 and 24 years after the operation, had retained visual acuity of $\frac{6}{9}$ and normal respectively. He concluded by saying that his wide experience had increased his confidence in iridectomy in glaucoma.

At the French Soc. of Ophth. Oct. 1900 M. Joegs summed up his opinion of the value of iridectomy in that it depended on the reestablishment of the communication between the two chambers .. .. and on the power of the tissues at the angle to resume their absorptive function.

I have already mentioned (page 21) M. Abadie's view expressed at the same meeting.

Grünert read a paper at the Heidelberg Ophthalmological Soc. on Hofmeister's operations on the superior cervical ganglion in 15 cases, 11 gangliectomies and 4 sympathicotomies. The results were, in 4 cases of chronic inflammatory glaucoma, improvement; in 3 of glaucoma absolutum, 1 failure, 1 good and 1 poor result; in 2 of glaucoma simplex no improvement; in 2 of hydrophthalmos, no result. Sympathicotomy was done in 4 cases in order to obviate the atrophy of the dilator iridis, which is believed to result from the removal of its trophic nerves in the

ganglion. Of these, two were hydrophthalmos and no good resulted; two were cases of glaucoma simplex and so far, these were improved.

So far the number of cases operated upon is not large enough to draw any definite conclusion from, but it would seem that in the chronic congestive type these operations have in certain cases a distinct therapeutical action.

I have never seen a case on which sympathicotomy or gangliectomy has been performed.

The only operation which rivals iridectomy is sclerotomy and as usually performed it is certainly second in its results. By most surgeons it is only used where an iridectomy has been already performed.

Local medicinal treatment now practically resolves itself into the application of myotics and with these local anaesthetics.

The myotic used is eserine or pilocarpin in varying strengths, usually about 1 in 500. The local anaesthetic applied is, as a rule, cocaine hydrochlorate in a strength of about 1 - 100.

Cocaine, although a dilator of the pupil, contracts the ciliary blood vessels and by diminishing the sensibility of the ciliary nerves relieves the pain.

More recently Dr. Hinshelwood has employed holocaine with eserine. Holocaine does not dilate the pupil and is a more powerful anaesthetic than cocaine. I have seen it used with advantage on several occasions. Holocaine dilates the conjunctival vessels, but I have seen no bad effect on this account in using it in glaucoma, probably on account of its dilating action being antagonised by the eserine.

M. M. Terson and Darier [14] speak highly of the use of dionine in 2½% solution in congestive glaucoma. M. Darier says the beneficial analgesic action of dionine is chiefly observed in the acuter forms. I have had no experience of its use.

Cucaine is not a suitable local anaesthetic as it is too irritating.

I certainly prefer holocaine in 1% solution.

With regard to the use of eserine or pilocarpin, some authors state that its beneficial action is due solely to its effect in contracting the pupil and thereby freeing the filtration angle. Priestley Smith says that where eserine cannot contract the pupil it does more harm than good and in such cases he advises the use of atropine.

In such cases he says that eserine causes hyperaemia. I think that this statement of Priestley Smith is incorrect, at least if he does not refer to an over dose. The effect of eserine or pilocarpin on all unstriped muscle is to cause first, vigorous contraction, and only if pushed does it cause paralysis. This may be seen in its action on the pupil, especially where a 1% solution is applied several times in the 24 hours, even where a fresh solution has been used. I have noticed a certain amount of idiosyncrasy in its effect. On one patient at least, while under treatment for phlyctenular ulcer, marked dilatation of the pupil followed two instillations of a 1% solution of pilocarpin which had been freshly prepared. The same solution did not produce mydriasis in another child into whose eye I instilled one drop.

E. Lilienfeld (16) (Prague) however having observed the same results (mydriasis after using pilocarpin) instituted chemical and physiological experiments on the constitution of pilocarpin salts as found in the market, and discovered an impurity identical with "Jaborin", which, when tested on the frog's heart, paralysed by muscarine, showed that it possessed the action of atropin.

Pilocarpin is said to cause contraction of the ciliary muscle fibres with approximation of the near point (Tweedy)

It is also said to cause diminished sensibility of the retina with consequent impairment of vision. This action would be difficult to determine in cases of glaucoma or ulcer of the cornea.

In small doses pilocarpin checks excessive secretion of sweat. Ringer gives 1/20 of a grain thrice daily in the sweating of phthisis, with good results. Dixon and Brodie found that pilocarpin produced constriction of the bronchioles.
(Journal Physiol. vol xxix No. 2 pp. 97 - 173)

It is perhaps a little curious that while von Graefe, before using iridectomy for glaucoma, noticed its curative effect in ulcer of the cornea, eserine or pilocarpin have now been found to be of great value in the treatment of ulcer of the cornea where there is no fear of adhesion from iritis. In phlyctenular ulcer I have seen the most astonishing results from the use of eserine. I have often seen the congestion accompanying the ulcer entirely abolished in from 24 to 48 hours.

That does not look as if eserine or pilocarpin produced hyperaemia. Pilocarpin has also a slight narcotic action.

In summing up my opinion on the use of eserine or pilocarpin in Glaucoma, I would say that its beneficial action is due to—I. its power of constricting the small arterioles, thus lessening the congestion and pain, the latter effect being assisted to a slight degree by its narcotic effect. II. diminishing the excessive secretion from the ciliary body and uveal tract generally. III. its power of contracting the iris, and so, by thinning it out, removing an obstacle to the outlet of fluid through the filtration angle; and by increasing the surface of the iris and lessening the congestion of its vessels, restoring the absorptive function which the anterior surface of the iris exerts on the aqueous humour, as demonstrated by Nuel and Benoit (of Liege), and Asayama (of Tokio, Japan)

P. S. (Dr. Karl Grossmann, at the meeting of the British Medical Association 1903, described a case of aniridia in which he had observed the action of eserine on the lens. He found that it caused bulging of the anterior and posterior surfaces with diminution of the equatorial diameter.

While he gave no explanation of the modus operandi, I think such an occurrence would result from the stimulation of the ciliary muscle fibres and would correspond to the act of accommodation without there being necessarily any direct effect of eserine on the lens itself. His patient had about 2.5 D. of accommodation.)

In summing up my views on the causation of Primary Glaucoma, deduced from theevidence brought forward by the various authorities I have quoted,and by what I have seen of the disease,I would say,that while little has actually been proved, much remains to be disproved.

Even in view of what has been brought forward in support of the blocking of the filtration angle, I am of opinion that it is still too premature to throw the theories of hypersecretion out of court.

Indeed, I think that in most instances of primary glaucoma,hypersecretion of intraocular fluid is a very early manifestation of the glaucomatous process,and that the bulging forward of the lens and iris is a direct result of the excessive secretion into the posterior chamber. I also think that in most instances such hypersecretion is of

inflammatory origin,but there is evidence also, both physiological and clinical, that such hypersecretion may be the result of nervous influence. Hypersecretion of the normal products of the body is seen as a result of inflammatory action, a common example being hypersecretion of mucous from the various tracts lined by that membrane; the occurrence of oedema around an inflamed area might also be cited.

Admitting then that inflammation may cause hypersecretion, is there any evidence that inflammation occurs during glaucoma and more especially as a very early phase in that disease?

Briefly, I would point out that the vitreous is sometimes found to be more membranous and coagulable. This is comparable to what we find in ascitic and pleuritic fluid the result of inflammation of these membranes,as opposed to purely circulatory disturbance in them.

Gasparinni's cases were preceded by distinct neuritis. My own observations on the similarity between glaucoma and sympathetic ophthalmia are also, I think, in favour of inflammatory action.

Brailey found inflammation of the ciliary body,iris, and optic nerve always present,and was of the opinion that

it was developed previous to increased tension. In cyclitis, glaucoma is found to occur in the serous form of the disease, that is, where there is undoubtedly an inflammatory hypersecretion.

In support of the nervous hypersecretion, we find hypersecretion of urine, saliva, and sweat under abnormal nervous conditions; and under abnormal mental conditions (emotional disturbance), we find hypersecretion of urine, as in hysterical paroxysms, and diarrhoea occurring in terror.

Sympathetic vaso-motor and vaso-inhibitory fibres have been demonstrated, and a sympathetic double nerve supply has been traced to various secreting glands, amongst which is the lachrymal gland, it is almost certain that the secreting apparatus of the intraocular fluid is also under the sympathetic nervous system to some extent.

Clinically, grief, worry and anxiety, and even joy have been clearly associated with the attacks of primary glaucoma in susceptible individuals.

I have noted the emotional and other nervous temperaments among my cases and especially among the congestive type.

And, finally, the effects of sympathicotomy and sympathectomy already noted cannot be ignored and tend to

prove that in some cases at any rate, the sympathetic nervous system plays a part in the causation of glaucoma.

Turning to the theory of obstruction to the exit of the intraocular fluids we have clear demonstrable proof that such obstruction does occur and more especially at the main outlet, the filtration angle, but the time at which this obstruction takes place is not yet proved.

It is difficult to see how the iris can be pushed forward by the ciliary processes and lens if the filtration angle be unable to permit of the exit of the fluid in the anterior chamber unless the fluid becomes absorbed by the anterior surface of the iris or finds its way through the cornea.

That the blocking of the corneo-iritic angle is a most serious event in the course of glaucoma, is admitted by all, but I think it is not the fons et origo mali.

The results of iridectomy and sclerotomy are in my opinion too variable to permit one to hold the opinion that the blocking of the corneo-iritic angle is the cause in all cases; for, while in many cases they result in cure of the disease, there is still a large number in which the operation is a total failure. The failure of the operation of iridectomy, especially in the more chronic cases, is not because of the iris being more closely adherent to the

cornea, for it has been demonstrated that the more chronic the disease the less adherent is the iris base to the sclero-cornea. It is not because the iris is more difficult in the majority of cases to excise, for it is only in a comparatively small number that the iris is found to be "rotten" and to tear in the grasp of the forceps applied as lightly as possible. The general conditions in chronic glaucoma are much more favourable to an accurate and careful excision of a portion of the iris, owing to the operation being painless, while in acute, unless a general anaesthetic is given, the operation is always painful, the worst pain being experienced at the most critical moment.

The failure, in my opinion is due to the operation having no direct effect on the cause, which, even excluding a hypersecretion, may still be due to blockage of the posterior routes of exit of the intraocular fluids. Such blockage has already been referred to at page ~~22~~ 14.

To refer again to the case of J.F. page 41; from the commencement of the symptoms of glaucoma of a chronic congestive type in the left eye, 4½ years sufficed in spite of an iridectomy to render the eye blind; while in the right eye, three months had reduced the vision to $\frac{20}{50}$, and during the next five months in spite of <u>four</u> sclerotomies and an iridectomy the central vision was reduced to $\frac{20}{70}$ and

the field of vision had become restricted to such an extent as to make walking about a dangerous and difficult task.

In view of the fact that neuritis is observed as an early symptom of glaucoma, I think it is worthy of note that optic nerve atrophy is the cause of the blindness in the last stages of glaucoma.

I have also been struck by the fact that although rheumatism and gout have been considered at various times to be etiological factors, I have never seen syphilis advocated as a cause nor have I seen potassium iodide recommended in the treatment. Possibly this is due to the early treatment by mercurials having had a negative result, but I am of opinion that in those cases where the cardio-vascular system is manifestly abnormal, potassium iodide ought to have a trial, apart from any idea of syphilis.

Finally, I feel that, just as in ascites, we have a collection of fluid from several totally different causes, requiring for its treatment different methods aimed at the different causes, so in glaucoma we have an increase of intraocular fluid from various causes and so long as we attempt to treat the common result by a method applicable to only one or two of the causes, so long shall we find that a portion only of our cases respond to such treatment.

That iridectomy is a mode of treatment which has been of incalculable benefit to that section of mankind afflicted with glaucoma, is undoubted, and the purport of my remarks is in no way intended to detract one whit from its value; nevertheless, I feel that anything tending to too complete satisfaction with its results would put back the time when, by a more definite grasp of the various causes, we shall be able to apply truly curative methods to all forms of glaucoma, even if it be too much to hope that preventive measures will be discovered which will reduce to a minimum the need for curative treatment.

[illegible] Dec. 1900.

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    (b) Ophthal. Review, 1887.
    (c) Pathology and Treatment of Glaucoma, 1891.
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A.

