To

The Dean of the Faculty of Medicine

A Thesis

On a new Method of investigating Scotomata, its Application, and some Results obtained therewith

by

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4 Mary Place
Edinburgh, 30th April 1890.

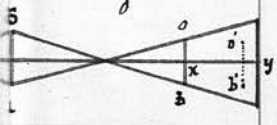
The Dean of the Faculty of Medicine

Sir,

My object in this paper is to bring forward a new method of testing for abnormal blind areas (scotomata) in various diseases affecting the eye & to set forth some results thus obtained, results which I hope will tend to clear up the causation of some of these scotomata & which will also show the value of this method in differential diagnosis

The method is a modification of De Wecker's campimetry & stated shortly the idea herein contained is to project scotomata to such a distance that very small ones & even areas in which the visual acuity is defective, can be recognized & mapped out by the aid of test objects much smaller than those usually employed. Thus suppose s a (fig 1) to be the defective area then a test object o b placed at x may be so large as to overlap the edges of the projection outwards of the defective area but on using a smaller test object o' b' & increasing the distance of the projection outwards, say to y we can readily recognize it & map out its boundaries.

Fig 1



In my investigations I use for this purpose a large screen of black velvet about 2 metres square in the centre of which is a fixation object of white ivory, 1 centimetre in diameter. From this as centre radii of fine black silk cord diverge, each including an angle of 15° so as to correspond with the radii on the

ordinary perimeter charts. These radii are all accurately measured & each is marked at intervals of 50 millimetres so that you can calculate at a glance the exact distance of your test object from the fixation point.

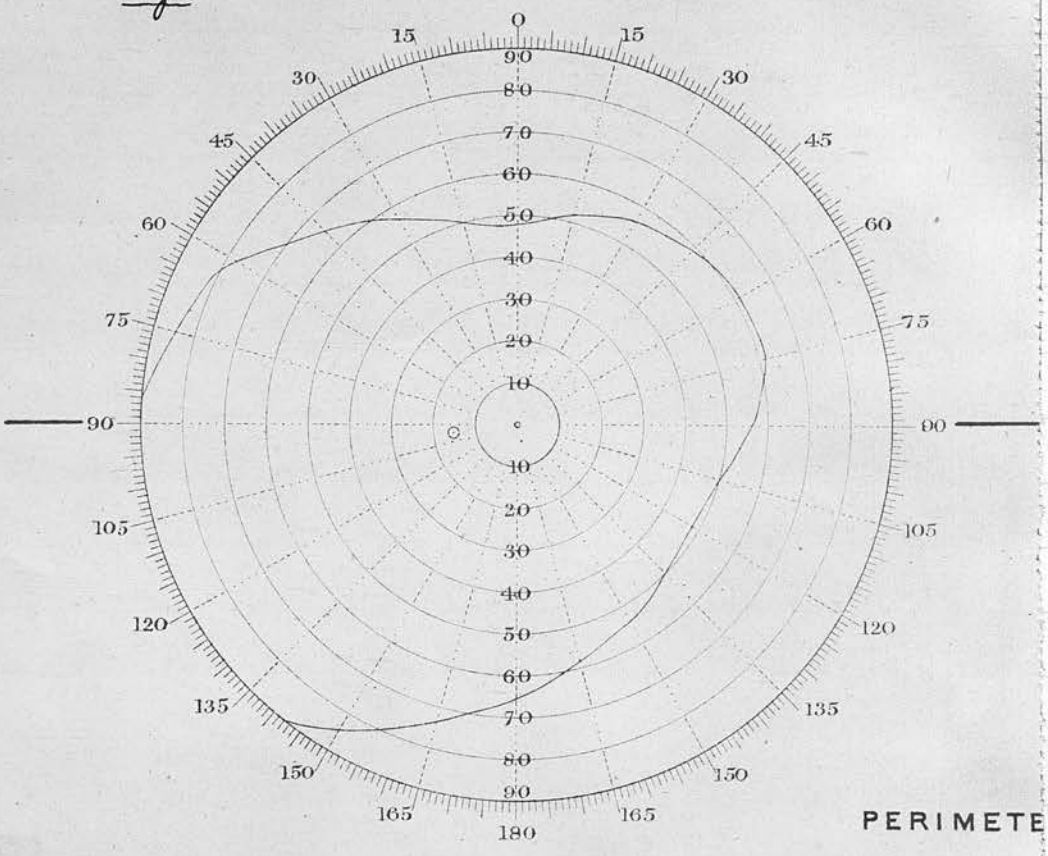
The test objects are small pieces of white ivory, 3 m.m & 5 m.m square respectively & are placed on the end of a rod covered with black velvet which is almost invisible against the screen at the distance at which the test is employed & so does not distract attention

The distances at which the test is employed are 1 metre & 2 metres respectively & to ensure the head of the patient being kept in one position I use a chin rest that can be raised or lowered as required & that also has two side pieces one of which is movable inwards & outwards & serves to prevent movement of the head from side to side when placed between them

The way I examine is that having fixed the head & the eye to be examined having been placed in an exact line & on a level with the central point of the screen & the other eye having been covered by a shade, the patient is then told to look steadily at the white spot in the centre of the screen (the fixation point) while the test object is advanced from the periphery towards the centre in a certain plane, e.g. the vertical, until it is just recognized by the eye under examination. This point corresponds to the limit of the visual field for that meridian & on

"Centre each chart with "pointer" at Zero before

Fig 2



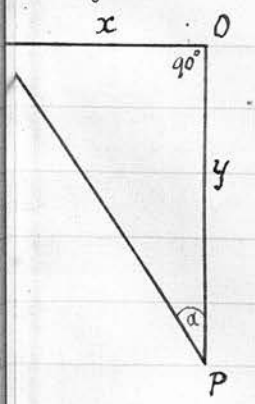
The eccentric continuous red line indicates the average normal Fiel

Designed for use with Prof. McHardy's Registering Perimeter.

continuing to advance the test object towards the centre its disappearance at any point at once points out any abnormal blind area existing & its distance from the centre is at once noted. The oblique & horizontal meridians are similarly tested & the data are transcribed on to a diagram or chart such as is here shown (fig 2). In this chart we have a series of concentric circles cut by numerous radii or diameters The centre corresponds to 0° or the point of fixation & the diameters to the different planes in which the measurements have been made. At the extremity of each radius a number shows the inclination of the corresponding meridian to the vertical while the radii themselves are divided into equal parts each subtending an angle of 10°

In the manner here described you get the exact area of the scotoma in millimetres & in order to convert these into degrees so that we can map out the area on the charts, it is necessary to make the following calculation (vide fig 3)

Fig 3.



$x = OJ$ = distance on the screen from the fixation point

$y = OP$ = distance of patient from screen (1 m. or 2 m)

The formula is $\text{Tan } \alpha = \frac{x}{y}$

Thus suppose $x = 650$ m.m and $y = 2$ m. then

$\text{Tan } \alpha = \frac{650}{2000} = .325 = 18°$

This calculation gives the tangent of the angle required as a decimal for whose value you refer to a table of natural tangents, the one I use being J. T. Bottomley's

, four figure mathematical tables.

In order to facilitate the mapping out of contours however, it is advisable to have a scale calculated out for 1 m and 2 m respectively so I accordingly use the following

	At 2 m. distant	At 1 m distant
$\frac{1}{20}$ m	= 50 m.m. = 1° 26′	2° 52′
$\frac{2}{20}$ m.	= 100 m.m = 2° 52′	5° 43′
$\frac{3}{20}$ m	= 150 m.m = 4° 14′	8° 32′
$\frac{4}{20}$ m	= 200 m.m = 5° 43′	11° 19′
$\frac{5}{20}$ m	= 250 m.m = 7° 7′	14° 2′
$\frac{6}{20}$ m	= 300 m.m = 8° 32′	16° 42′
$\frac{7}{20}$ m	= 350 m.m = 9° 56′	19° 14′
$\frac{8}{20}$ m	= 400 m.m = 11° 19′	21° 48′
$\frac{9}{20}$ m	= 450 m.m = 12° 41′	24° 14′
$\frac{10}{20}$ m	= 500 m.m = 14° 2′	26° 34′
$\frac{11}{20}$ m	= 550 m.m = 15° 22′	28° 48′
$\frac{12}{20}$ m	= 600 m.m = 16° 42′	30° 58′
$\frac{13}{20}$ m	= 650 m.m = 18°	33° 1′
$\frac{14}{20}$ m	= 700 m.m = 19° 18′	35°
$\frac{15}{20}$ m	= 750 m.m = 20° 33′	36° 52′
$\frac{16}{20}$ m	= 800 m.m = 21° 48′	38° 40′
$\frac{17}{20}$ m	= 850 m.m = 23° 1′	40° 22′
$\frac{18}{20}$ m	= 900 m.m = 24° 14′	41° 59′
$\frac{19}{20}$ m	= 950 m.m = 25° 24′	43° 32′
$\frac{20}{20}$ m	= 1000 m.m = 26° 34′	45°

The great difficulty here is to ensure that the

patient is always fixing correctly the centre spot & to obviate this as far as possible I tested his visual field several times & took the average.

It is important to have the screen amply illuminated by diffused white light or otherwise considerable variations in results will ensue.

Moreover any existing Ametropia of your patient must be remedied by using the requisite correcting glasses.

Before proceeding however to investigate scotomata it is necessary to know 1. the average normal limit of the visual field, i.e. the extent of a plane at right angles to the visual axis over which the eye can recognize objects; 2. also the size & position of the blind spot, i.e. the external projection of the optic disc at which situation the retina does not exist.

1 After examining a large number of normal eyes I found that the <u>average minimum field</u> of vision for a test object 3 m.m square at 2 m was Upwards 25° Downwards 28° Inwards 30° Outwards 35° while that for 5 m.m square at 2 m. was Upwards 35° Downwards 34° Inwards 40° Outwards 49°

Considerable variations in the visual field are met with normally though the dimensions stated are the average minimum. Variations up to 10° are often met with so too much weight must not be given to slight concentric limitations

unless the other eye is also tested. It is the irregular limitations, the more or less sector or wedge shaped defects & scotomata that are important for individual peculiarities are met with in concentric limitations but never normally as marked indentations or scotomata.

2 The Blind Spot is 250 m.m. in diameter or 7°7' & it is situated 13° from the fixation point directly outwards. It varies in shape & size but is in the majority of cases oval with the broadest end uppermost. Its dimensions are 7° wide & 8° long extending 5° below the central line of the visual field & 3° above it.

It is interesting to note that by this method you can map out the retinal vessels as they leave the disc to run in front of the percipient elements of the retina & I have often traced them as fine blind streaks extending as far as from ½ to 1 ft from the blind spot

Another point of interest which to my knowledge has hitherto not been observed is that immediately surrounding the blind spot is a zone about 20 m.m broad in which both form & colour sense is lowered though the defective zone for colour is a little larger than that for form as it is 50 m.m in breadth

This fact easily demonstrated by this method, $\frac{3}{2000}$ (I may here state once & for all that in future throughout this paper I will designate this method $\frac{3}{2000}$ or $\frac{5}{2000}$ while that of the ordinary method, the perimeter, will be $\frac{10}{300}$)

shows that immediately surrounding the optic disc there is a band of nerve fibres whose sensibility for form & colour is lowered compared to that of the rest of the retina.

A third point of interest is to compare the fineness of this method with that in ordinary use i.e. the perimeter such as Mc Hardy's which consists of an arc or quadrant of a circle which in turning on a point describes a hemisphere where the observed eye forms the centre & the radius of which is 30 c.m & the test object 1 c.m. in diameter while I use a distance of 2 m. & a test object 3 m.m. in diameter. In the first case the visual angle subtended is $\frac{10}{300}$, in the second $\frac{3}{2000}$ or approximately 2° and 5′ respectively, the exact amounts being 1°53′ and 5′.

As one retinal element has a diameter of .003 or 1′ approximately, in the first case we stimulate thousands of them $115 \times 115 = 13,225$ while in the second case we only stimulate $5 \times 5 = 25$

A further comparison of the visual fields in the two methods is:-

Perimetric		Campimetric	
Test object of 1 c.m		Test object of 3 m.m.	Test object of 5 m.m
Up	55°	25°	35°
Down	70°	28°	34°
In	60°	30°	40°
Out	90°	35°	49°
Blind Spot = 2° diameter 14° to 15° from fixation point		4° diameter 13° from fixation point	

A screen of 2 m diameter thus admits of testing up to about 27° from the point of fixation if it be in the middle of the screen; to about 45° if it be at the edge.

I wish here to state my deep obligations to Dr. G.A. Berry to whom I am indebted for this idea as he has been in the habit of testing scotomata in Toxic Amblyopia somewhat in this manner for some years & who also kindly permitted me to examine cases from his Clinique & assisted me with works of reference otherwise not easily procured.

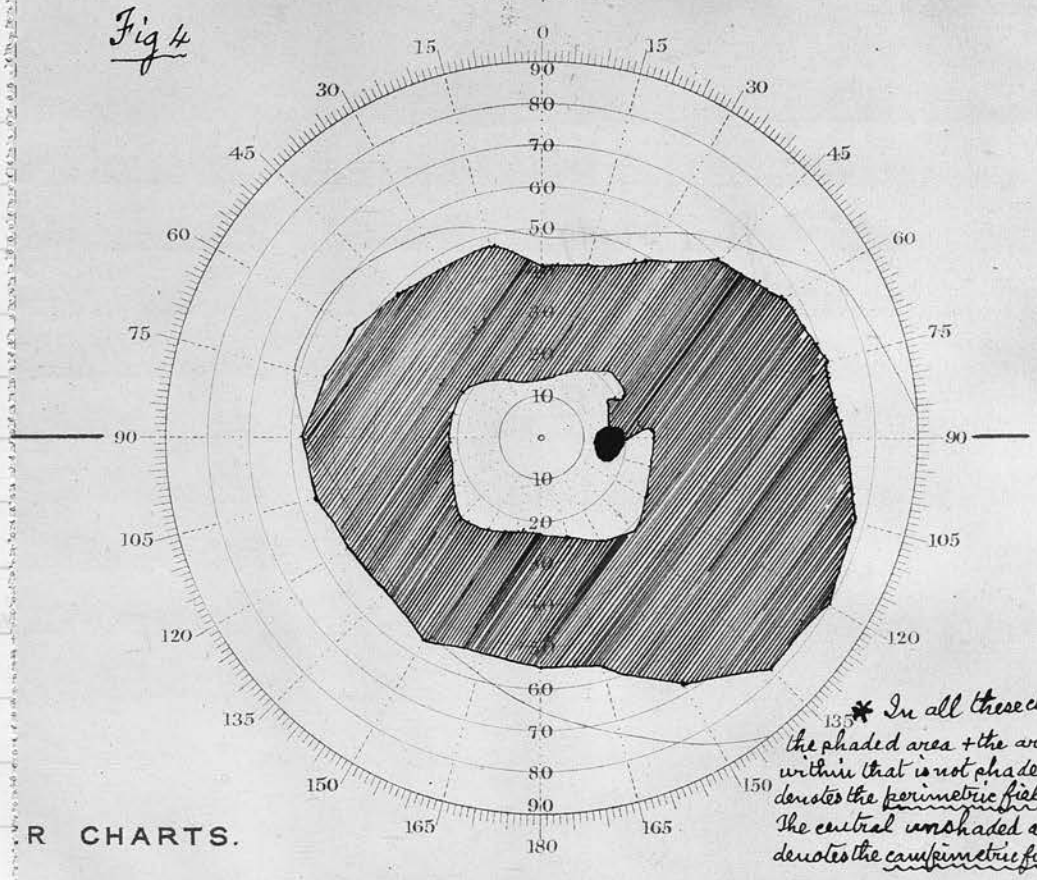
Having estimated as described the normal boundaries of the visual field we can now turn to consider abnormalities in it met with in disease. & the first class of cases to which I wish specially to direct attention is that of "Glaucoma"

Here we meet with a very curious contraction of the visual field, hitherto unknown & one which I firmly believe to be especially characteristic of "Glaucoma" & which helps to throw light upon the cause of the defective visual acuity usually displayed in this disease. I propose firstly to state the notes of the cases which I have examined, showing at the same time the charts derived by this method & those obtained by Mc Hardy's perimeter & then secondly to discuss the conclusions to be drawn from them.

commencing to use the Automatic Registration".

RIGHT.

Fig 4



* In all these charts the phaded area + the area within that is not phaded denotes the perimetric field The central unshaded area denotes the campimetric field

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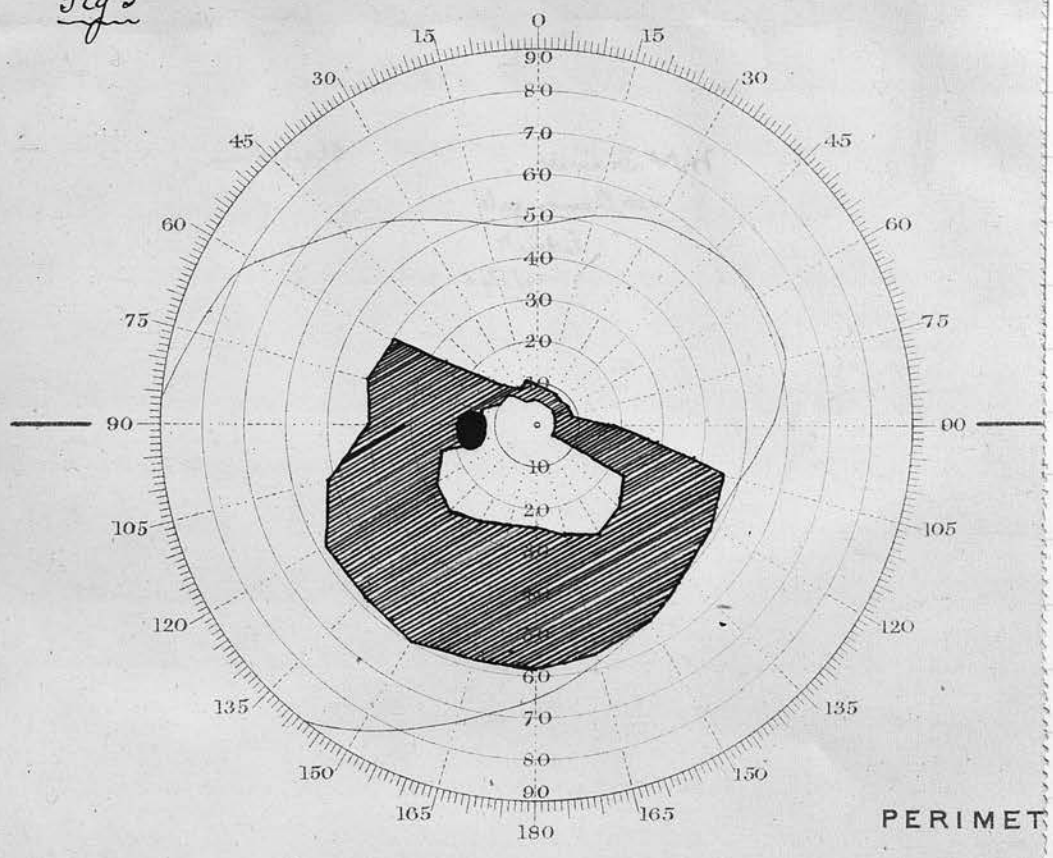
"Centre each chart with "pointer" at Zero befor

LEFT.

Fig 5



PERIMET

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Case 1. (fig 4) Jane G. aet 52 30/12/89 Subacute Glaucoma in right eye (O.D.)
She had had prodromata consisting of haloes round gas flames & misty vision for three months previously but they were checked by Eserin until at this date a subacute attack occurred
V = fingers counted at 10 ft. Anterior chamber shallow. Pupil dilated & motionless. T+2. Intense congestion
31/12/89 Iridectomy performed. T normal after operation
16/1/89 V = fingers counted at 12 ft 25/2/90 with +/- 60 lens V = $\frac{6}{18}$ Snellen & read No 4 Jaeger Chemosis & irritable conjunctivitis lasted for a month after operation
Disc exhibited well marked cupping & was pale – veins enlarged & arteries small – outer margin of disc hazy
By $\frac{10}{300}$ the visual field was slightly contracted all round except downwards & inwards
By $\frac{3}{2000}$ the field of vision was slightly constricted all round & at the upper & outer part a wedge shaped scotoma extended right up to the blind spot
Left Eye (O.S.) 10/1/90 Had haloes around flames & misty vision & slightly plus tension Checked by Eserin. Visual field normal when examined No recurrence since.

Case 2. (fig 5) Ann J. aet 54. 1883 Subacute Glaucoma of O.S (left eye)
Had had prodromata & then inflammatory symptoms & visual acuity much reduced (Subacute Attack)
Iridectomy performed No other notes to be obtained
19/3/90 Disc slightly cupped & pale – vessels diminished in some degree V = $\frac{6}{12}$
By $\frac{10}{300}$ the visual field is of normal extent downwards &

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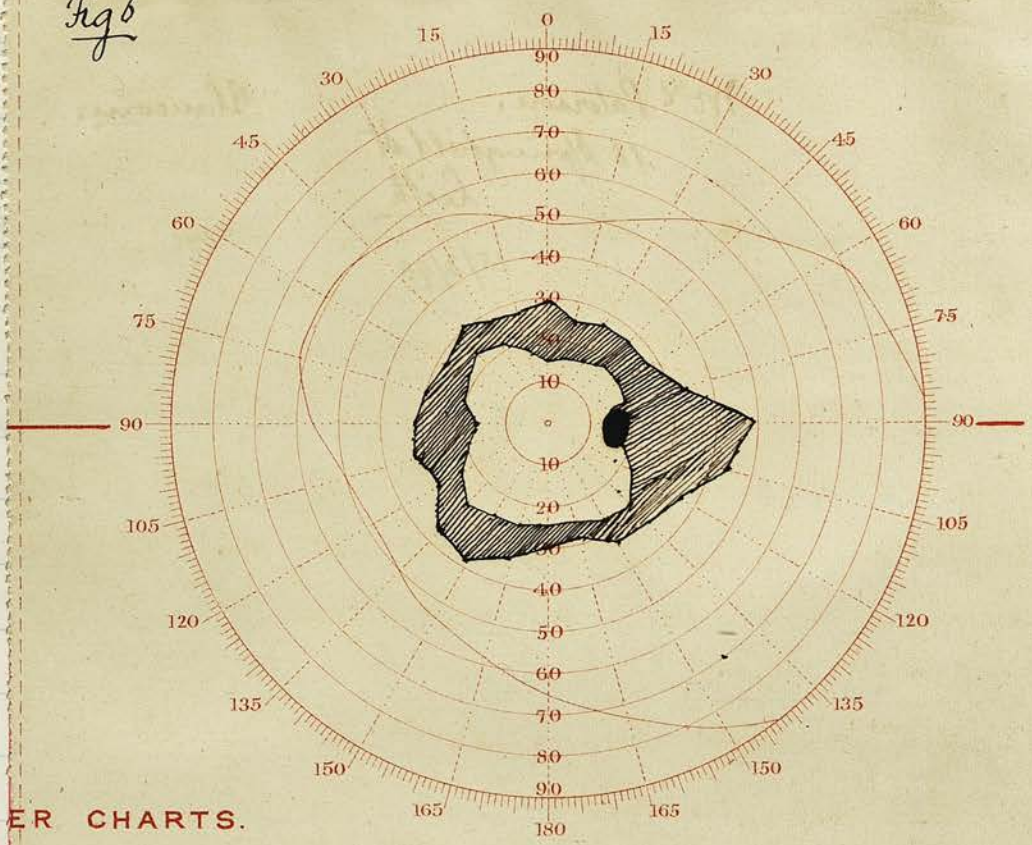
RIGHT.

Fig 6



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inwards but markedly restricted elsewhere especially above & to the inner side where it is contracted to within 10° of the point of fixation
By $\frac{3}{2000}$ while about of normal extent downwards & inwards it is elsewhere concentrically contracted to that for $\frac{10}{300}$ except to the outer side & above where a sector like scotoma extends up to the blind spot.
Right Eye (O.D.) Chronic Glaucoma complicated by Cataract V = barely hand movements T+3. Anterior chamber very shallow. Iris atrophied Pupil largely dilated & immobile 9/1/90 Iridectomy performed T+ for two days afterwards & then normal 9/2/90 Extraction of cataract 20/3/90 Needled V = fingers counted at 4 ft Disc pale & blueish & typically excavated — yellow ring around it — vessels much diminished.

Case 3. (fig 6) Mary P. aet 60. Chronic Glaucoma of right eye (O.D.) O.S. Glaucoma almost Absolute. 14/3/90 Anterior chamber very shallow T+2 Pupil dilated O.D. V = $\frac{3}{60}$ 19/3/90 Iridectomy performed V improved since operation By $\frac{10}{300}$ the visual field is found contracted all round but especially to the outer side.
By $\frac{3}{2000}$ the visual field is also contracted all round but on the outer side a sector like scotoma extends up to the blind spot. Disc well cupped

Case 4. (fig 7) Martin G. aet 69 Glaucoma Simplex in O.D. O.S. Glaucoma Absolute & T+3. Had had prodromata

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RIGHT.