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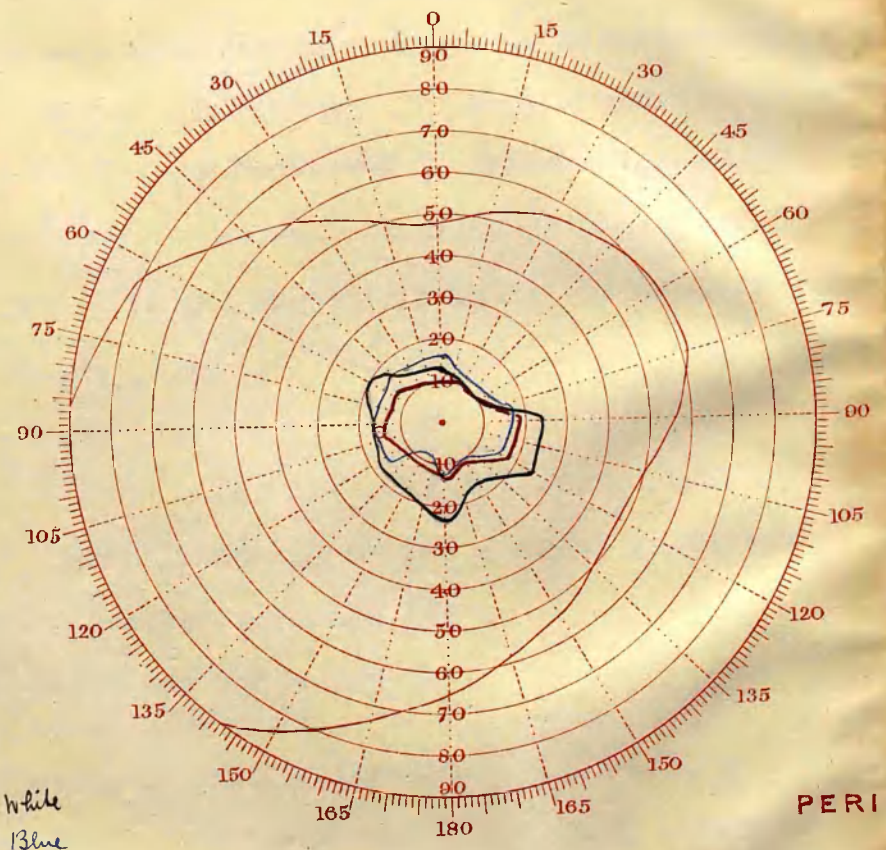
LEFT.



PERIMETE

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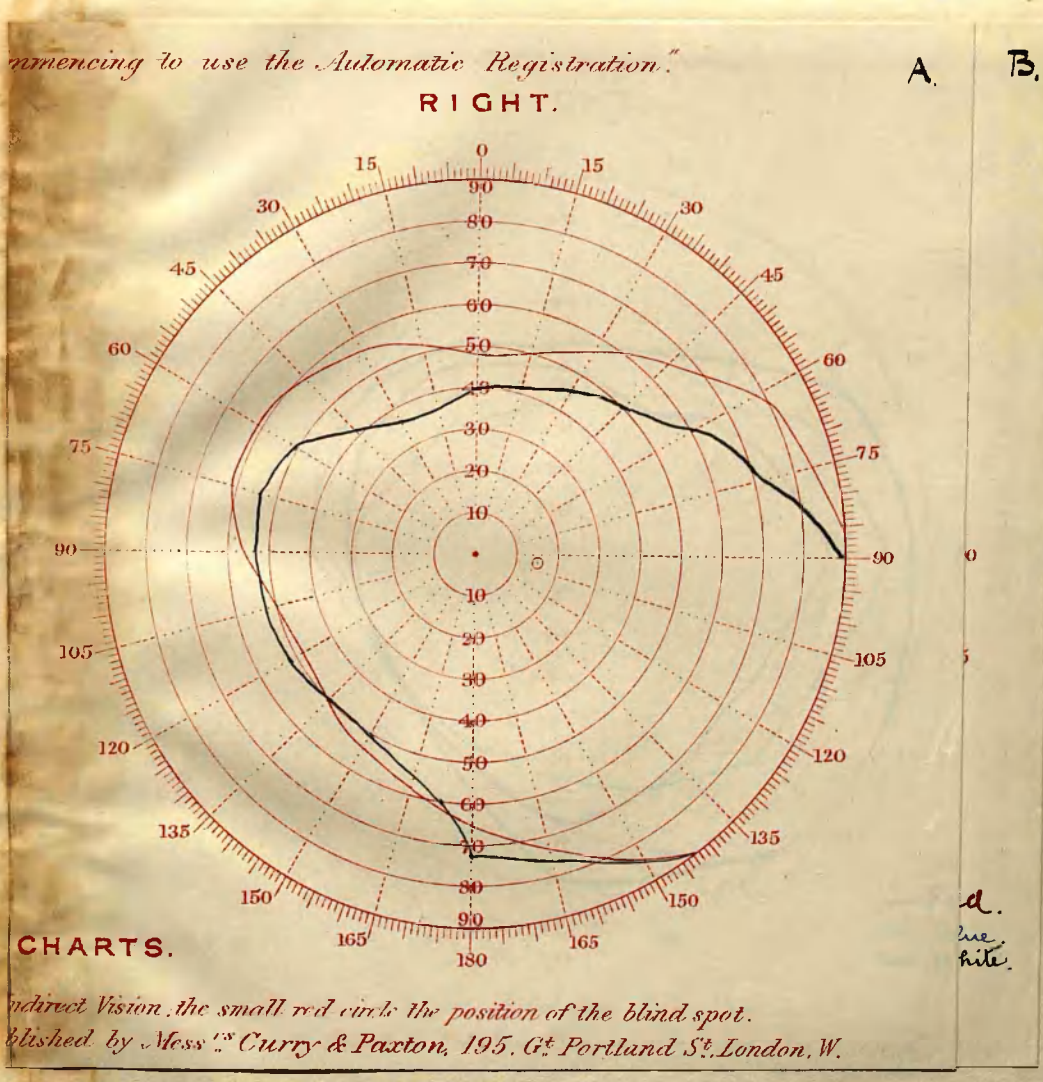

D.McI. A. Before operation.

B. After enucleation of left eye.

For details of case see p. 34 et seq.

B.

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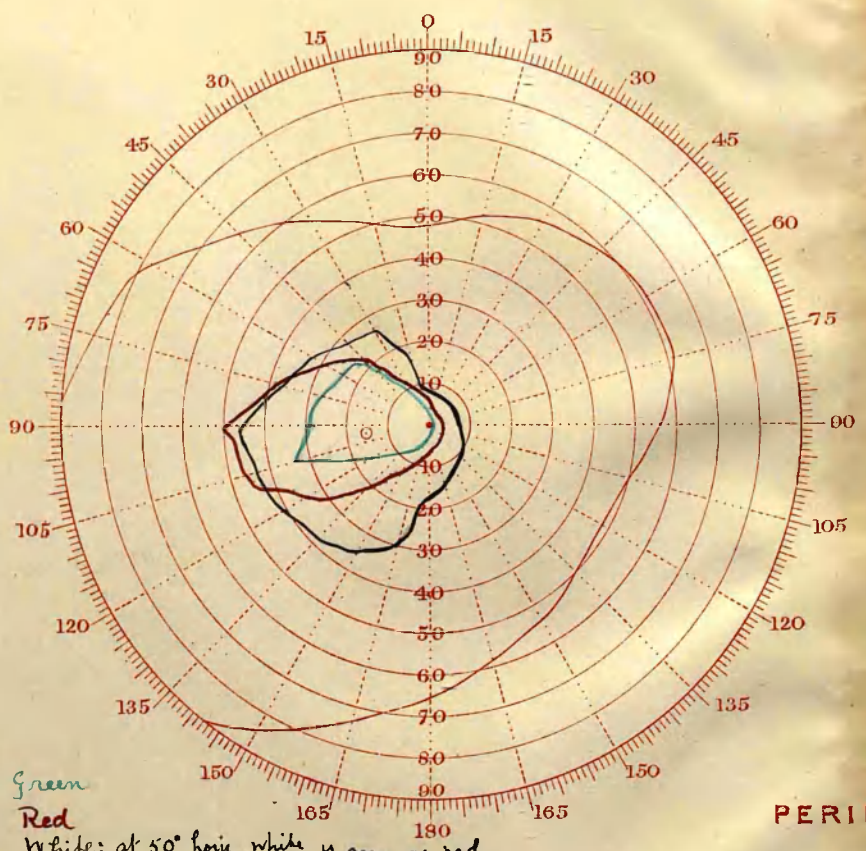

— Green
— Red
— White; at 50° long. white is seen as red.

PERIMETE

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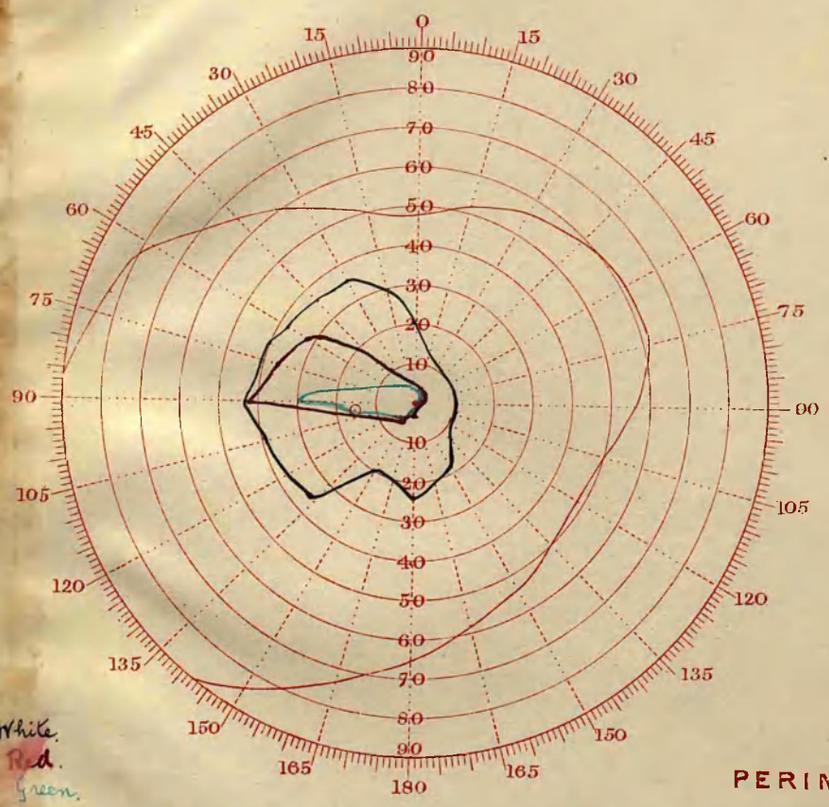

PERIMETER CHARTS.

The eccentric continuous red line indicates the average normal Field of Indirect Vision, the small red circle the position of the blind spot.

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A.

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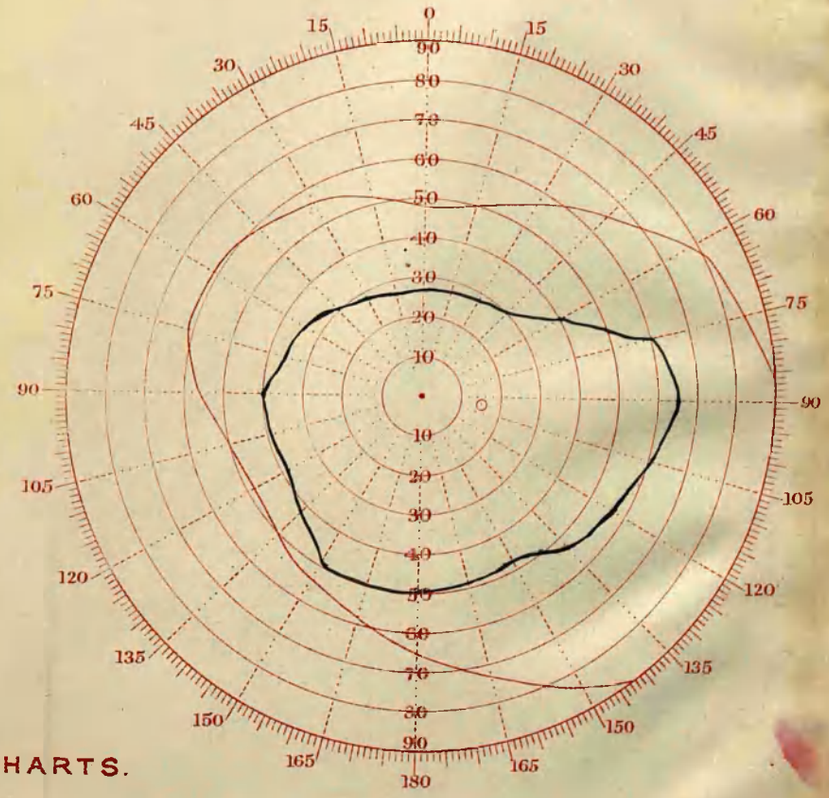

H.Mc.K. Chronic Glaucoma.