Fig 7

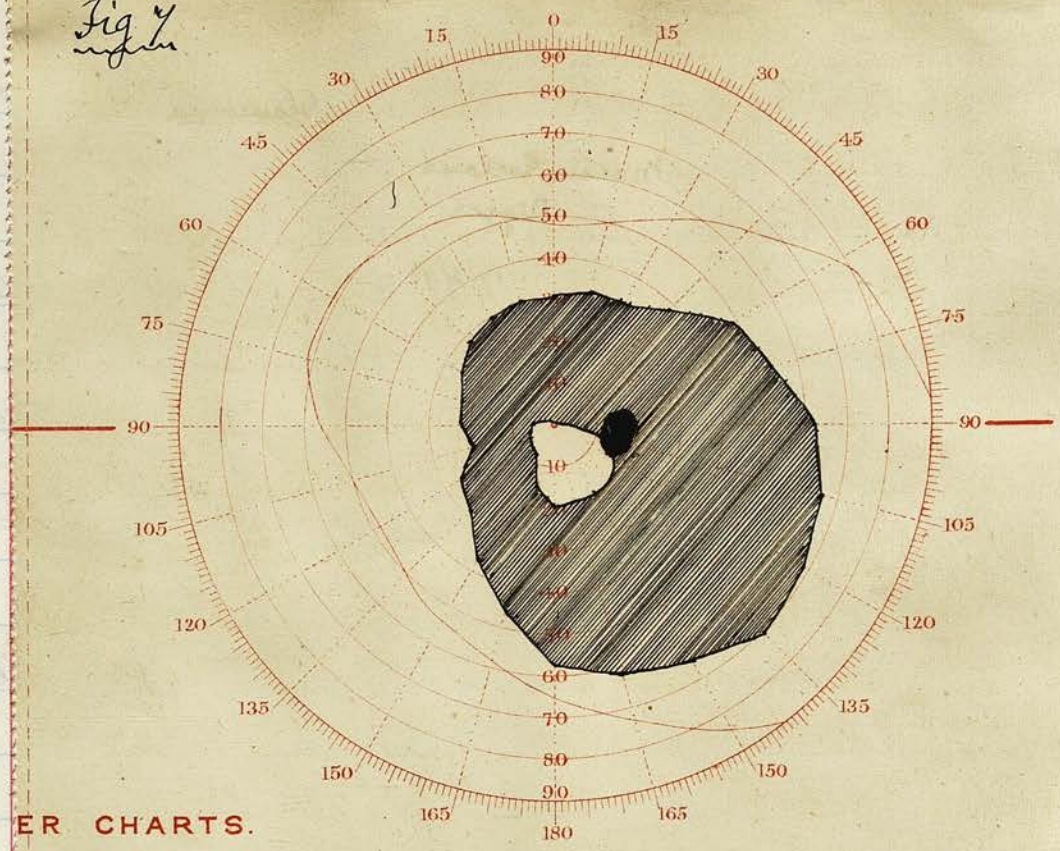

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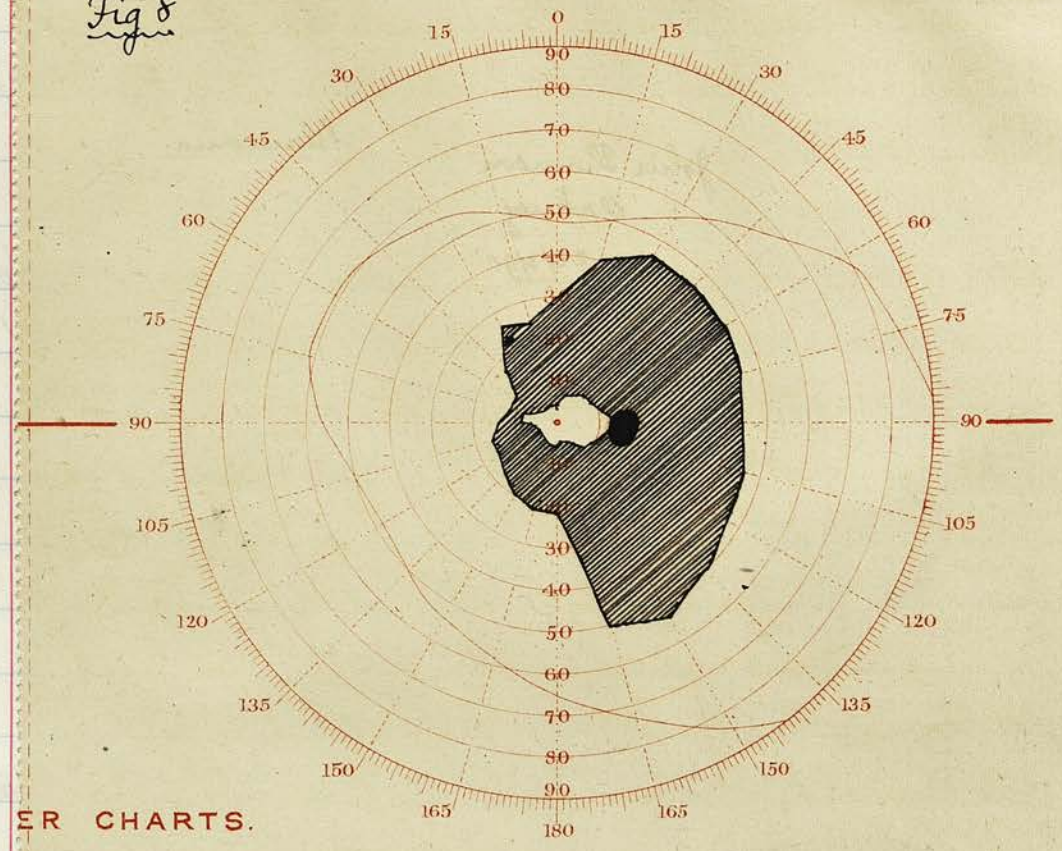
Fig 8



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in O.S. two years previously until one year ago a sudden subacute attack caused blindness & then prodromata began in O.D. till finally four weeks ago a subacute attack occurred 18/2/90 O.D. anterior chamber greatly shallowed – pupil dilated & immobile – media hazy & T+2 V= fingers counted at 12 ft 22/2/90 Iridectomy performed Tn afterwards Chemosis & irritable conjunctivitis ensued 19/3/90 O.D. V= $\frac{6}{60}$ Tn Disc moderately cupped & a yellow ring around it – media clear – vessels diminished & white lines along them.

By $\frac{10}{300}$ Visual field contracted all round but especially to the nasal side. By $\frac{3}{2000}$ the visual field extends from the blind spot along the central line to 6° to the inner side of the point of fixation & from thence 20° downwards & outwards being concentric to the field obtained by $\frac{10}{300}$ The scotoma extends up to the blind spot all round except at the lower & inner part where the visual area reaches it.

Case 5. (fig 8) John I. aet 60 Subacute Glaucoma in O.D. Glaucoma Absolute in O.S. A year previously had had prodromata that lasted six months & then an acute attack rendered O.S. blind when prodromata started in O.D. & lasted two months before a subacute attack occurred 20/3/90 O.D. Anterior chamber shallow – pupil dilated & motionless T+1 V= $\frac{4}{60}$ Disc well cupped & pale – arteries & veins diminished in size – white lines along the vessels – media fairly clear.

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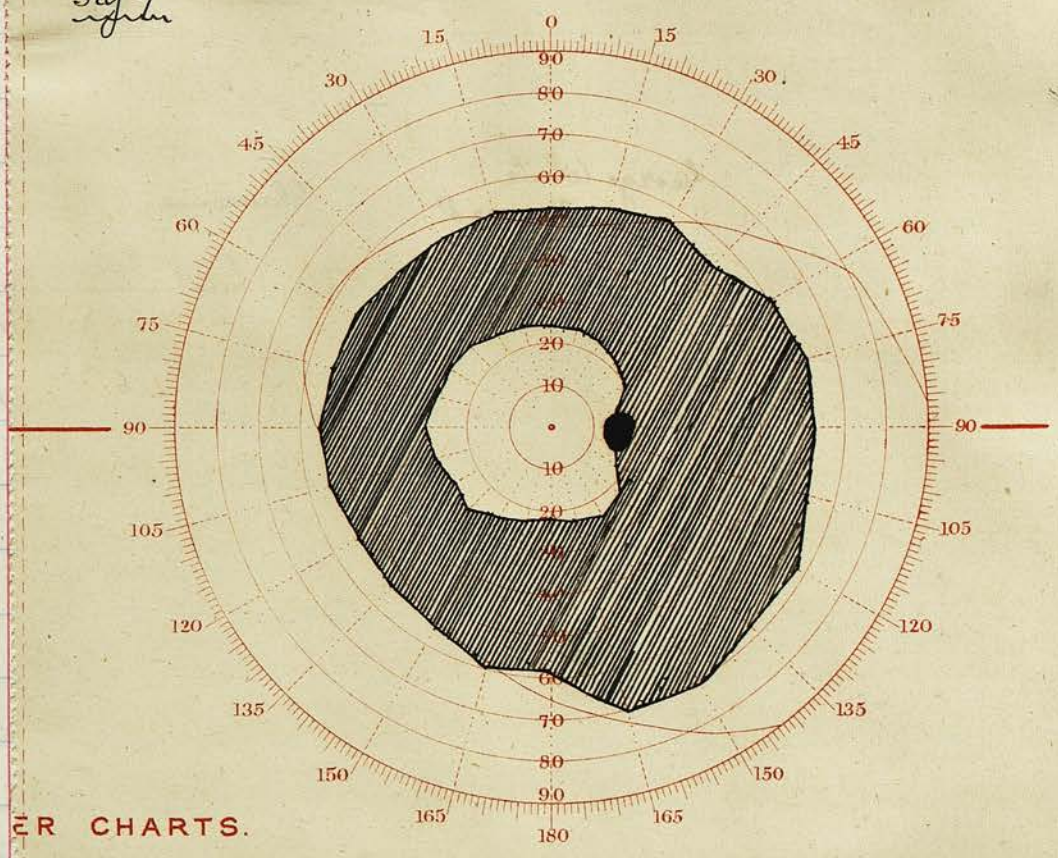
Fig 9

RIGHT.



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No operation but administered E serin
By $\frac{10}{300}$ the visual field is restricted all round but more especially to the nasal side where it is within 14° of the fixation point & on the outer side within 45°
By $\frac{3}{2000}$ there is only left intact the papillo-macular area extending from the blind spot to 8° to the inner side of the fixation point. Elsewhere the scotoma extends right up to the blind spot. The papillo-macular area being intact accounts for such relatively good vision $\left(\frac{4}{60}\right)$ in such a contracted field.

Case 6. (fig 9) George W. aet 60. Chronic Glaucoma of O.S. now almost Absolute. Had had prodromata for three years at intervals but no subacute or acute attacks.
20/1/89 Iridectomy performed 29/3/90 Anterior chamber nil as iris is against the cornea – pupil dilated & immobile T+2 V= fingers counted at 1 ft Disc well cupped & pale – lamina cribrosa stippled – vessels diminished in size O.D. Prodromata have existed for the last four months – pupil sluggish & slightly dilated – anterior chamber slightly shallowed T+1(?) Disc pale slightly & a very shallow cup probably only an exaggeration of the physiological cup on the Temporal side – margins regular V = $\frac{6}{12}$ With +4·50 Sph reads No 1 J, E serin ordered.
By $\frac{10}{300}$ we notice a rare form of restriction as it is limited almost entirely to the outer side & is seen very slightly indeed on the inner Elsewhere normal.
In 100 cases cited by Benge, this form of restriction

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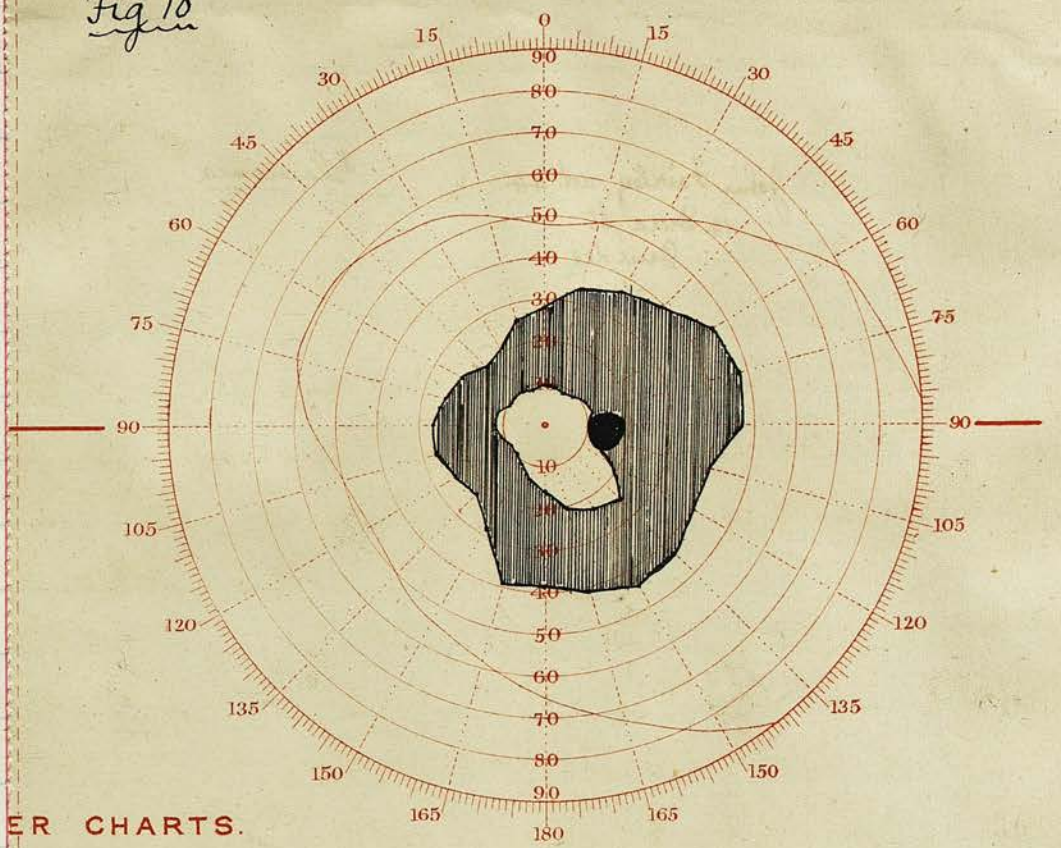
Fig 10



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only occurs in four (Berry, Diseases of the Eye, p 209)
By $\frac{3}{2000}$ we note that the restriction is almost entirely on the outer side where a sector like scotoma extends up to the blind spot Restriction below is slight.
Elsewhere normal

Case 4. (fig 10) John F. aet 44. Glaucoma Simplex of O.D. complicated by cataract. Prodromata existed a year previously until three months ago when he was blind
4/1/90 Anterior chamber shallow – cornea slightly hazy – pupil dilated & motionless – perception & projection of light good – lens opaque T+3 12/1/90 Iridectomy performed T normal afterwards 23/1/90 Cataract extracted & was found to be slightly Morgagnian. Disc well cupped & hazy & paler than disc of O.S. – yellow ring around disc & widest at outer side where it was 3/4 breadth of disc – arteries small & veins enlarged T normal 3/2/90
V with +10·0 Sph = $\frac{6}{18}$.
By $\frac{10}{300}$ the visual field is contracted all round but more especially to inner & outer sides.
By $\frac{3}{2000}$ the visual field embraces the papillo-macular area & extends inwards to 12° from fixation point & 25° downwards & outwards. Elsewhere a sector like scotoma extends up to the blind spot. The preservation of the papillo-macular area accounts for the good vision ($\frac{6}{18}$) in so contracted a field.