Note marked contraction of V.F. of right eye associated with V.A. = $\frac{6}{6}$

A. Before operation (sclerotomy). B. After operation, three weeks.

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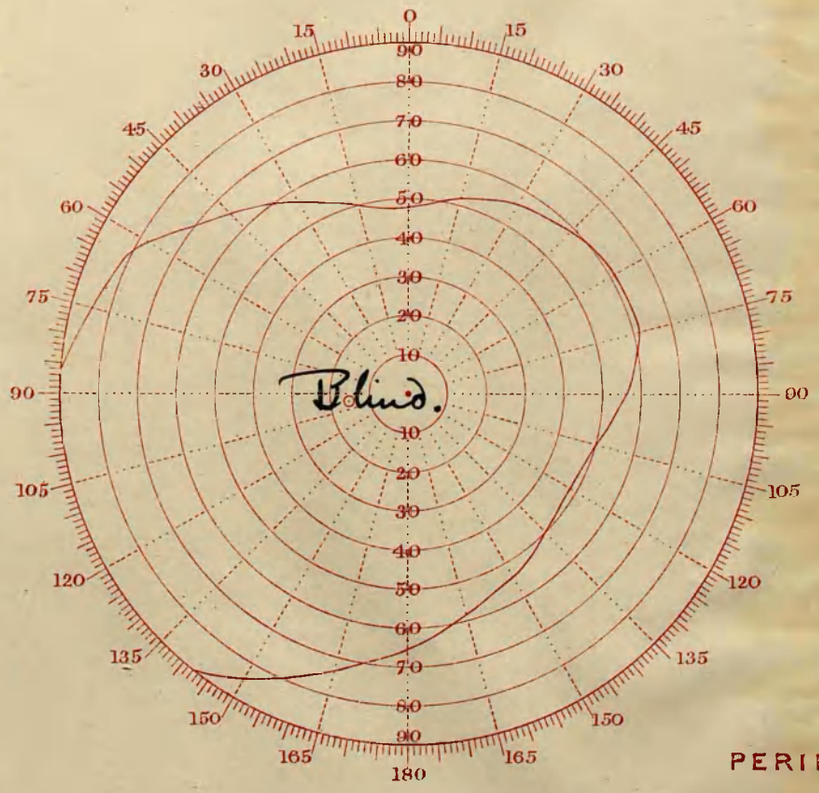

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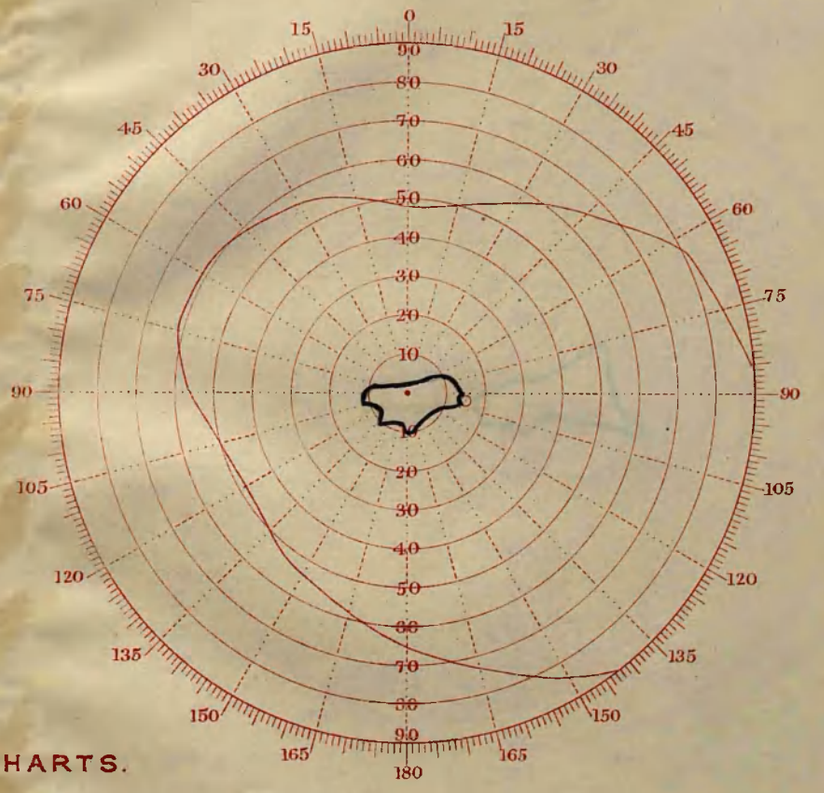

PERIMETER CHARTS.

The eccentric continuous red line indicates the average normal Field of Indirect Vision, the small red circle the position of the blind spot.

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J. F. A. before 4th. operation. V.A. = $\frac{20}{100}$.

B. after 5th. operation. V.A. = $\frac{20}{70}$

For details of case see page 41 et seq.

commencing to use the Automatic Registration."

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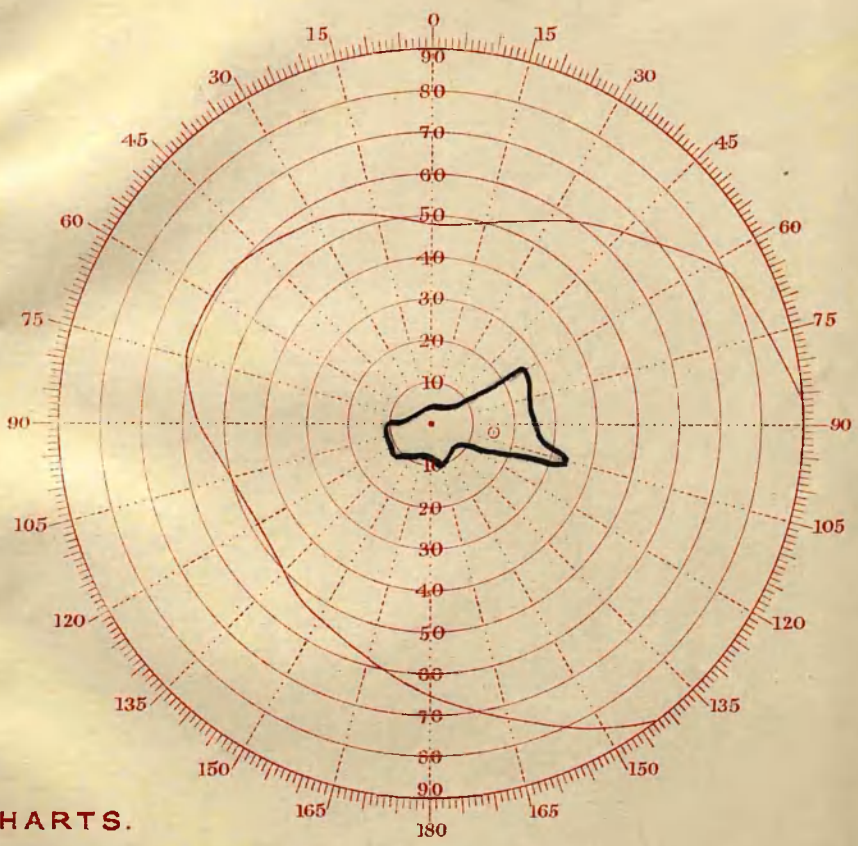
B



R CHARTS.

of Indirect Vision, the small red circle the position of the blind spot.

Published by Mess^rs. Curry & Paxton, 195, G^t. Portland S^t. London, W.

J. F. A. before 4th. operation. V.A. = $\frac{20}{100}$.

B. after 5th. operation. V.A. = $\frac{20}{70}$

For details of case see page 41 et seq.

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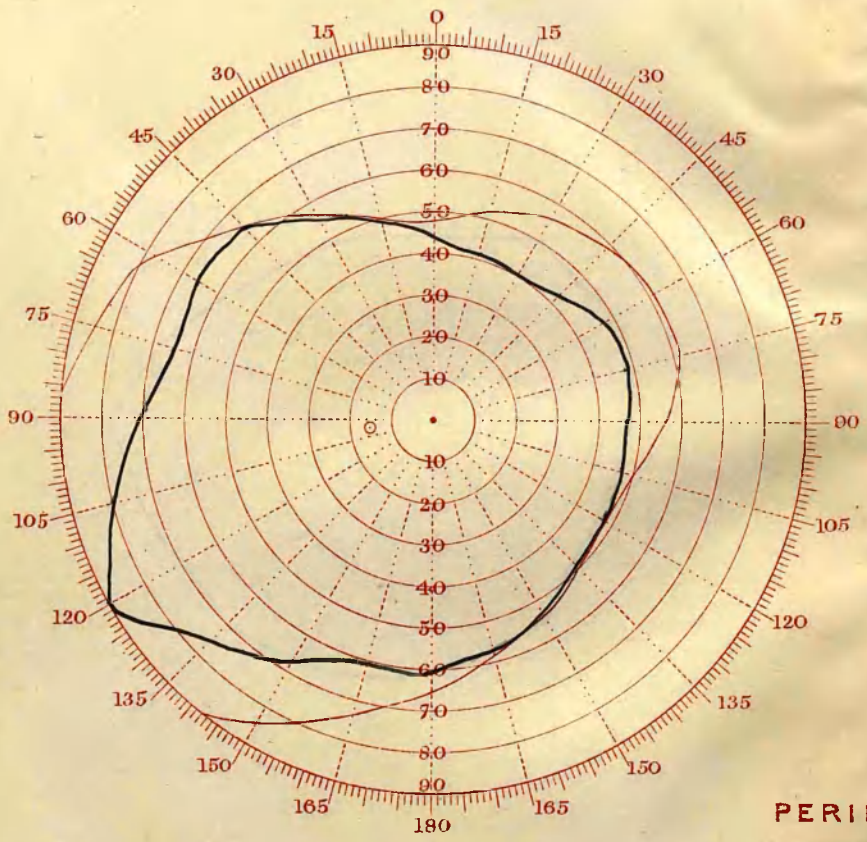

PERIMETE

The eccentric continuous red line indicates the average normal Field

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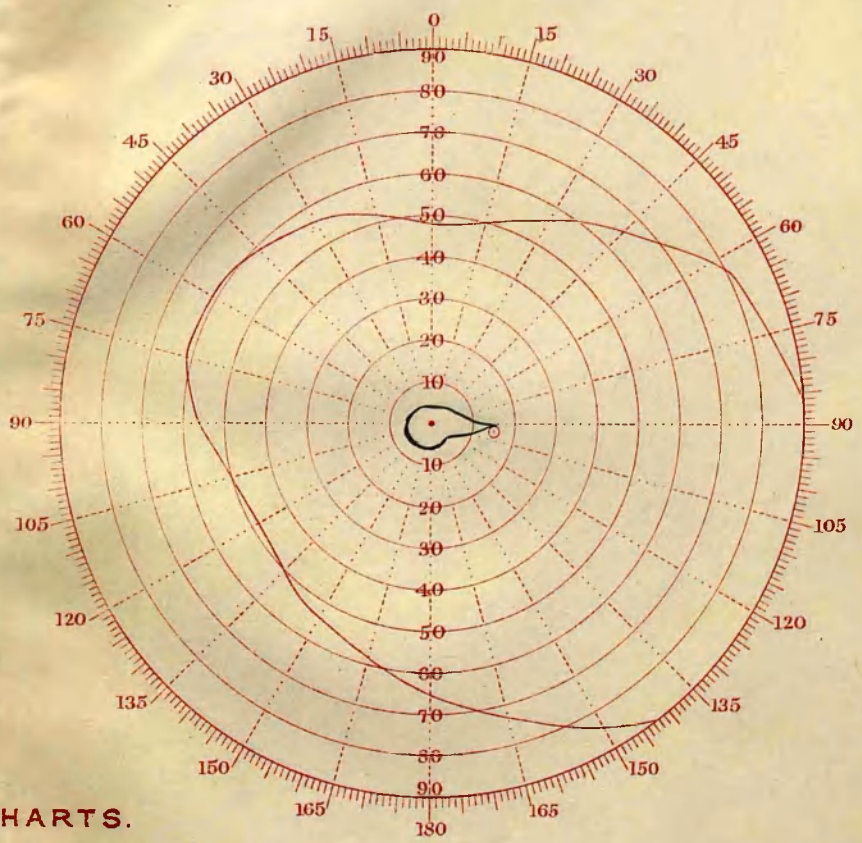

R CHARTS.

of Indirect Vision: the small red circle the position of the blind spot.

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C. F. A. Before operation. V.A.L. = $\frac{20.}{40}$ V.A.R. = fingers

B. After operation on right (one month)

For details of case see page also page

B.

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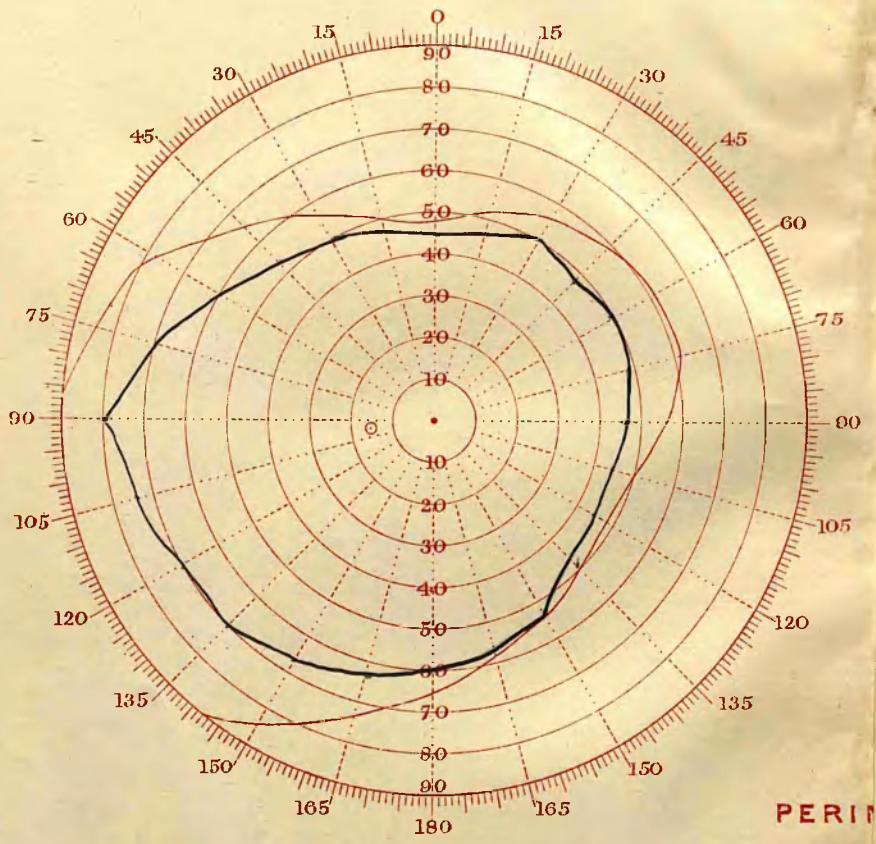

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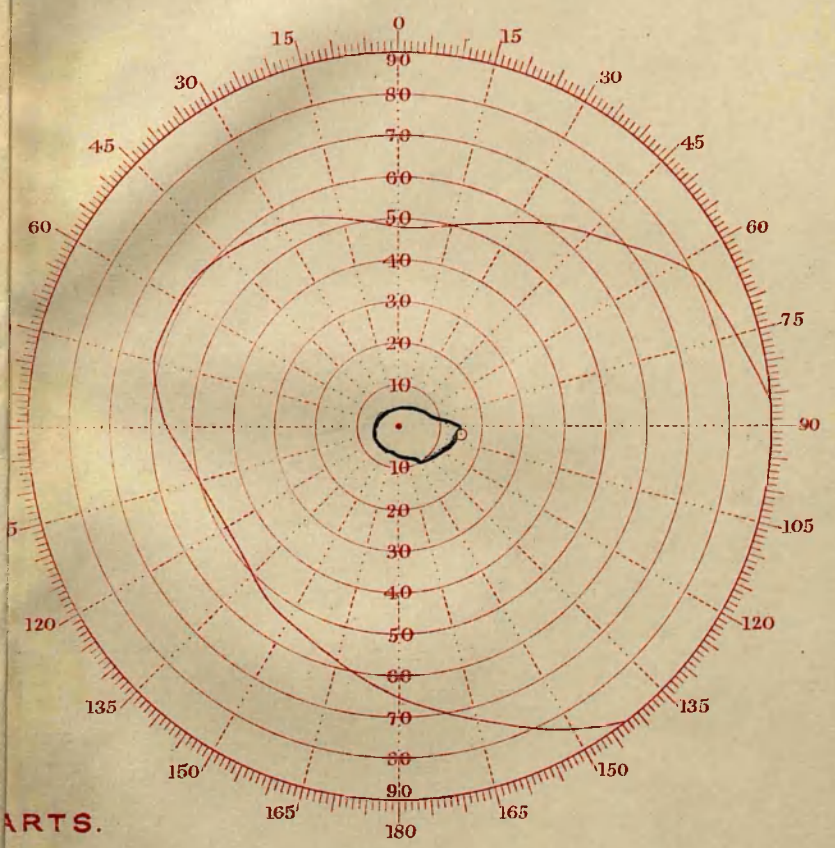

ARTS.

Vision: the small red circle the position of the blind spot.

d by Mess.rs Curry & Paxton, 195, Gt Portland St. London, W.

C. F. A. Before operation. V.A.L. = $\frac{20}{40}$ V.A.R. = fingers

B. After operation on right (one month)

For details of case see page also page

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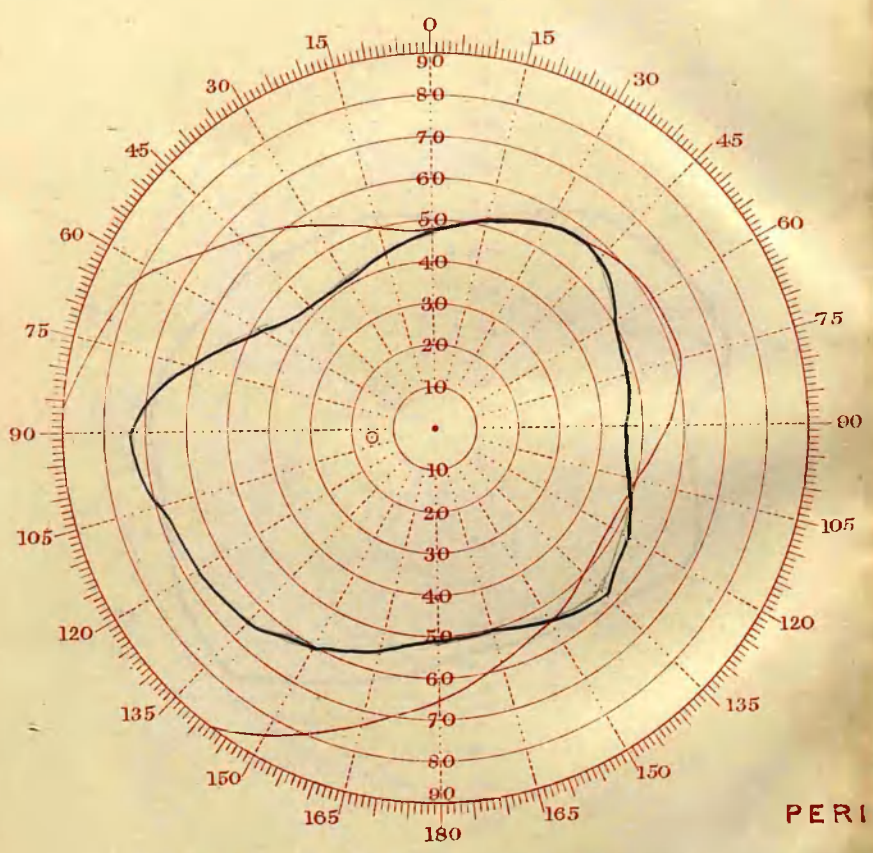

PERINETER

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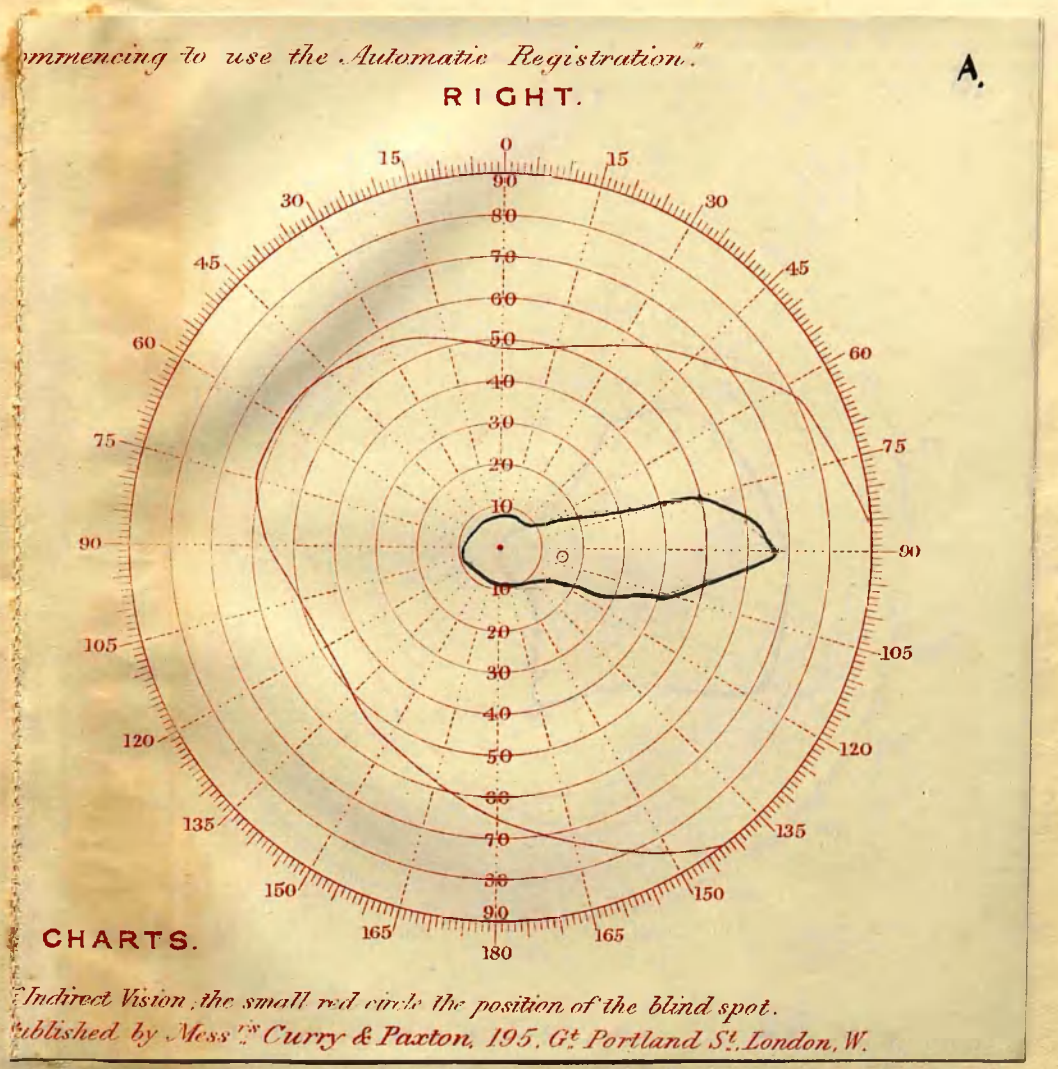
Designed for use with Prof: Mc Hardy's Registering Perimeter.



J. H. A. Before operation on either eye. V.A.R. $\frac{20}{80}$

V.A.L. $\frac{20}{80}$.

B. One week after operation on right.

C. 18 days after operation on right.

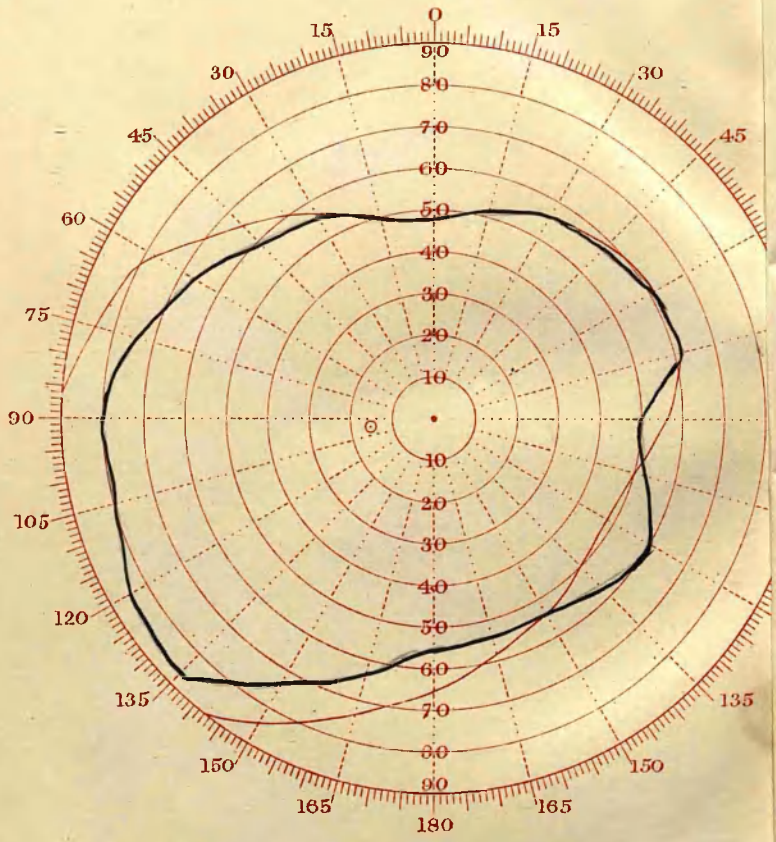
C^1. 10 days after operation on left.