Case 5. (fig 11) John R. aet 64. Subacute Glaucoma of O.D.

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Fig 11

RIGHT.

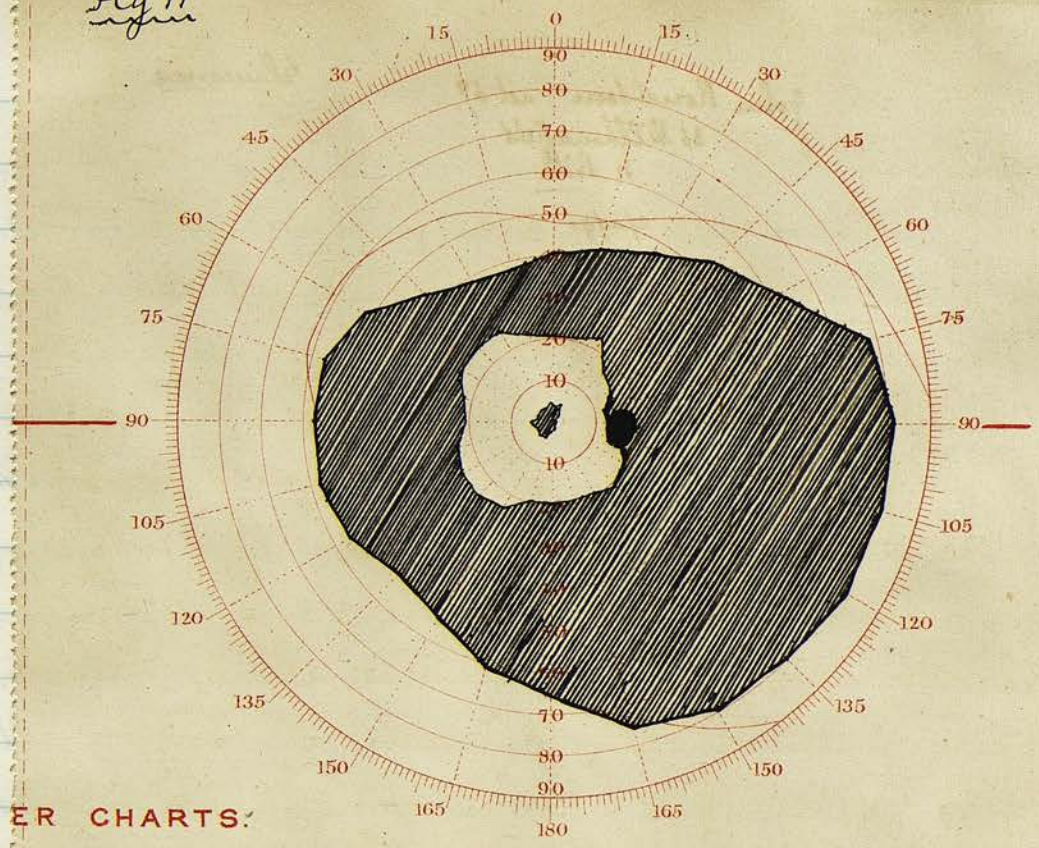

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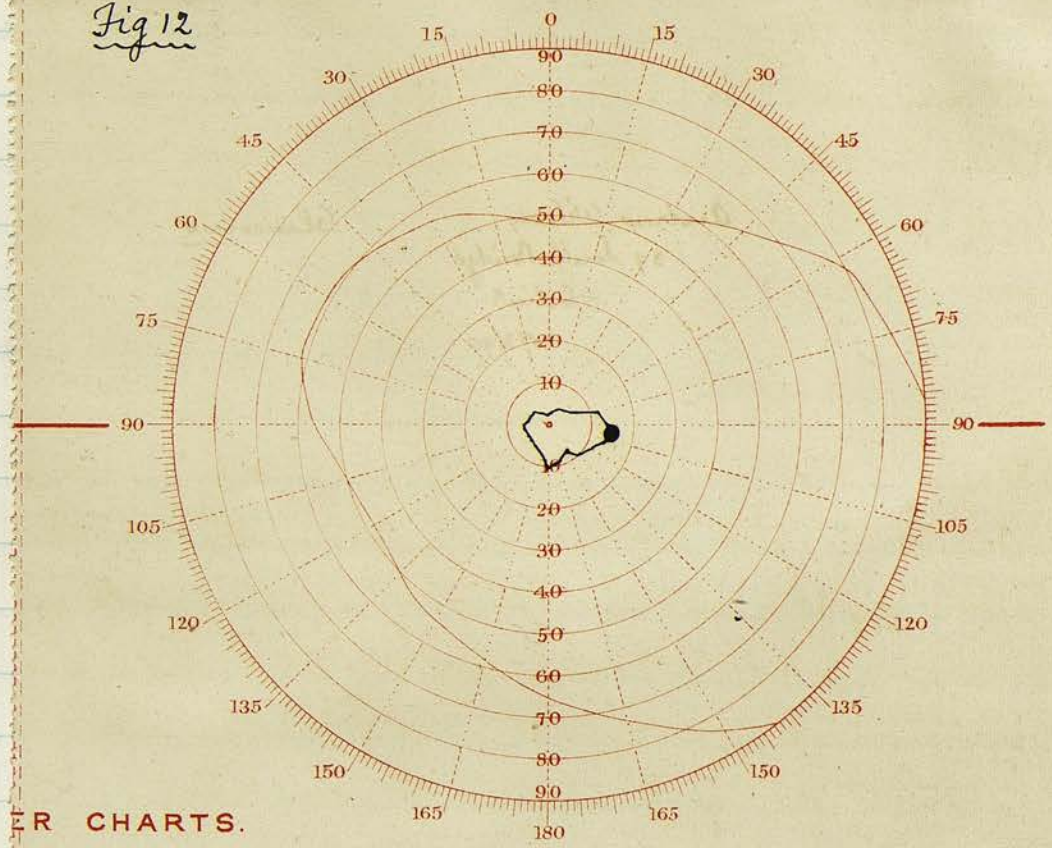
Fig 12

RIGHT.



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7/2/89 A year previously had had orbital pain, haloes round gas flames & misty vision which recurred at intervals Now anterior chamber is very shallow - iris pressed against cornea - pupil motionless & dilated - cornea & media hazy T+1 V= fingers counted at 6ft Visual field not much contracted as tested by hand. 8/2/89 Iridectomy performed T normal after operation 26/3/89 Return of symptoms — Pilocarpine ordered V= fingers counted at 14 ft. 30/3/90 V= $\frac{6}{18}$ Disc pale & slightly cupped - margins indistinct above & to inner side - white lines along the vessels. T normal By $\frac{10}{300}$ the visual field is slightly contracted to inner, upper & outer sides Normal below. By $\frac{3}{2000}$ there is slight constriction all round, being most marked on the outer side where a sector like defect extends up to the blind spot. A small central scotoma involving the fixation point is also brought out which probably would have involved the papillo-macular area & caused great deterioration of sight but for the relief of pressure given by iridectomy.

Case 9. (fig 12) Andrew W. act 84. Chronic Glaucoma of O.D. Glaucoma Absolute in O.S. 31/1/90 Three years previously prodromata existed in O.S. and in one year caused blindness without any acute or subacute attacks Then prodromata began in O.D. & now the media are hazy & there is a characteristic greenish appearance Anterior chamber extremely shallow - iris applied to cornea - pupil dilated & motionless T+2

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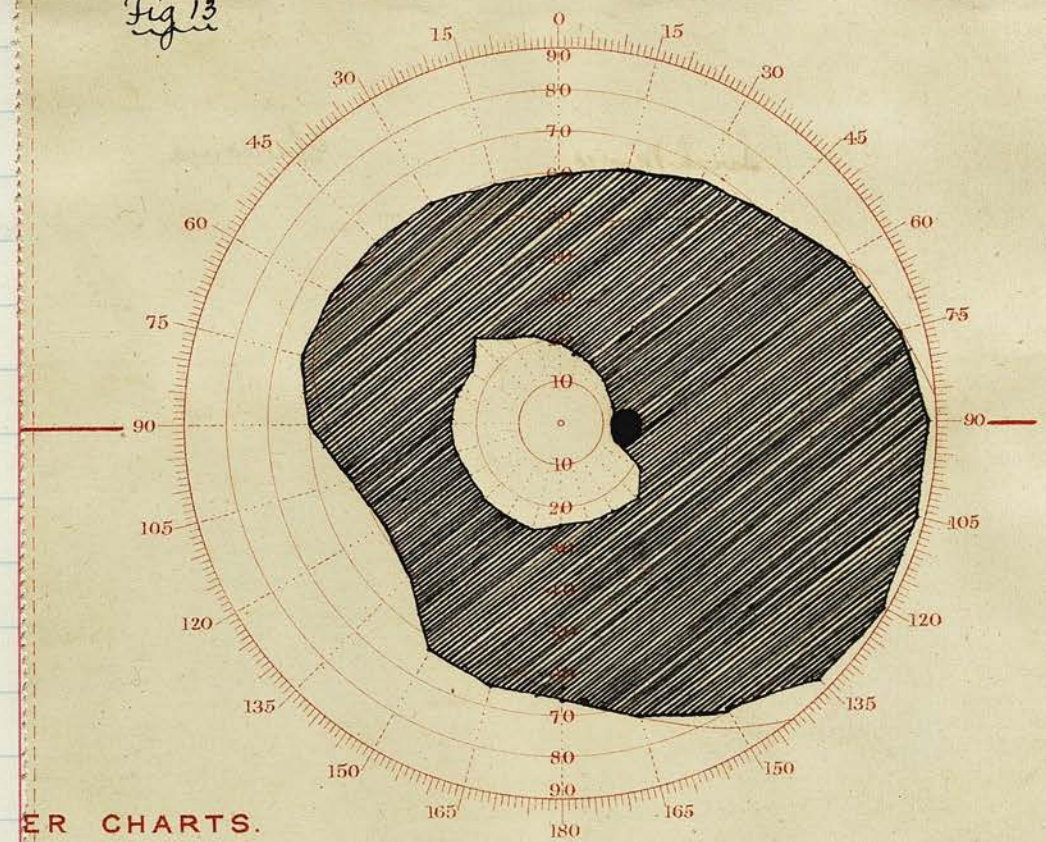
Fig 13



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V= fingers counted at 8 ft. Disc hazy, pale & well cupped - vessels greatly diminished
By $\frac{10}{300}$ the visual field includes the papillo-macular area chiefly extending from the blind spot to 6° to inner side of fixation point & downwards for 10°.
Could not test by $\frac{3}{2000}$

Case 10. (fig 13) Sarah Moores, aet 47. Glaucoma Absolute of O.S.
10/12/88 Had subacute attack in O.S. 11/12/88 Iridectomy performed & V = $\frac{6}{24}$ afterwards 16/4/89 Another subacute attack 19/4/89 Sclerotomy performed V= fingers at 2 ft 3/4/90 Absolute Glaucoma in O.S. Two days previously she had prodromata of haloes round gas flames & misty vision in O.D. after a period of mental anxiety & loss of sleep from a death in the family. On this date the anterior chamber was a little shallow - cornea dull & stippled - pupil large & sluggish T+2 V = $\frac{6}{24}$
By $\frac{10}{300}$ the visual field is if anything very slightly restricted outwards but otherwise normal.
By $\frac{3}{2000}$ there is slight constriction all round, most marked at the outer side where a sector like scotoma extends up to the blind spot Disc apparently normal
Eserine was administered & a week later T was normal, vision much improved, being $\frac{6}{12}$ & most important of all the sector like scotoma was disappearing rapidly & did not quite extend up to the blind spot.

Case 11. (fig 14) Hector Mc B. aet 49. Chronic Glaucoma of O.D.