See page

C

"Centre each chart with "pointer" a

LEFT.



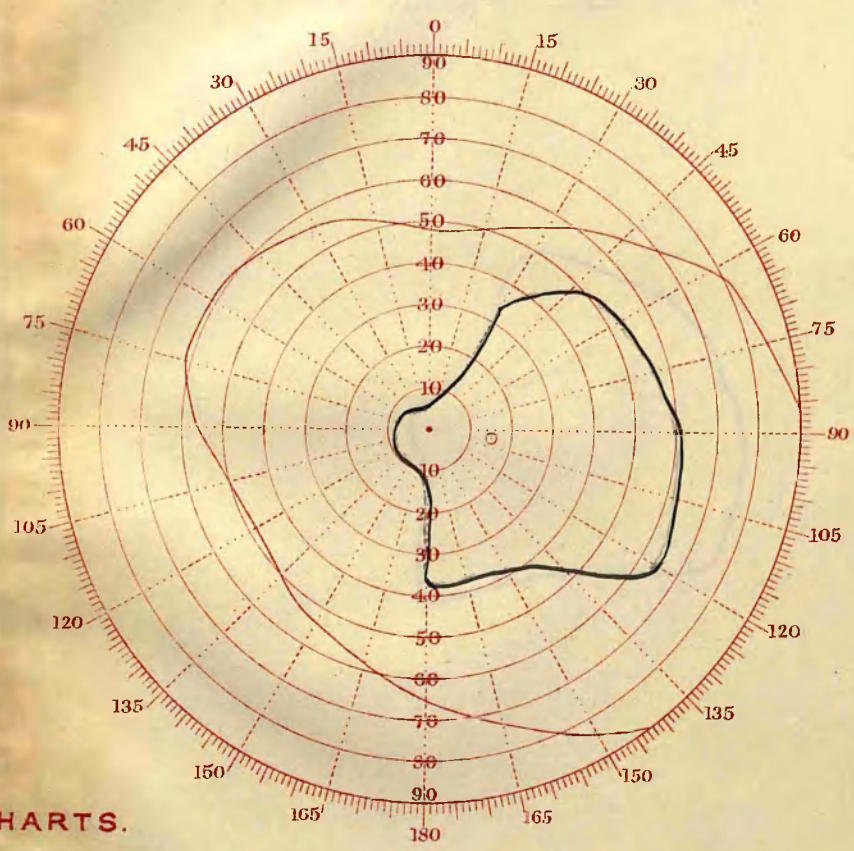
The eccentric continuous red line indicates the aver

Designed for use with Prof: Mc Hardy's Registering P

mencing to use the Automatic Registration".

RIGHT.

B.



CHARTS.

direct Vision the small red circle the position of the blind spot.

ished by Mess^rs Curry & Paxton, 195, G^t Portland St. London, W.

J. H. A. Before operation on either eye. V.A.R. $\frac{20}{80}$

V.A.L. $\frac{20}{80}$.

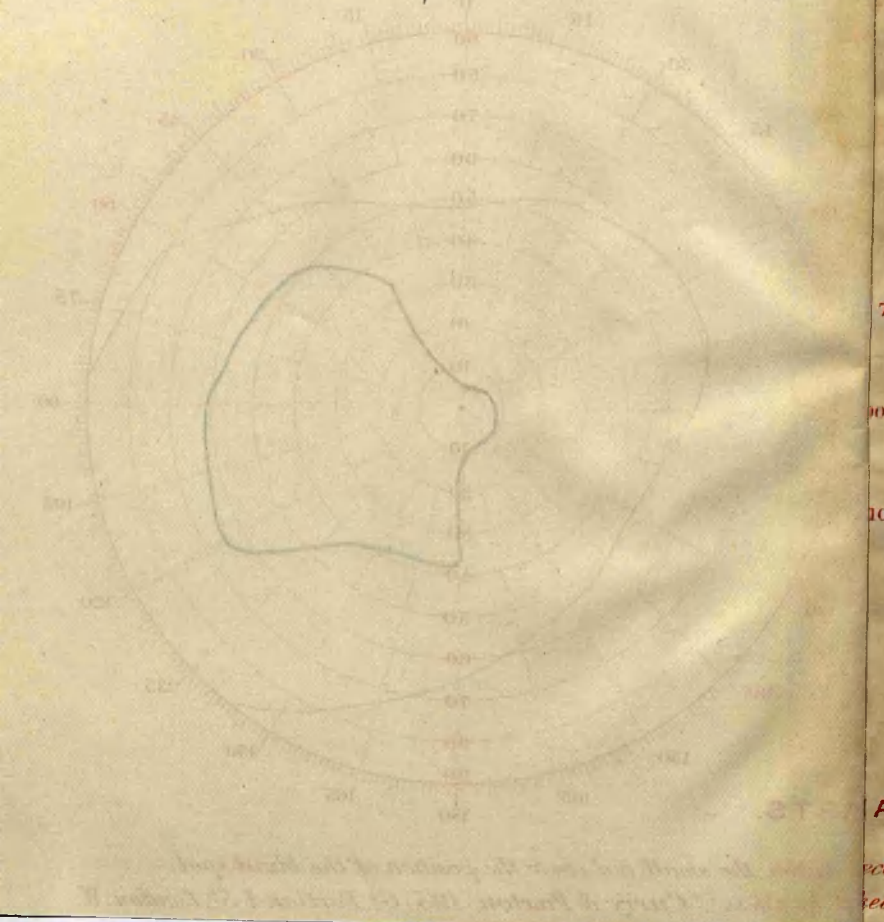
B. One week after operation on right.

C. 18 days after operation on right.

C[1]. 10 days after operation on left.

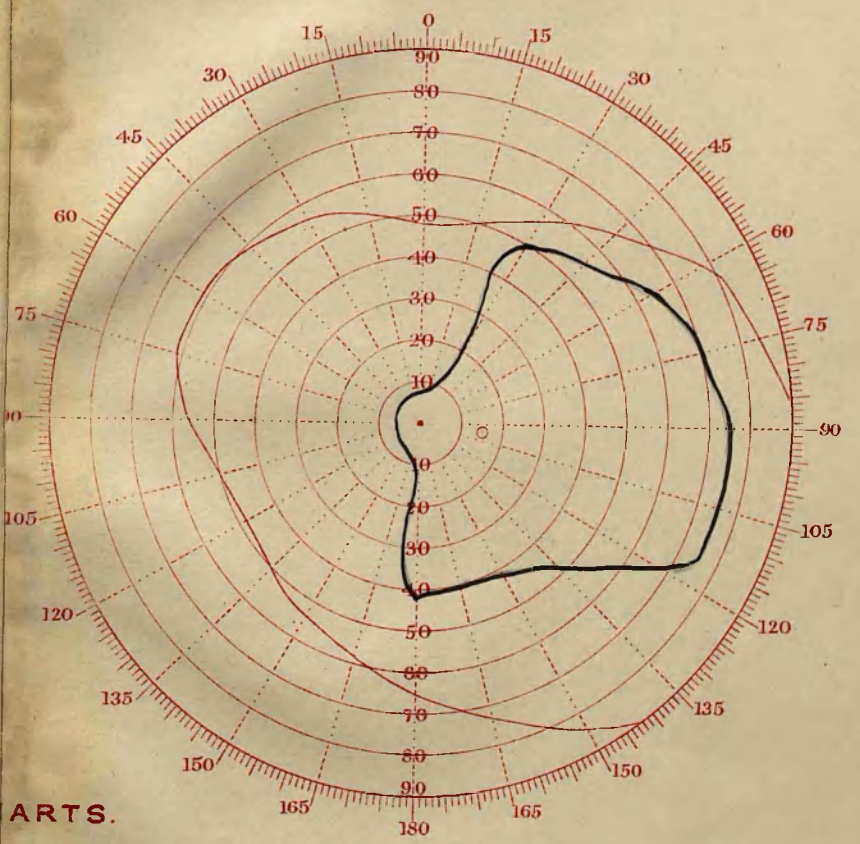
See page

James Haldane. 19-3-00.
One wk after op in R.



ncing to use the Automatic Registration." C

RIGHT.



ARTS.

ct Vision: the small red circle the position of the blind spot.
ed by Mess.rs Curry & Paxton, 195. Gt Portland St. London, W.

J. H. A. Before operation on either eye. V.A.R. $\frac{20}{80}$

V.A.L. $\frac{20}{80}$.

B. One week after operation on right.

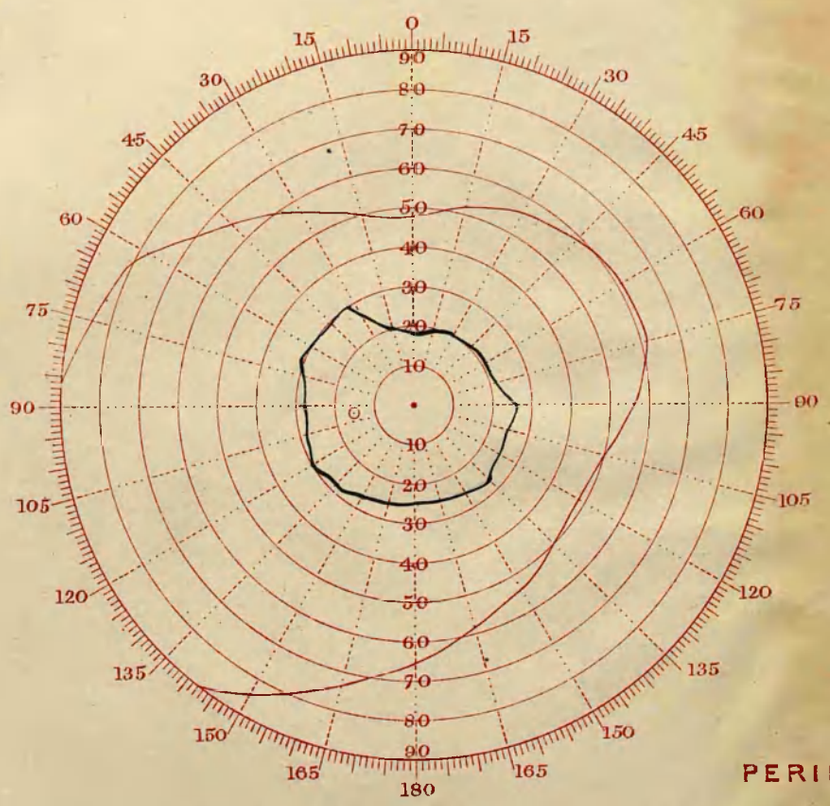
C. 18 days after operation on right.

C^1. 10 days after operation on left.

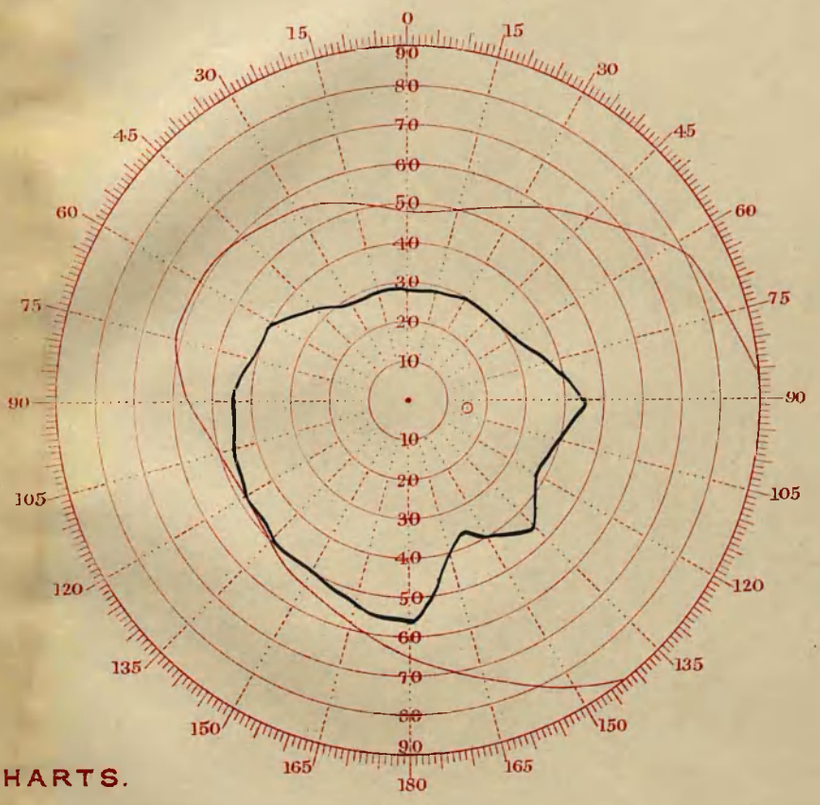
See page

"Centre each chart with "pointer" at Zero before commencing to use the Automatic Registration."