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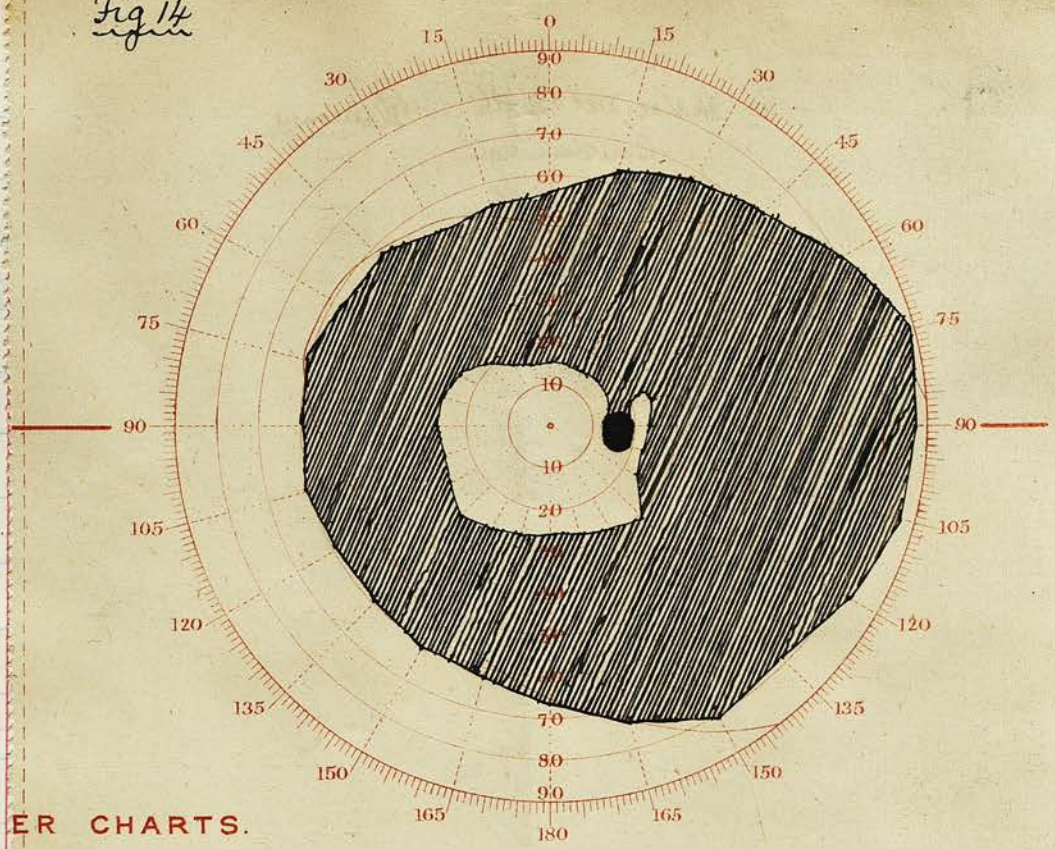
RIGHT.

Fig 14



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

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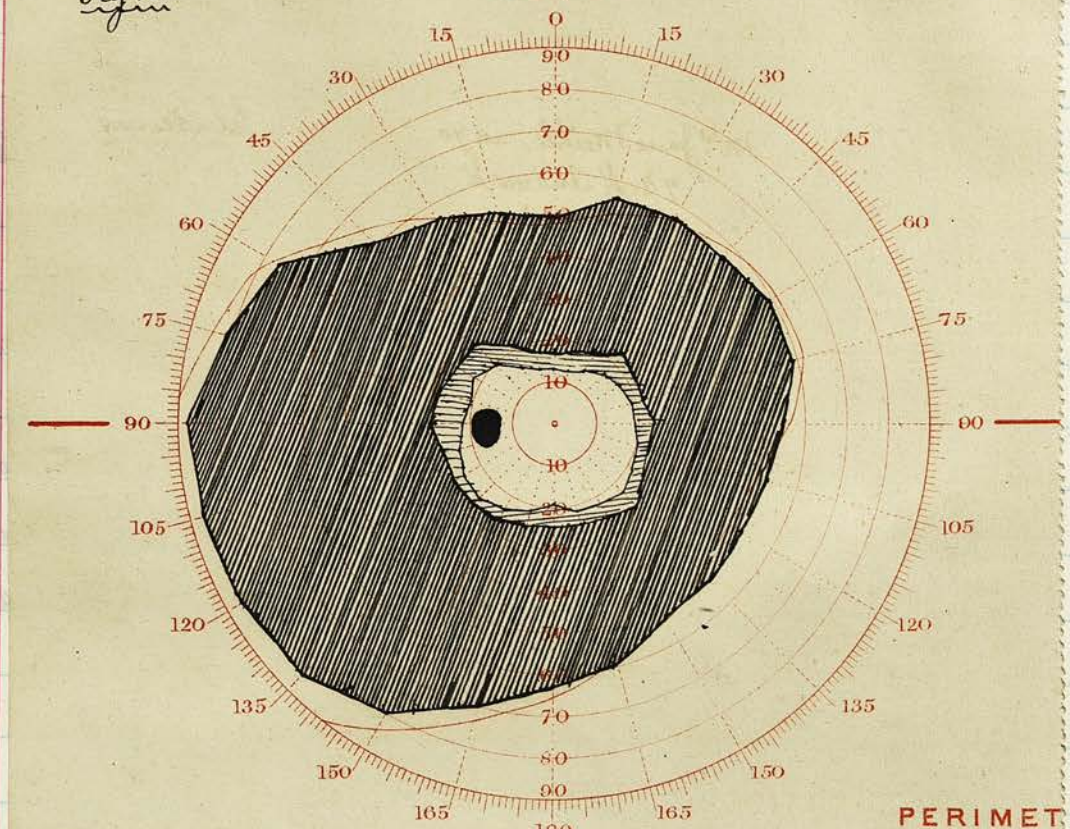
Fig 15



PERIMET

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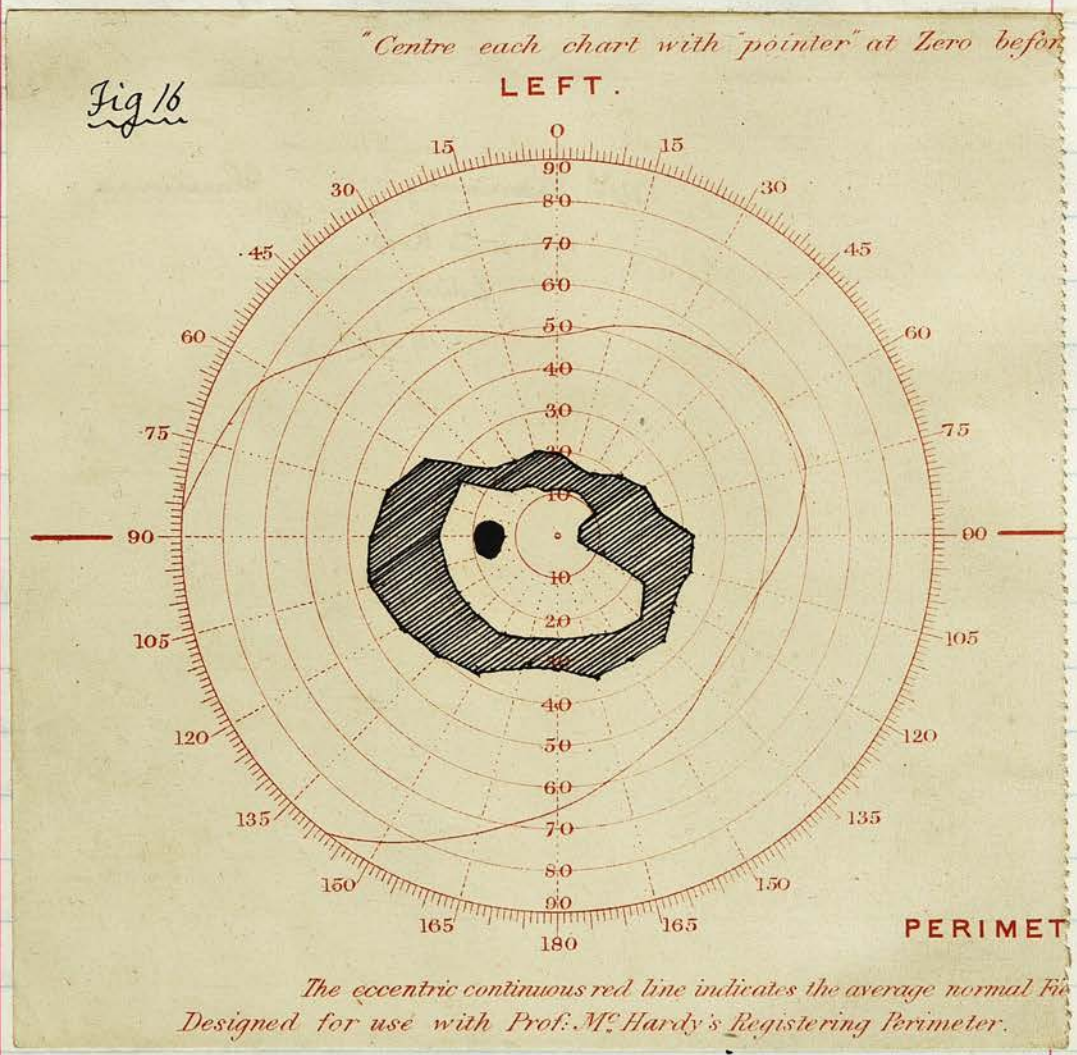
Has had misty vision becoming worse at times but no haloes around gasflames nor inflammatory signs.
24/3/90 Anterior chamber shallow - pupil dilated & sluggish - cornea stippled - episcleral veins enlarged T+ slightly Disc has a saucer-like cup, is hazy & the vessels are dimly seen at its bottom - scleral ring well marked - arteries small & veins enlarged By $\frac{10}{300}$ the visual field is slightly restricted to the outer side. Field for colours restricted to outer side concentrically to that for white By $\frac{3}{2000}$ the visual field is slightly restricted all round especially above where a wedge shaped scotoma extends up to the blind spot. Eserine was administered Ten days later the tension was normal & the wedge shaped scotoma began to disappear.

Case 12. (fig 15) Jane M. aet 70. Subacute Glaucoma of O.D. on 12/2/89. Four months previously when ill in bed the eye became painful, bloodshot & felt like a stone while she could only see fingers 3ft distant The notes are deficient here however. 13/2/89 Iridectomy performed 20/2/89 V = fingers counted at 12 ft 2/4/90 Absolute Glaucoma in O.D. & media too hazy to see the disc.
2/4/90 Has had haloes around gasflames & misty vision at intervals for the last six months in O.S. & two or three days previous to this date had had an attack. Anterior chamber shallow - cornea hazy - pupil large & sluggish T+1. By $\frac{10}{300}$ there is slight constriction to inner & outer sides By $\frac{3}{2000}$ there is marked constriction all round

Fig 16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Designed for use with Prof: Mc Hardy's Registering Perimeter.

, more especially to the outer side where it extends to within 2° of the blind spot. After the use of Eserin for ten days the field enlarged especially on the outer side (see chart where the part marked by cross shading denotes the area of enlargement) & the tension became normal. Probably had I seen the patient a day or two earlier I would have found the scotoma extending up to the blind spot on the outer side for when I saw her the attack was passing off.

Case 13. (fig 16) Abigail G. aet 49. Subacute Glaucoma of both eyes. 29/10/89 No prodromata at all. Five weeks previously had a sudden subacute attack during which patient states she was almost completely blind for ten days Misty vision & haloes were complained of at intervals till another subacute attack occurred on this date Corneae steamy & stippled – anterior chambers shallow – pupils dilated & sluggish T+2 O.D. V = fingers at 12 ft O.S. V = fingers at 18 ft 30/10/89 Iridectomy in O.D. 5/11/89 Iridectomy in O.S. T normal after operations Discs a little pale but no cupping. 13/11/89 O.D. +1·50 = $\frac{6}{36}$ O.S. +1·50 = $\frac{6}{60}$ 14/1/90 +2·0 Sph = $\frac{6}{18}$ in both eyes Reads No 1 J By $\frac{10}{300}$ there is great restriction about equally all round By $\frac{3}{2000}$ there is marked restriction all round except downwards & inwards. The scotoma nowhere approaches within 8° of the blind spot.

I introduce this case to show that this characteristic scotoma extending up to the blind spot from the periphery

is not met with in all cases of Glaucoma & the explanation here I believe to be that the disease was recent, occurred in the subacute form & that the high tension was not of long duration & promptly reduced to normal by operation

I now turn to consider the conclusions to be drawn from these facts & their bearing upon the defective visual acuity in Glaucoma.

The deductions I draw from these facts are:-

1. That the scotoma here demonstrated is characteristic of Glaucoma.
2. That the cause of the scotoma is the pressure element.
3. That the pressure element acts upon the optic nerve.

I The scotoma is characteristic of Glaucoma

This I believe is borne out by the facts that (1) it never exists in normal visual fields. I have examined thirty or forty normal cases & never once found anything at all resembling this (2) In no other disease have I yet seen it although I have examined many cases of Optic Atrophy, Choroiditis (Disseminata & Central Senile), Toxic Amblyopia, Retrobulbar Neuritis, Retinitis Pigmentosa &c.

This scotoma extends from the periphery right up to the optic disc (see cases recorded) & the important point is that with few exceptions the most defective area & that in which the function is best retained reach up to the blind spot & show an interference in

the function of the retina which always starts from the papilla & pretty clearly points out that whether from pressure or otherwise the defect is a result of the destruction of the nerve fibres in the papilla at the margins or sides of the excavation.

Bjerrum in an able paper (Nordisk Ophthalmologisk Tidsskrift, II 3, p 154 et seq) also demonstrated this fact in confirmed Glaucoma but I also examined cases in which the disease was not confirmed but merely showed premonitory symptoms (Cases X, XI, XII) & in these too I found this characteristic defect — & therefore consider it a valuable diagnostic point in the initial stage of Glaucoma where we are sometimes in doubt as to whether we are dealing with Optic Atrophy or this disease.

II. The pressure element is the cause of the scotoma

In Case X. Sarah M. p 15 during a premonitory attack I found T+2 & a slight restriction of the visual field all round except at the outer side where a sector like scotoma extended upto the blind spot. On administration of Eserin for a week & then the tension was reduced to normal & the sector like scotoma was disappearing rapidly & did not extend upto the blind spot as formerly.

In Case XI Hector McB. p 16. I found T+ & the visual field restricted slightly all round while above a wedge shaped scotoma extended in to the blind spot. Ten days administration of Eserin

reduced tension to normal & the wedge shaped scotoma was disappearing & did not now reach to the blind spot.

In Case XII Jane M. p 16 I found during a premonitory attack T+1 & great restriction of the visual field all round especially on the outer side where the defective area extended to within 2° of the blind spot. Here also ten days administration of Eserin reduced tension to normal & there was great enlargement of the field which was as much as 8° on the outer side (see chart)

The facts that when there is excessive tension this characteristic scotoma is to be found & that it slowly clears away on the reduction of tension, appear to me to show that the pressure element is the cause of the scotoma. From repeated attacks of high tension & long duration of it this scotoma becomes permanent from interference with the retinal functions.

III The pressure element acts upon the Optic Nerve

Before discussing this however it is as well to know how the visual acuity diminishes & the visual field becomes restricted.

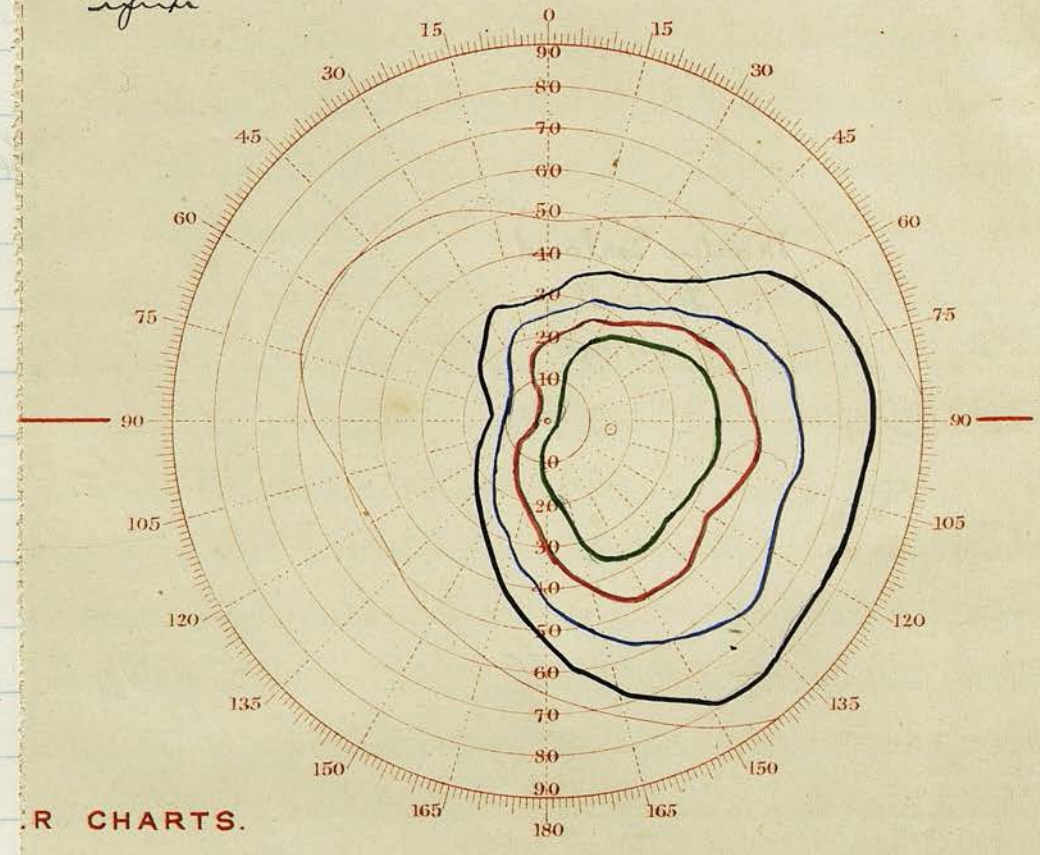
Acuteness of vision begins to diminish & becomes permanently impaired as soon as Glaucoma has passed from the premonitory to the fully developed stage. During the existence of the premonitory symptoms more or less diminution of vision occurs but is recovered from as soon

as the attack passes off. We can thus distinguish between a transitory & a permanent element in the amblyopia even in cases advanced to true Glaucoma. During each exacerbation occurring in Inflammatory Glaucoma the vision is worse than before but after the acute symptoms have subsided there is a gradual recovery to a certain extent which however does not as a rule lead to a restoration of visual acuteness as complete as before the attack. Each attack therefore leaves vision more impaired than before until it is eventually lost altogether. There is a very great difference in rapidity with which this destruction takes place which depends on the nature & severity as well as the frequency of acute attacks & upon the continuation of plus tension. In acute cases where symptoms of congestion are excessive, vision may be totally lost in a few hours & these cases are called "Glaucoma Fulminans" while chronic cases especially non-congestive ones may not lead to complete loss of vision till after many years. The blindness is absolute black darkness quite unlike that of many other diseases in which the patient can often tell light from darkness. These differences render it possible that vision is lost in two ways ① As the result of increased tension ② Where there is rapid amaurosis as the direct result as well of the vascular state which gives rise to the plus tension Here probably the suddenness & completeness of the ischaemia of the retinal vessels is enough to permanently abolish vision as in cases of Embolism of the Central Artery of the Retina.

commencing to use the Automatic Registration".

Fig 17

RIGHT.



.R CHARTS.

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

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Restriction of the Visual Field occurs at the same time as the defect in the acuity of central vision but there is no absolute constancy in the manner in which the field is invaded though most frequently we find the nasal portion abolished to a greater extent at first than other parts of the field & often this part alone can be shown to be defective (see Cases II & V). However wherever the restriction exists it is continuous, i.e. there are not as a rule isolated scotomata but if one part of the retina has lost its function that of parts more peripheral to it in the same direction is usually abolished.

The blind portion of the field is bordered by an amblyopic area in which colour vision is either absent or defective but concentric to that for white, thus differing from Optic Atrophy in which the diminution in the field of colour vision is not at all concentric to that of white. The limit at which different colours are recognizable is usually much the same for all & only slightly more retracted than the corresponding boundary for uncoloured impressions (pl. chart fig 17) while even in cases where the limitation is very great there still remains colour perception.

This limitation of colour perception concentrically to the boundary for white is characteristic of Glaucoma.

It is the rule for this greater restriction to the nasal side to progress & eventually involve the centre before complete blindness sets in so that in an advanced stage of the disease only an eccentric portion of the temporal

side remains. Preponderance of the nasal invasion of the field appears to be more common in chronic cases than in acute ones. The manner of restriction next most frequently met with is the concentric limitation where there is a tolerably proportionate interference with the functions of all parts of the retina. (see Cases III, IV, VII, XIII) It is very rare indeed to find restriction most marked outwards (Case VI) & still more so for this portion to be the only part in which the field is diminished. In not a few cases central vision is markedly diminished without any easily demonstrable defect in the field (see Case VIII) while the opposite condition of great restriction of the field with good central vision is rare (Case IX)

Dr. Berry in Diseases of the Eye, p 209 quotes Bruge's statistics in 100 cases of Glaucoma as follows:-

Defect in nasal portion alone	=	24 cases
Predominating in nasal portion	=	44 cases
Field remaining assuming form of peripapillary oval	=	4 cases
Destruction of whole field including centre with exception of a small temporal piece	=	9 cases
Central or paracentral scotoma with or without slight restriction of nasal periphery	=	4 cases
Restriction only upwards	=	2 cases
Preponderance of defect in temporal half	=	4 cases
Concentric constriction	=	6 cases

While these statistics hold good for $\frac{10}{300}$ (perimeter), this new method $\frac{3}{2000}$ however will

considerably alter them but as yet I have not tested a sufficient number of cases to be able to form a table.

Any explanation of the blindness must therefore account for ① the want of constancy in the way in which peripheral vision is lost; ② the tendency at the same time for the nasal portion to disappear before other equally peripheral parts.

As already stated the increased tension causes the amblyopia & there are three views as to how it does so

A. Pressure upon the fibres of the Optic nerve directly

B. Increased pressure on the retina itself

C. That the functional activity of the retina is gradually lost owing to an interference caused by the pressure, in the blood supply to the delicate structures of the retina.

B. Increased pressure on the retina itself.

This appears to me to be the least consistent with clinical facts. It was supposed by Donders that the reason why pressure should affect the periphery in the first place is that the nerve fibres to this region are more superficial but on this supposition it is difficult to understand why the manner in which the field is invaded should not be more regular than it is even granting although it is not proved & is most unlikely that a pressure exerted in the way the fluid pressure in the eyes is exerted would have the effect of completely & permanently interfering with the function of the more superficially coursing fibres while that of the deeper ones remained relatively undisturbed. It does not moreover explain

why the scotoma should extend right upto the blind spot in some places & not in others & therefore this theory is most unlikely.

A. Direct pressure on the optic nerve itself

This view I believe to be the correct one & the results obtained by Bjerrum & myself strongly favour it The pressure acts here in two ways ① Directly on the nerve fibres as they bend over the edge of the disc & ② Indirectly through the nerve fibres being dragged upon, displaced & atrophied by yielding of the Lamina Cribrosa & cupping of the disc. The evidence of scotomata in nearly all cases of Glaucoma extending from the blind spot to the periphery as demonstrated by Bjerrum (Nordisk Ophthalmologisk Tidsskrift II, 3. p. 134 et seq) & myself (cases quoted here) shows an interference in the function of the retina which always starts from the papilla & the defect is a result of the destruction of the nerve fibres in the papilla at the margins or sides of the excavation. Further Bunge considers that the more centrally situated fibres in the optic nerve supply the more peripheral parts of the retina & according to his scheme an excavation occupying more especially & primarily one portion of the disc might possibly explain the visual defects both central & peripheral which are most commonly found to exist if we were to assume at the same time that the fibres contained in the excavated portion of the disc were those whose function were most interfered with as doubtless they are since

they are most liable to pressure through having to bend round the margins of the disc most sharply.

In this disease we find that chronic inflammation of the optic nerve exists as demonstrated by Nettleship (vide St Thomas Hospital Reports p. 73. 74 Vol XIV) where he states that "the optic disc is often very pale as if atrophied' '& frequently shows the signs which are commonly taken to' 'indicate previous inflammation i.e. slight haziness of' 'the margins & white lines along the vessels. Although we' 'never have the opportunity of seeing with the ophthalmoscope' 'the appearances of Papillitis in Glaucoma, because in' 'the cases where it probably occurs the haze of the media' 'prevents an inspection, there can be no reasonable' 'doubt that it does occur in some degree." These observations I have several times been able to confirm (vide Cases IV. V, VIII) Brailey also upholds this view regarding inflammation of the optic nerve & further states that in primary glaucoma these changes begin before the occurrence of glaucomatous symptoms

Brailey, Ophthalmic Hospital Reports p 86 Vol X "In every' 'case of increased tension whether accompanied by retinal' 'haemorrhage or not & whether primary or secondary to' 'injury, corneal ulcer or inflammation of the uveal tract' 'there is found some change in the optic nerve. This in' 'the majority of cases examined presents itself as an excessive' 'amount of a tissue roughly resembling through far more' 'dense than the ordinary fibrous stroma of the nerve".

Again at p 282 of the same Vol X he states "it is certain'

'that inflammation of the ciliary body, iris & optic nerve is'
'always present & that it is one of the earliest symptoms of'
'primary glaucoma, being developed previous to the tension".

Moreover we often see in Surgery that inflamed tissues are more apt to yield to pressure than normal ones & we are therefore justified in believing that this inflammatory process in the optic nerve predisposes to cupping on an increase of tension within the eye occurring & further that it also tends to atrophy of the nerve which in itself is usually accompanied by shallow cupping of the disc.

Probably also there are individual differences existing with respect to the portions of the nerve which yield to the greatest extent on pressure & the same in regard to the Lamina Cribrosa thus accounting for differences in the visual defect. Probably also the existence of a well marked physiological cup favours the action of the pressure by allowing it to act more readily upon the Lamina Cribrosa which being the least resistant part of the globe more easily yields. Differences in the position of this cup in the nerve may possibly account also for differences in the visual defect e.g. if temporal, as is often seen, then the temporal part would yield most readily & this as it corresponds to the nasal portion of the visual field would explain the tendency for it to disappear first in most cases while if the cup were central then peripheral concentric restriction of the field would result

According as the inflammation of the optic nerve

involved it wholly or in part we would tend to get differences in the defect of visual acuity.

An objection to this view of pressure on the optic nerve was that when there is a complete loss of the nasal periphery of the field there ought at the same time were this the only cause to be a blind portion somewhere near the macula or a paracentral scotoma which defects were very seldom found by $\frac{10}{300}$ but Bjerrum in his paper & I in my investig-ations overthrow this objection by showing that such a defect nearly always occurs.

Dr Berry, Diseases of the Eye p228 urges that this view does not explain acute cases where vision is rapidly & permanently lost but in Surgery we know that the application of a tourniquet pressing on nerves & maintained in that position for some time will often cause a paralysis of function that may remain permanent & reasoning on analogy I believe that a similar explanation may be applied to these cases. Dr Berry further urges that this theory does not account for cases in which well defined scotomata are found in the visual field but I explain as follows :- Landois & Stirling, Textbook of Human Physiology, p 784, Vol II state that if a nerve be separated from its centre by compression or section, within a short time it loses its excitability & the peripheral end undergoes fatty degeneration. Therefore since the pressure on the

optic nerve cuts off the ends of the nerve to that part of the retina where the scotomata exist from its centre, degeneration ensues which is however checked from proceeding far by the removal of the pressure for an intermittent nature of the pressure is characteristic of Glaucoma. Frequent renewals of pressure would doubtless convert these isolated scotomata into a regular one extending completely up to the optic disc

I therefore believe that the pressure acts upon the optic nerve at the disc &, favoured in its effects as already described by cupping &, cuts off by compression some part or portions of the nerve fibres of the retina from their centre & there then sets in at the periphery a degeneration which may or may not be recovered from according as the pressure be transitory or long maintained or permanent.