LEFT.



RIGHT.



PERIMETER CHARTS.

The eccentric continuous red line indicates the average normal Field of Indirect Vision, the small red circle the position of the blind spot.

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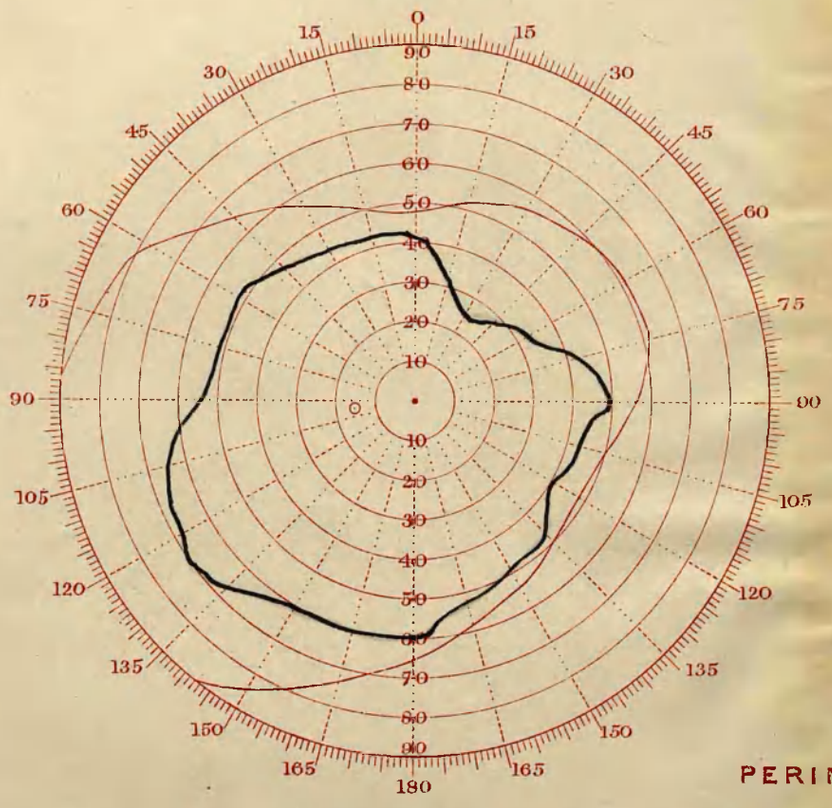
L. G. Sub acute attack of Glaucoma in left eye.

V.A.L. = $\frac{6}{36}$ J 20 (?) V.A.R. $\frac{6}{24}$ J. 16.

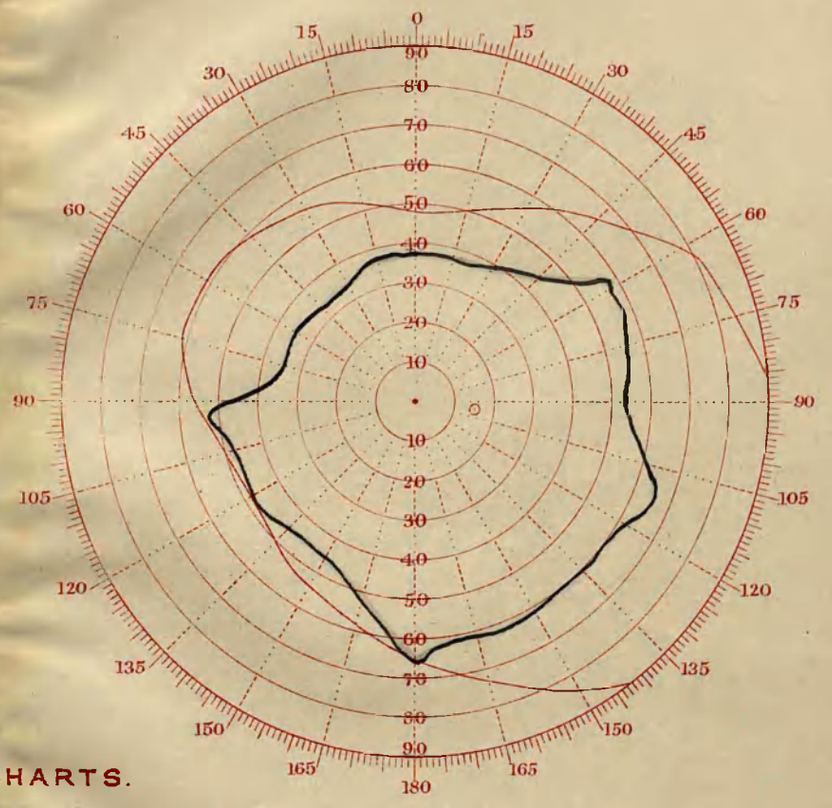
T in left + 2. T in right = n.

"Centre each chart with "pointer" at Zero before commencing to use the Automatic Registration."

LEFT.



RIGHT.



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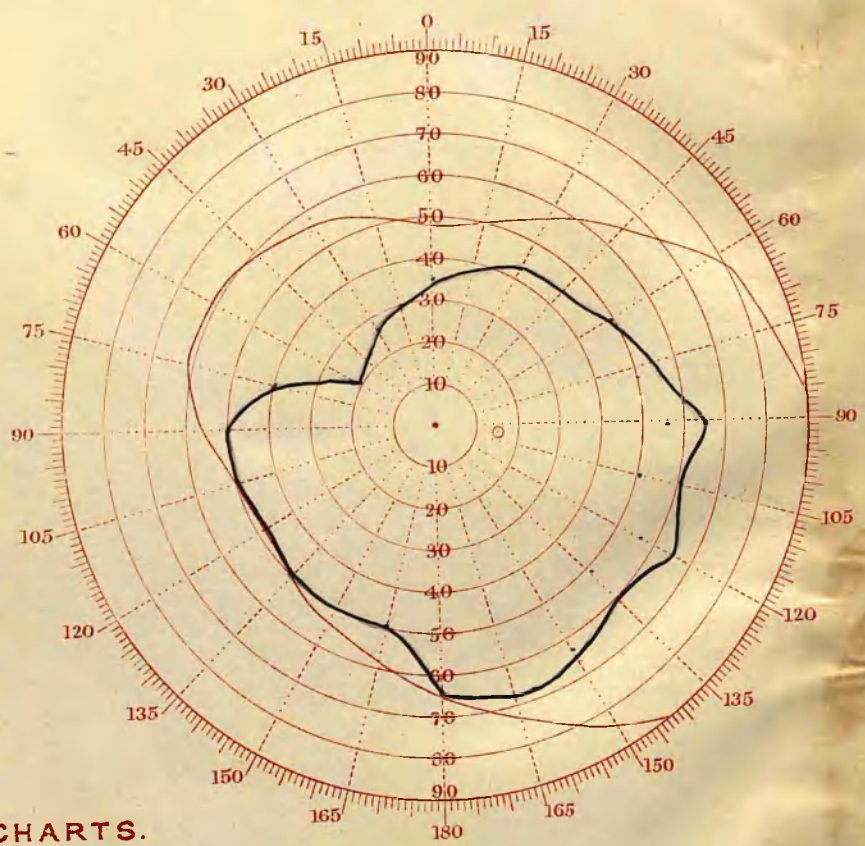
D. S. (See page 44 for report).

B. Charts taken 13 days after iridectomy on left eye.

commencing to use the Automatic Registration."

A.

RIGHT.



R CHARTS.

d of Indirect Vision; the small red circle the position of the blind spot.

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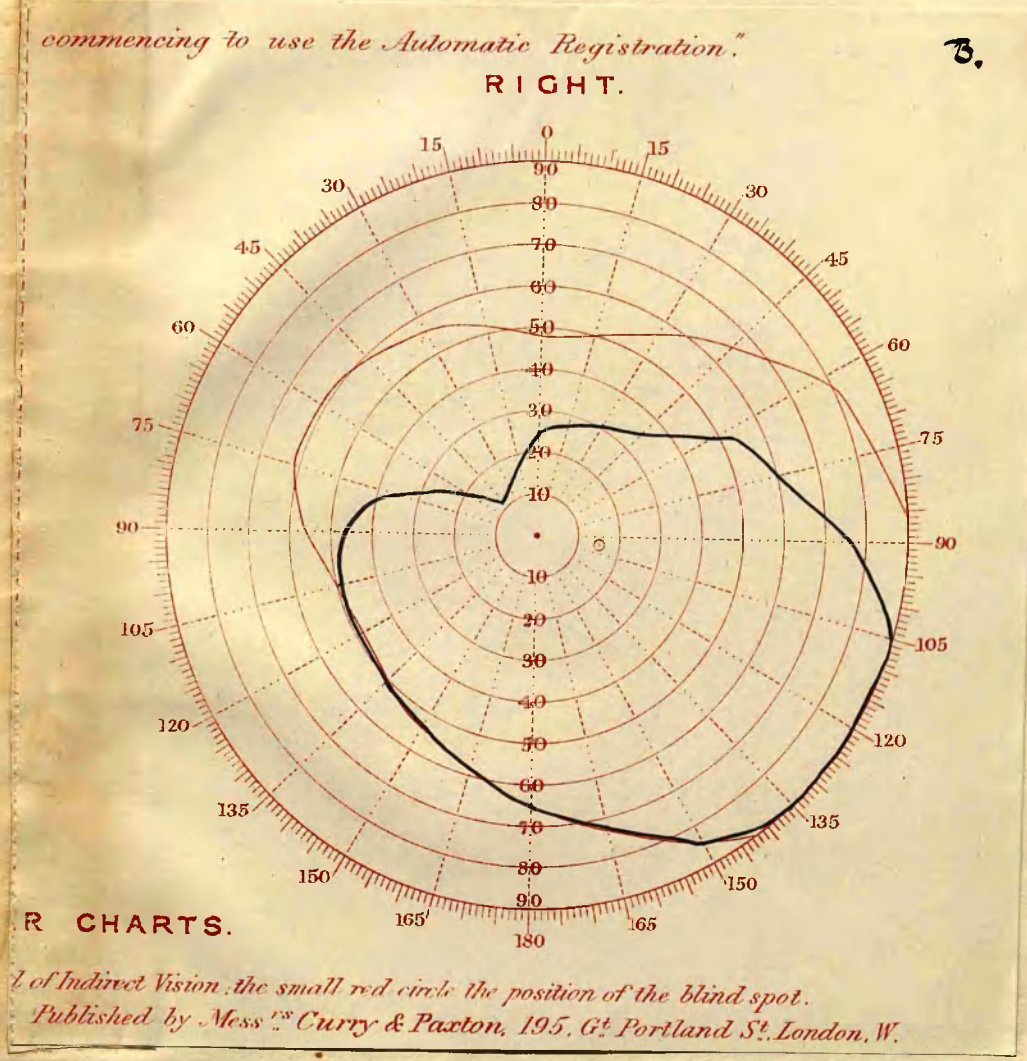


W.P. A. V.A.R. $\frac{20}{200}$ Chart taken after 1st. sclerotomy.