C. Pressure causing interference in the blood supply to, & diminished functional activity of, the retina

This is very unlikely from pressure alone as the only way in which this could well be done would be for the pressure to produce an almost complete arrest of the circulation while if this were to happen it could not be a diffused pressure all over the retina which would cause narrowing of both arteries & veins but a pressure which acts mainly on the vessels as they enter the retina from the nerve causing diminution of arteries & engorgement of veins corresponding to the

appearance met with in Glaucoma. The possibility of an interference in the circulation taking place mainly in this way would depend either upon whether a certain degree of pressure would be more effectual in compressing the vessels in the nerve than elsewhere or whether there really existed a greater degree of pressure in this particular direction neither of which conditions there are any reasons to assume actually exist. Although the structures in the nerve against which the vessels would be compressed are probably no more resistent than in the retina, the same amount of pressure might cause greater interference with the circulation when exerted on points where sudden bends take place in the vessels as happens where they pass into the retina. The pressure might act most effectually on the smaller terminal branches of these vessels directly The restriction of the visual field however would then be much more regular. It has been suggested that changes which take place in the retinal arteries might independently lead to blindness & the occurrence of haemorrhage after iridectomy is suggestive in this respect. Brailey & Edmunds, Ophthalmic Hospital Reports, Vol X p 134, state "thus we see that in a considerable' 'proportion of cases of Glaucoma the retinal arteries are' 'hypertrophied. The thickened vessels in a later stage' 'contract & their lumen becomes smaller" Again at p 136 they state that "as to the causation of these vascular' 'changes, the starting point seems to be the inflammation'

'(or sclerosis) which as we have said is found in all cases'
'of Glaucoma. This inflammation affects the central'
'artery in its course through the optic nerve & thus'
'occasions the changes in its lymph sheath: the inflamma-'
'-tion is propagated forwards to the retinal arteries".

Degeneration of the arteries however is only got in one half the cases of Glaucoma & so is not a constant factor & as it is due to inflammation of the optic nerve it is probable that the defective visual acuity is dependent rather on changes in the nerve than in the vessels. Moreover a similar condition of the vascular walls is found in the eyes as well as in other organs of many patients who are the subjects of various general diseases & who have no defective visual acuity so accordingly we are justified in concluding that degeneration of retinal arteries has no bearing upon the amblyopia in Glaucoma.

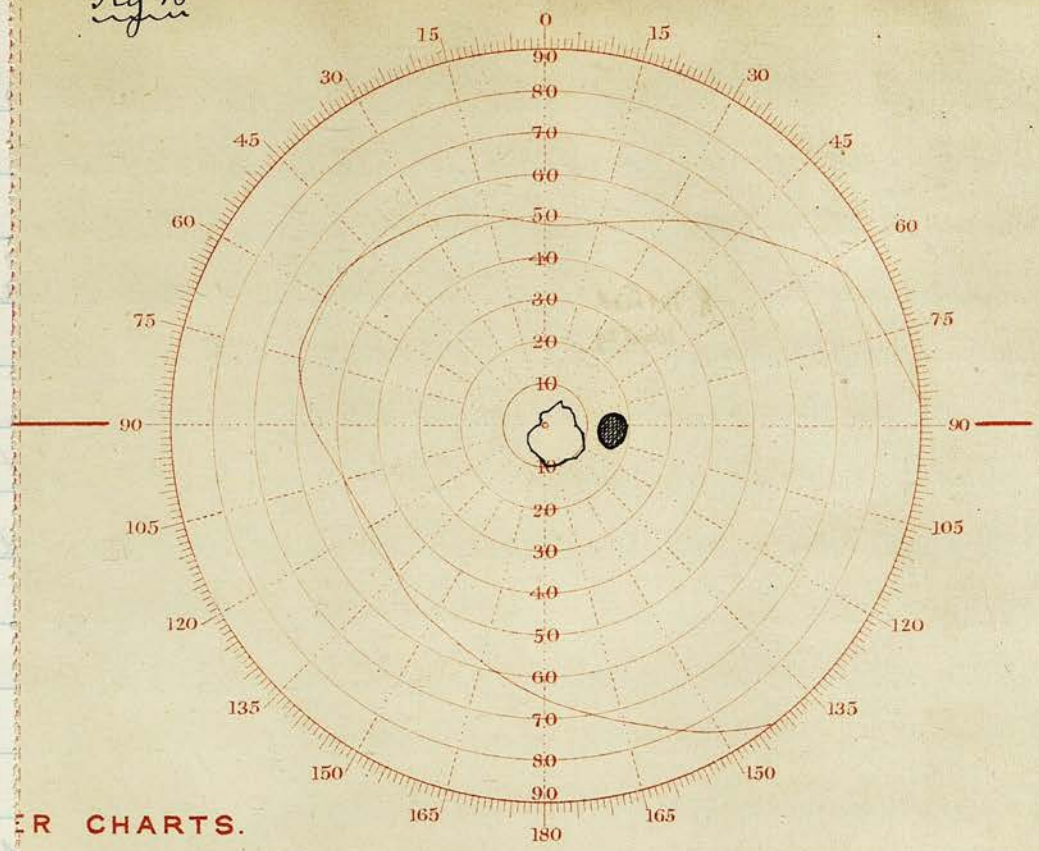
A second class of cases that I wish to bring under notice is "Optic Atrophy" which I introduce here partly to show the difference between the scotomata in them & in Glaucoma & partly to show the value of this method in diagnosis.

Juler in his Handbook of Ophthalmic Science and Practice, p 182, states that the alteration of the visual field generally consists in a general contraction the outline of which is concentric with the macula;

commencing to use the Automatic Registration."

Fig 18

RIGHT.



ER CHARTS.

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"Centre each chart with pointer at Zero before

Fig 19

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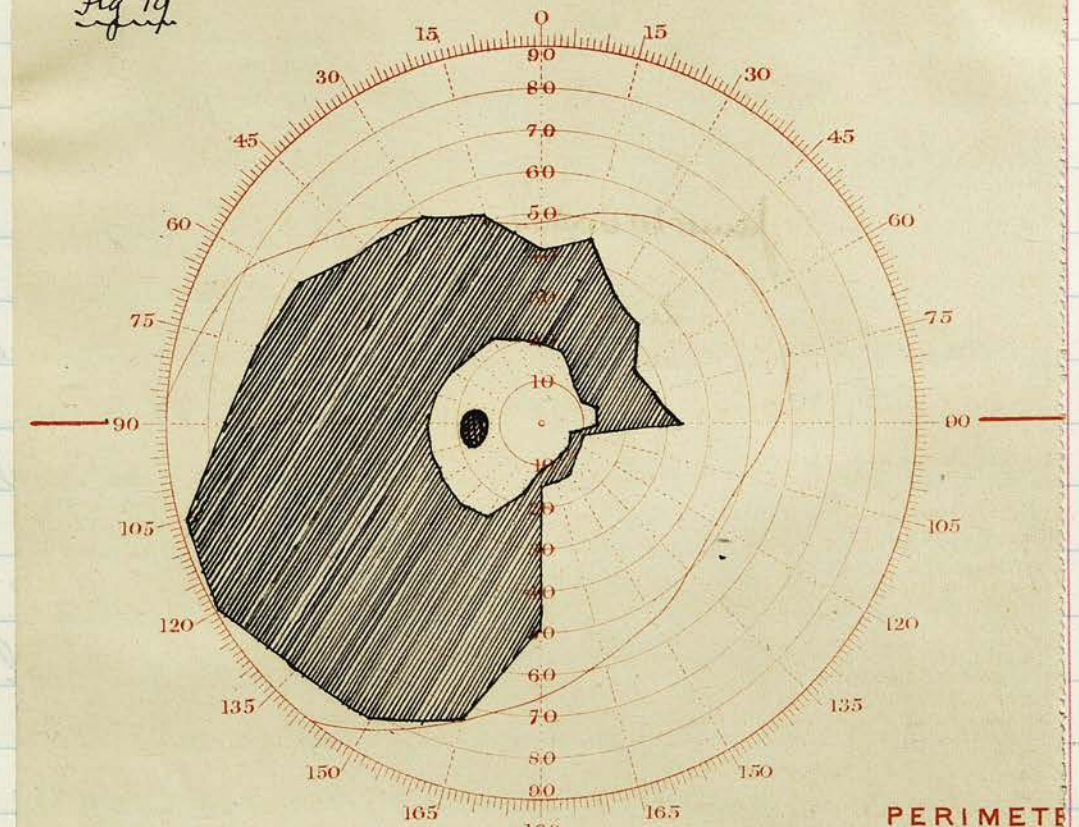

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The eccentric continuous red line indicates the average normal Fiel

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it may, however, take the form of a sector like defect or one half of the field (apart from Hemianopsia of cerebral disease) may be lost; lastly the alteration may consist in an irregular scotoma in the middle of the field.

Case 14. (fig 18) Robert McL. had all the symptoms & signs of Locomotor Ataxy & well marked Progressive optic Atrophy. His visual field by $\frac{3}{2000}$ well exemplifies the concentric limitation mentioned by Juler. In cases similar to this (Locomotor Ataxy) central vision tends to remain longest whilst in Glaucoma we usually find that it is some eccentric part of the temporal portion of the visual field that remains longest. Although I have examined many of these cases for brevity I merely mention this one to show how different the visual field is usually from that of a case of Glaucoma.

Case 15. (figs 19 and 20) James McC. van driver. Eight months previous to examination he noticed a dimness of sight in O.D. which gradually increased until the outer part of his visual field was abolished. Three months ago the same symptoms began in O.S.
7/3/90 $V = \frac{6}{18}$ partly in O.D. $V = \frac{6}{9}$ partly in O.S. No improvement with lenses. For Bjerrum's Types $V = \frac{6}{36}$ in O.D. $V = \frac{6}{18}$ in O.S. Discs pale, vessels diminished in size & slight cupping in both eyes. Colour vision very defective & the restriction is irregular & not concentric to that for white.
This case exemplifies the sector like defect that Juler refers to.

Case 16. (fig 21) James M. Tenter. Nine months ago he noticed a dimness of sight that has gradually become worse. Smoked 4 g tobacco

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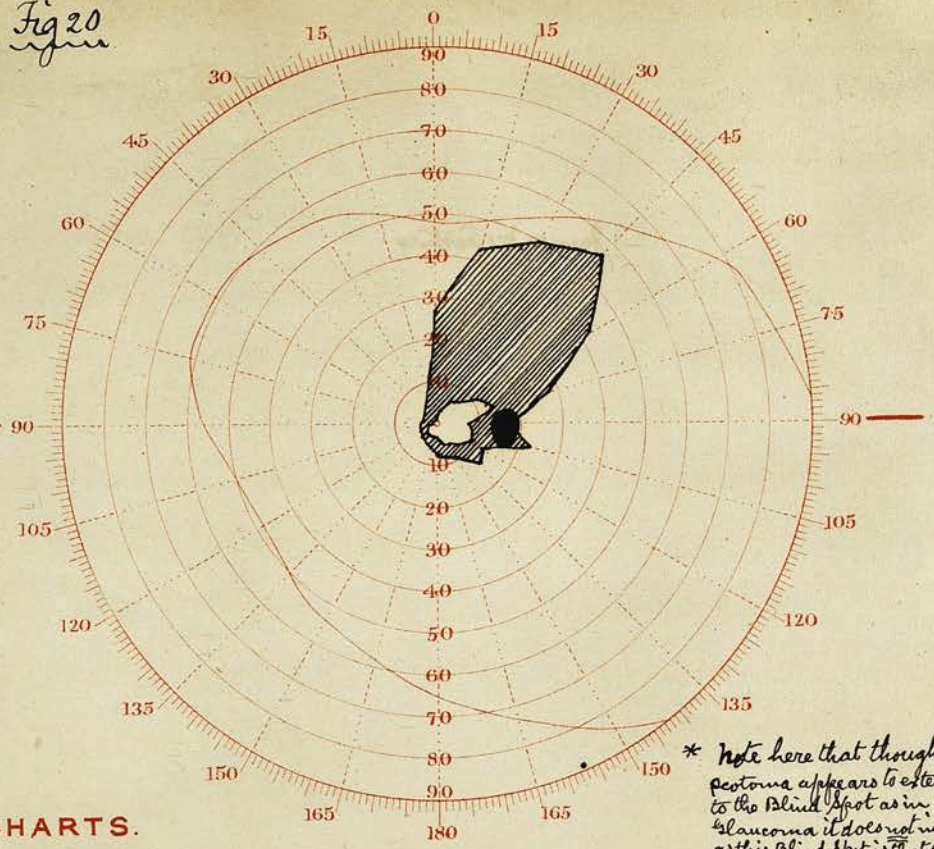
RIGHT.

Fig 20



* Note here that though the scotoma appears to extend up to the Blind Spot as in Glaucoma it does not in reality as this Blind Spot is that found by $\frac{3}{2000}$ & not by $\frac{10}{300}$ which does not extend so far outwards

R CHARTS.

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

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commencing to use the Automatic Registration".