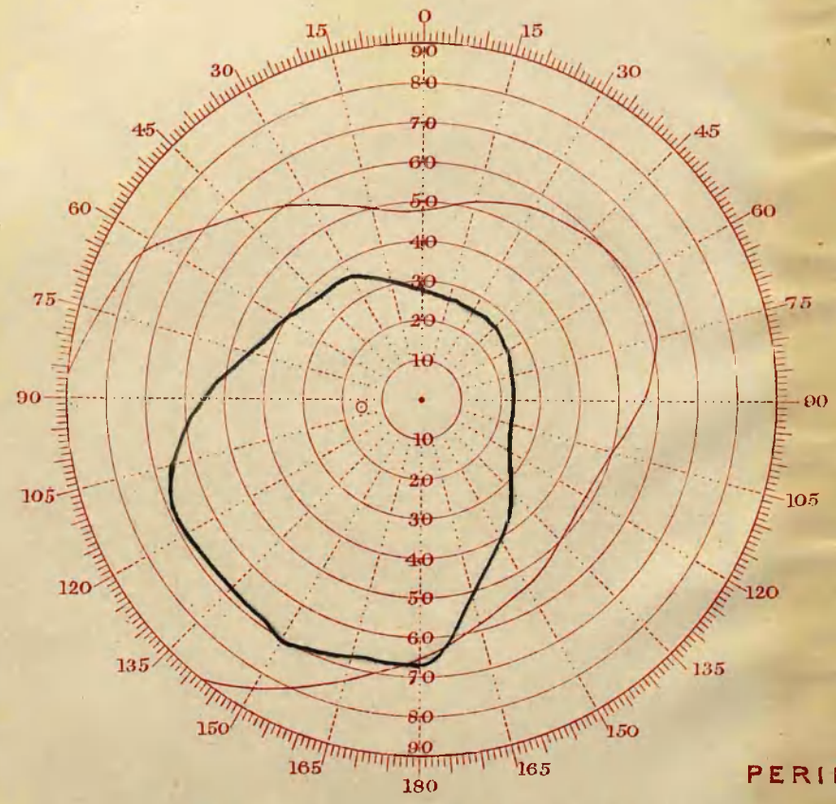
B. V.A.R. $\frac{10}{200}$ Charted 14 days after 2nd. sclerotomy and 2½ months after A.

For report, see pages

A

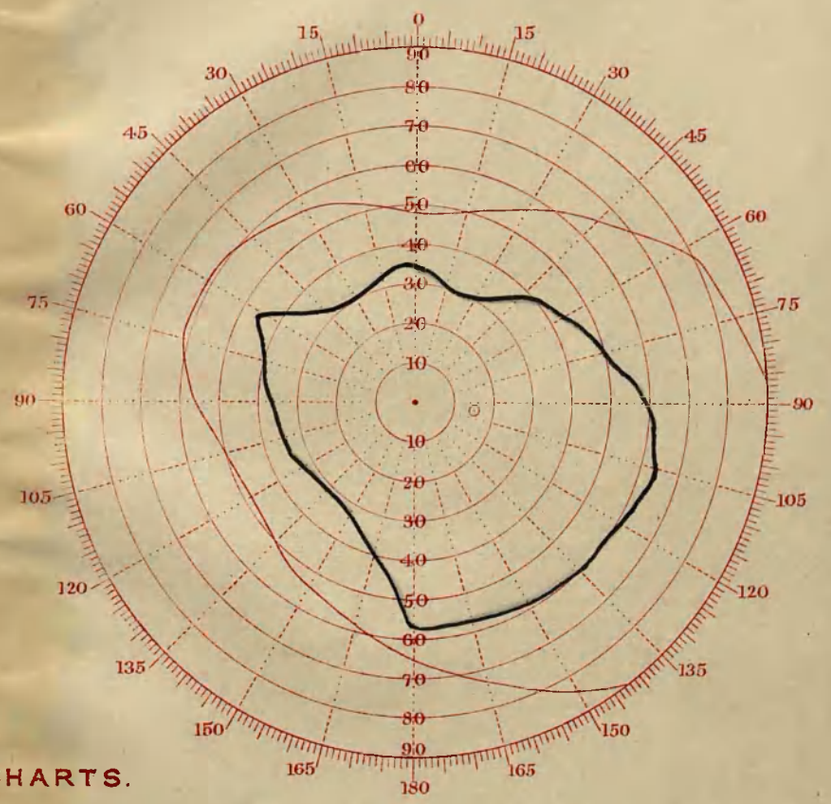
"Centre each chart with "pointer" at Zero before commencing to use the Automatic Registration."

LEFT.



RIGHT.

A



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H. C. V.A.R. $= \frac{10}{200}$ V.A.L. $= \frac{8}{200}$

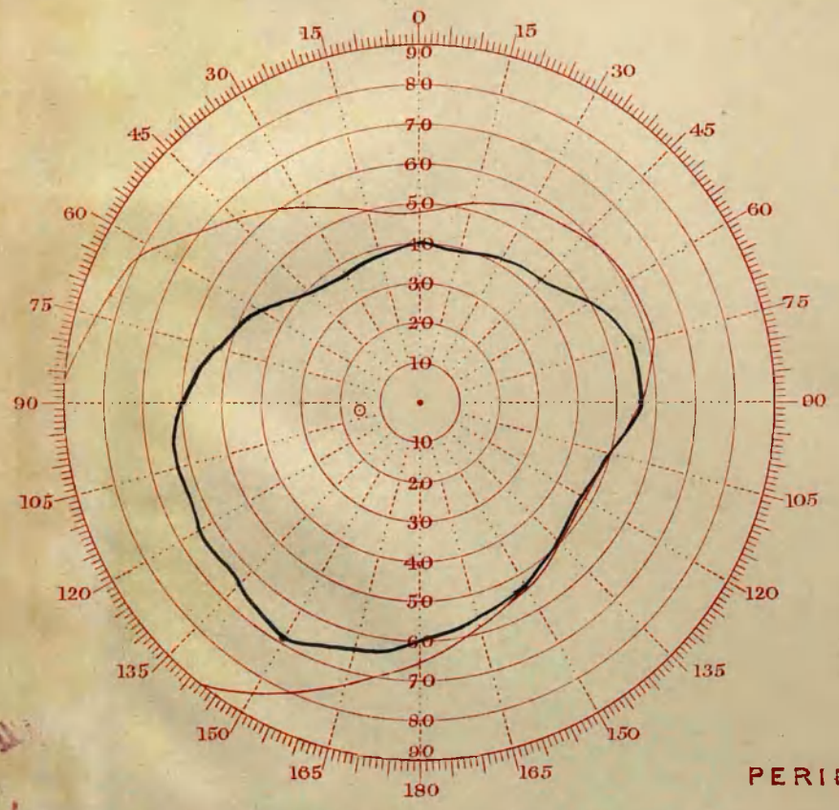
T in both eyes over + 1.

Marked cupping of both discs.

"Centre each chart with "pointer" at Zero before commencing to use the Automatic Registration."

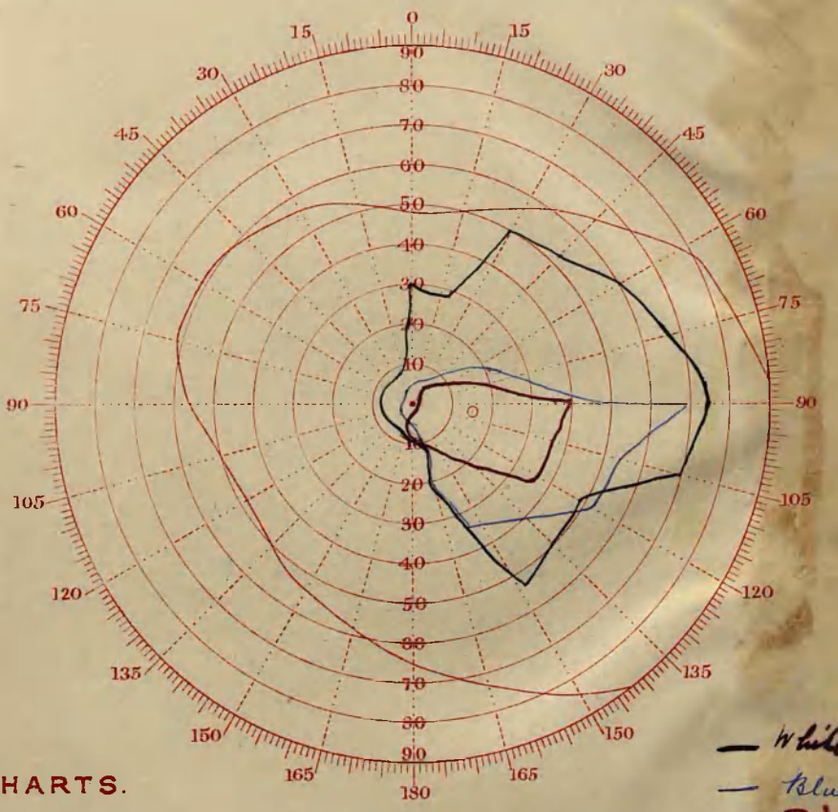
A.

LEFT.



A.

RIGHT.



White
Blue
Red.

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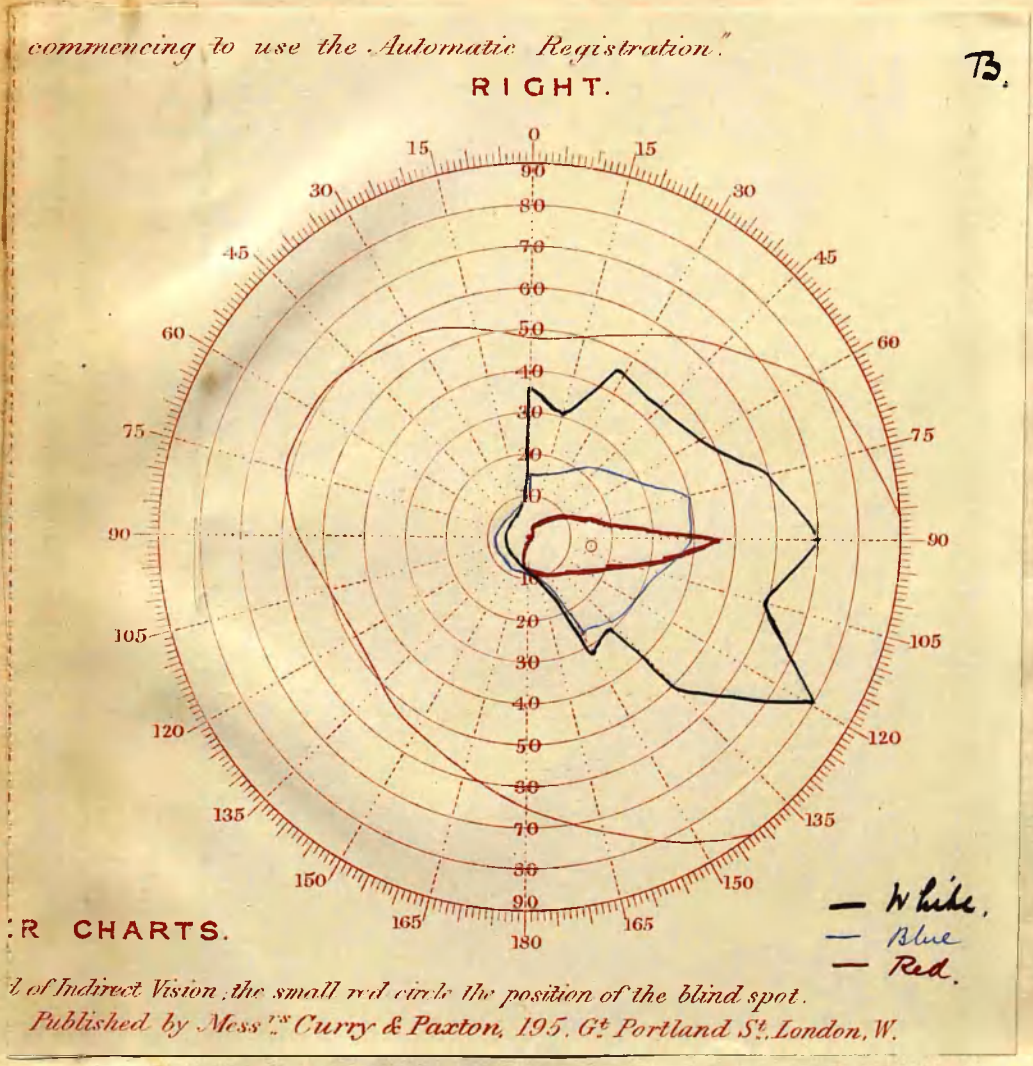


R. F. V.A.R. = L and S. V.A.L. = $\frac{15}{200}$ before operation A.

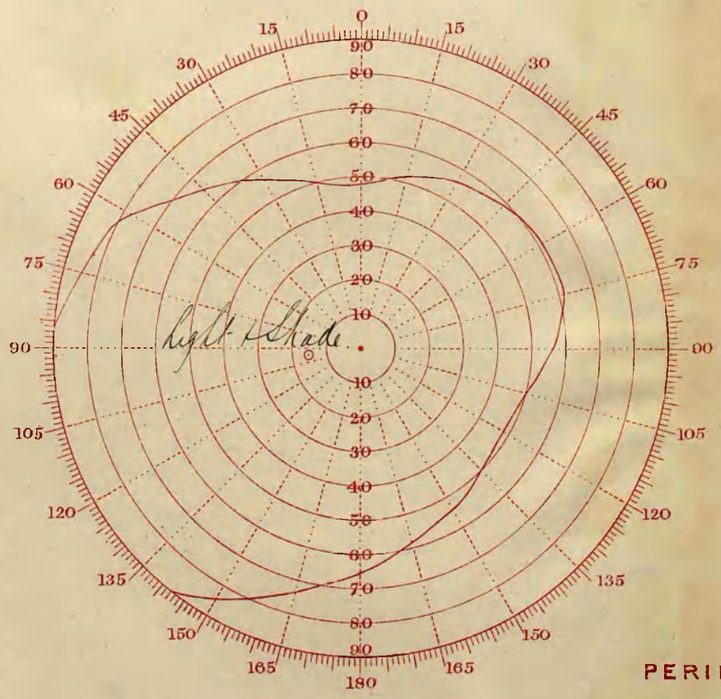
B. V.F. 12 days after operation.

Case of chronic glaucoma. Dimness of vision 12 years. T in left n. T in right + .

A

"Centre each chart with "pointer" at Zero before commencing to use the Automatic Registration".

LEFT.



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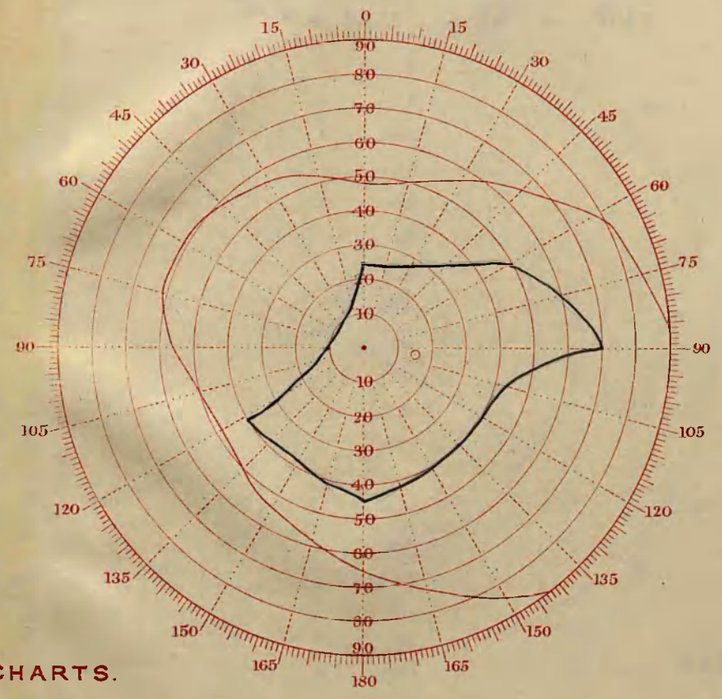
Designed for use with Prof.r Mc. Hardy's Registering Perimeter.

A

"Centre each chart with "pointer" at Zero before commencing to use the Automatic Registration".

RIGHT.



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B.

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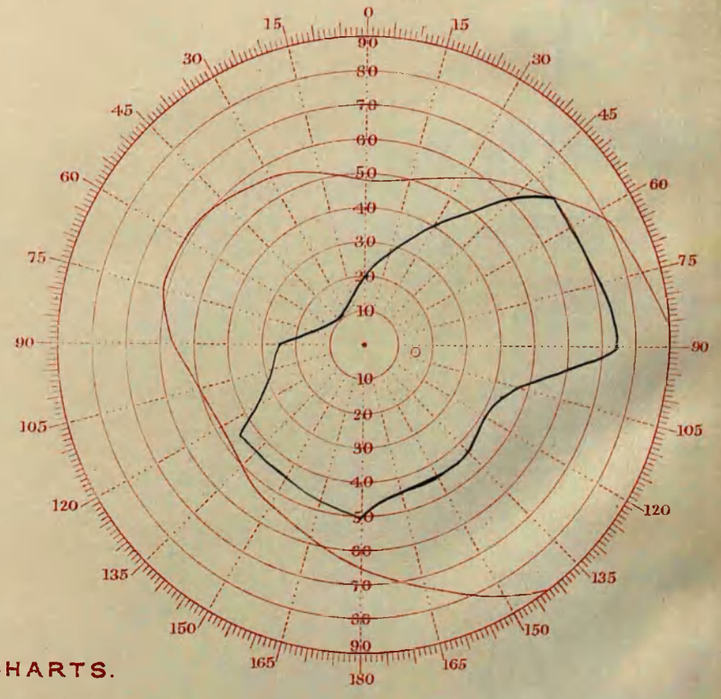
RIGHT.



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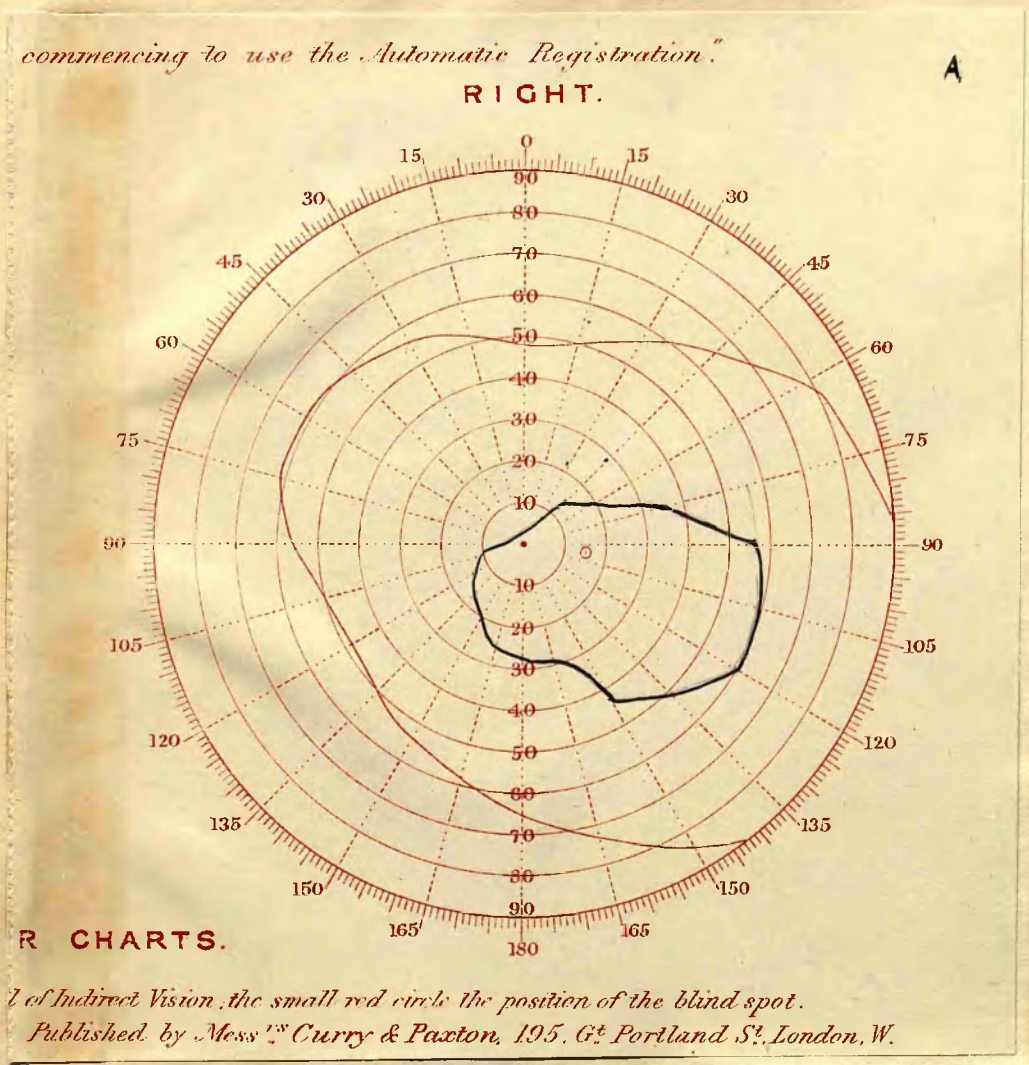
L. M. A. V.A.L. = L and S. V.A.R. $= \frac{20}{100}$

B. 16 days after sclerotomy. V.A. $\frac{20}{70}$

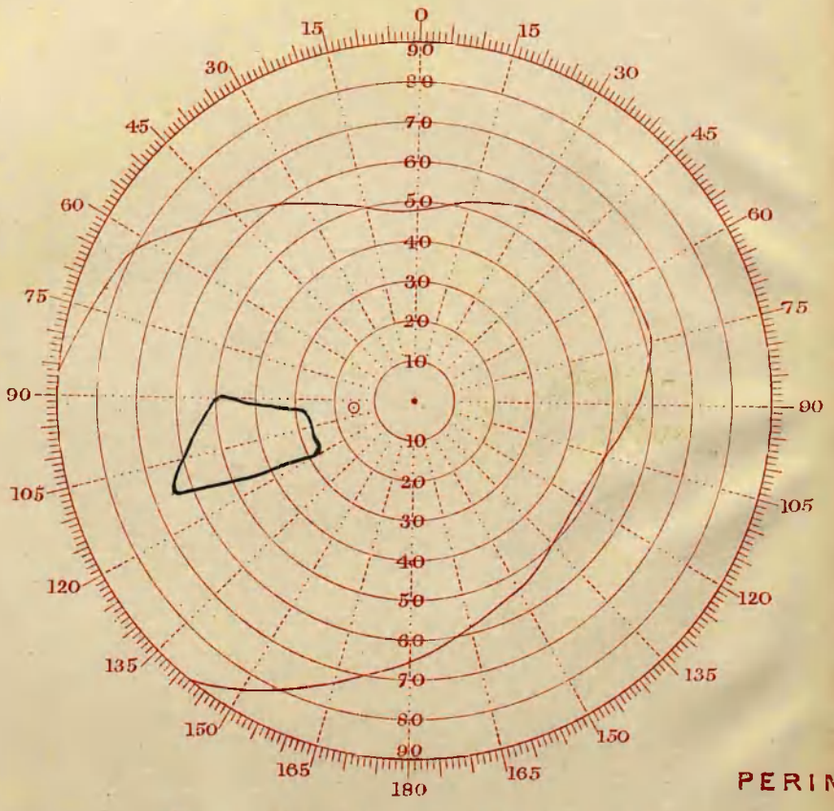


P. C. (age 77). V.A.R. = Fingers at 3 ft. V.A.L.

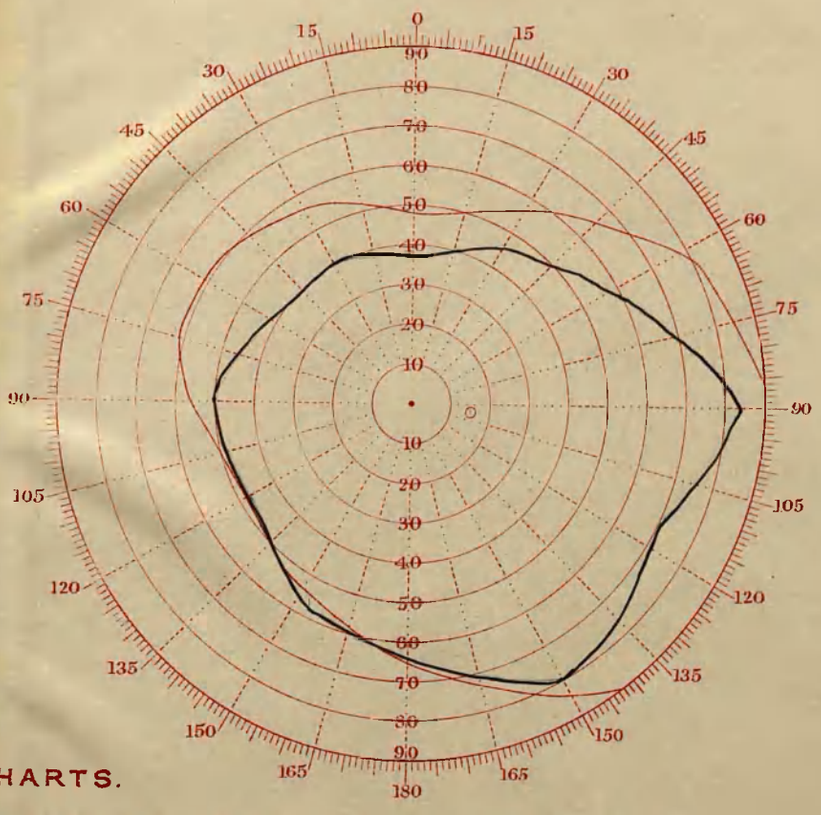
L and S. projection not good.

"Centre each chart with "pointer" at Zero before commencing to use the Automatic Registration."

LEFT.



RIGHT.



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J. P. Chronic Glaucoma. Duration 2 years. No pain whatever.