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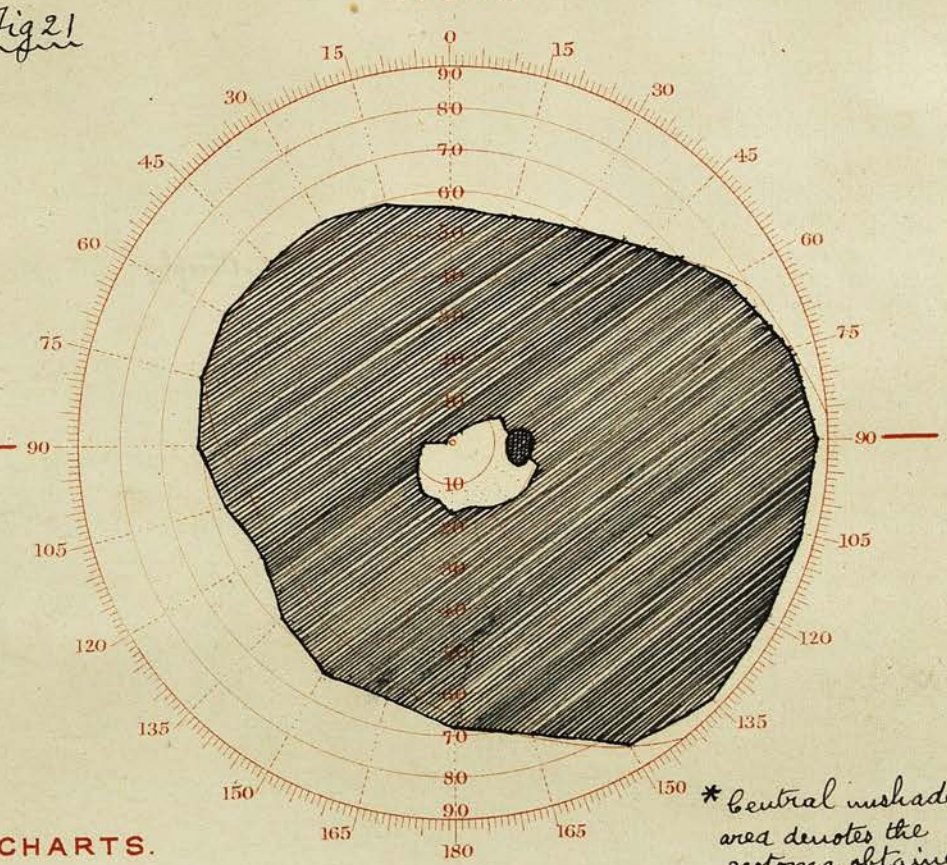
Fig 21



* Central unshaded area denotes the scotoma obtained by $\frac{3}{2000}$

ER CHARTS.

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

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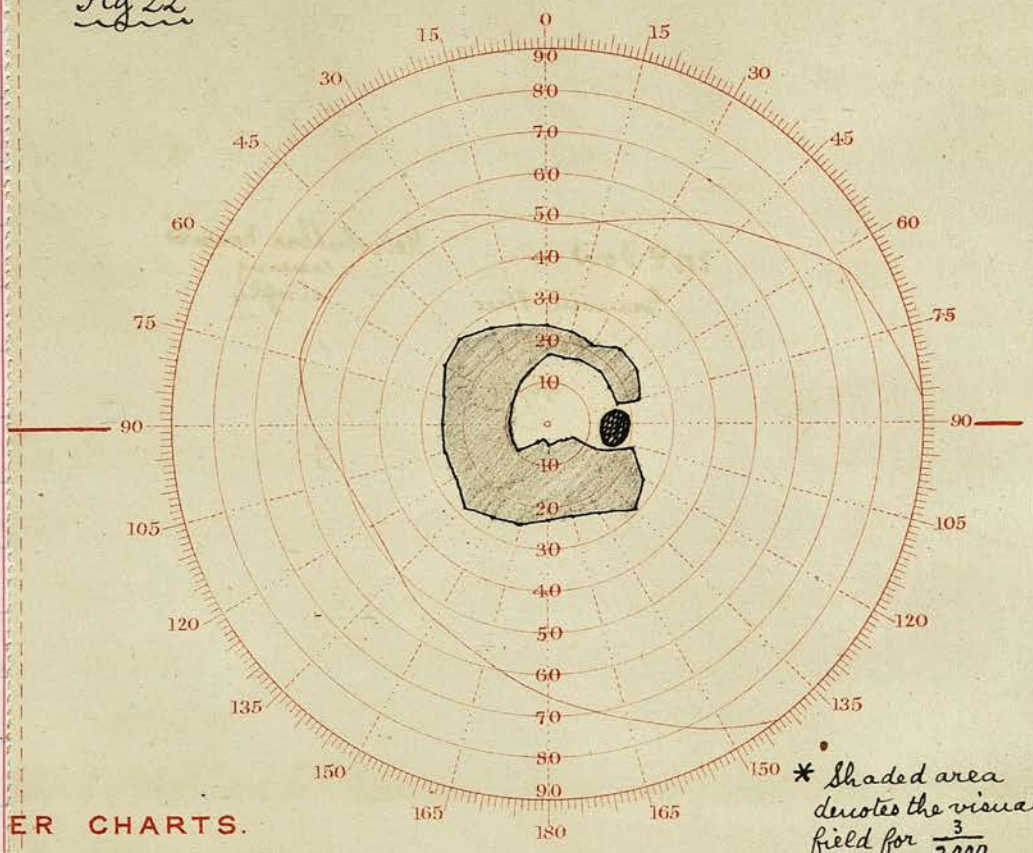
per week & also chewed. Moderate drinker but is dyspeptic & lately had much mental anxiety 14/4/90 V = $\frac{6}{60}$ in both eyes.
For Bjerrum's Types V = scarcely $\frac{1}{60}$ in both eyes. Hypermetropic No improvement with lenses. Field for blue, red & green normal on inner side of visual field but in the area of the scotoma colour perception is totally abolished. Discs very pale on the outer sides & vessels slightly diminished in size
By $\frac{10}{300}$ there is no appreciable defect but by $\frac{3}{2000}$ there is found a central scotoma well marked extending downwards to 14° from the point of fixation & 22° downwards & outwards The case has been one of Toxic Amblyopia that has gone on to Incomplete Optic Atrophy & well exemplifies the irregular scotomata in the middle of the field referred to by Juler

Case 14. (fig 22) Mrs D. Housewife. 3/4/90 Patient had noticed a dimness in O.D. for the last fourteen months but had had no pain or inflammatory symptoms V = $\frac{6}{60}$ in O.D. No improvement with lenses V = $\frac{6}{12}$ in O.S. With + 1·50 Sph, V = $\frac{6}{6}$ in O.S.
For Bjerrum's Types V = $\frac{6}{18}$ in O.S. but is nil in O.D. as when the types are within 12 in off from her face she only notices some washed out looking marks. Ophthalmoscopically the disc of O.D. is intensely hyperaemic & its margins indistinct — veins & arteries engorged — no haemorrhages & no parallactic movement. Colour vision totally abolished as colours seem gray but in O.S. colour perception is normal.
By $\frac{10}{300}$ there is nothing noteworthy but a slight restriction all round of the visual field. By $\frac{3}{2000}$ the area in which the functional activity of the retina is best maintained extends

commencing to use the Automatic Registration."

Fig 22

RIGHT.



ER CHARTS.

* Shaded area denotes the visual field for $\frac{3}{2000}$

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

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as $\frac{3}{4}$ of a circle around the papillo-macular area which is deficient It nowhere extends up to the blind spot & without it the field is everywhere defective, there being also a well marked central scotoma from defect of the papillo-macular area. The scotoma is absolute as colour vision is totally abolished within it, colours not even appearing gray. It is a well known fact that the early stages of Papillitis are as a rule unattended by impairment of the visual function as is also the case with simple Hyperaemia of the disc (Juler, Handbook of Ophthalmic Science & Practice, p. 176) therefore the dimness of vision & impairment of light sense existing here, combined with the scotoma for form & colour revealed by $\frac{3}{2000}$ place it beyond doubt that there has been a descending neuritis followed by atrophy of the optic nerve. This view is favoured by the history of long duration & slowly increasing dimness of vision & also explains why the visual area in no place extends up to the blind spot.

I introduce this case merely to show the value of this method of examination in differential diagnosis.

In Optic Atrophy the visual field for small objects is often, comparatively speaking, good when the colour vision is much reduced while the opposite condition may frequently be found in Glaucoma. The visual field here is by no means as a rule similar in form when tested with a small object as by the ordinary test owing to the defective visual acuity being more readily mapped out by the smaller size.

In Glaucoma a central or paracentral scotoma, which is not uncommon, is found to spread towards the periphery in all directions sometimes more in one than another

except outwards where it never passes beyond the blind spot thus differing from those met with in optic atrophy (primary) (vide Case 14) At all events there can only occasionally be a resemblance & then only at one stage in the development of the latter.

A third class of cases is that of "Retrobulbar Neuritis" which I bring forward here as they are closely connected to the latter class but my chief object in so doing is to point out how this method of examination differentiates these cases from Toxic Amblyopia with which they are often confounded. The characteristics of this group are dimness of vision, very slight changes in the papilla & the presence of a more or less definite central scotoma for form & colour. The changes in the papilla may be so slight as altogether to escape detection or they may amount to merely some slight haziness of its margins & perhaps a trace of perivascular inflammation. After some time the outer half of the disc may exhibit an abnormal degree of pallor whilst at the same time the contrast between it & the nasal half may be increased by some injection of the latter. At other times a general pallor of the disc results. The central scotoma has often very much the same form as that which characterises Toxic Amblyopia but it may differ from it in an important manner both in shape & extent (see cases quoted). Just as in these toxic cases it is usually negative i.e. does not give rise to a sensation of a dark spot in the visual field. Often a faint cloudiness is complained of

in a bright light while at other times a more or less dark generally buff coloured mist is complained of. The colour defect too in the region of the scotoma is the same as in Toxic Amblyopia & will be there described. Both eyes may be affected though usually only one & where it is bilateral it rarely causes an equal degree of amblyopia in the two eyes. Moreover it is about equally common in men & women whilst Toxic Amblyopia is more common among men.

The points for differential diagnosis are (1) the irregular shape of the scotoma & its extension when large to the nasal side of the point of fixation. The scotoma is not confined to the area stretching from the disc to the macula or that which is supplied by the so called papillo-macular fibres as in Toxic cases & further it is irregularly shaped. In typical Toxic Amblyopia the scotoma does not extend far (barely 2°) if at all to the inner side of the point of fixation & moreover is regularly shaped. (2) Restriction of the visual field. It is slight & usually concentric. (3) Often pain on pressure of the eye backwards into the orbit or on moving it in some particular direction (4) When bilateral both eyes are never affected in the same degree & frequently there is a history of the dimness of vision lasting some time in one eye before the other began to fail also.

What I wish to emphasize is the fact that very often the scotoma for form cannot be demonstrated by $\frac{10}{300}$ & can only be inferred from the test for the colour defect

commencing to use the Automatic Registration".

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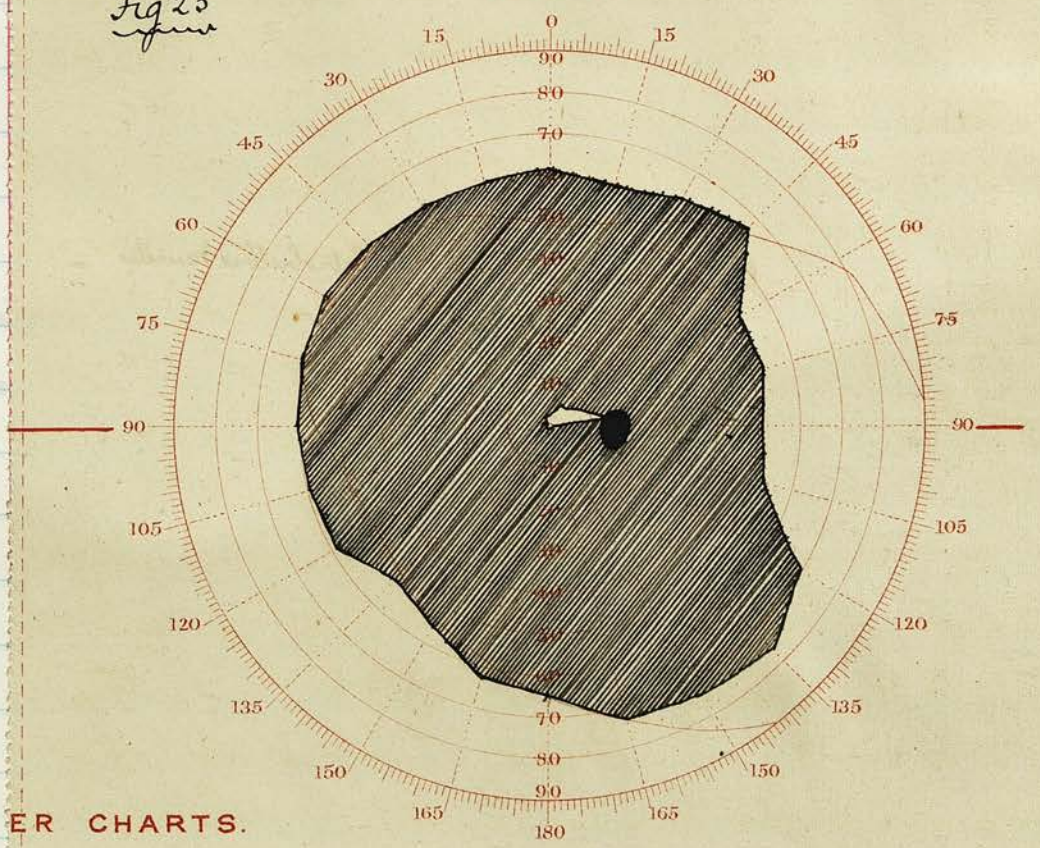
Fig 23



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

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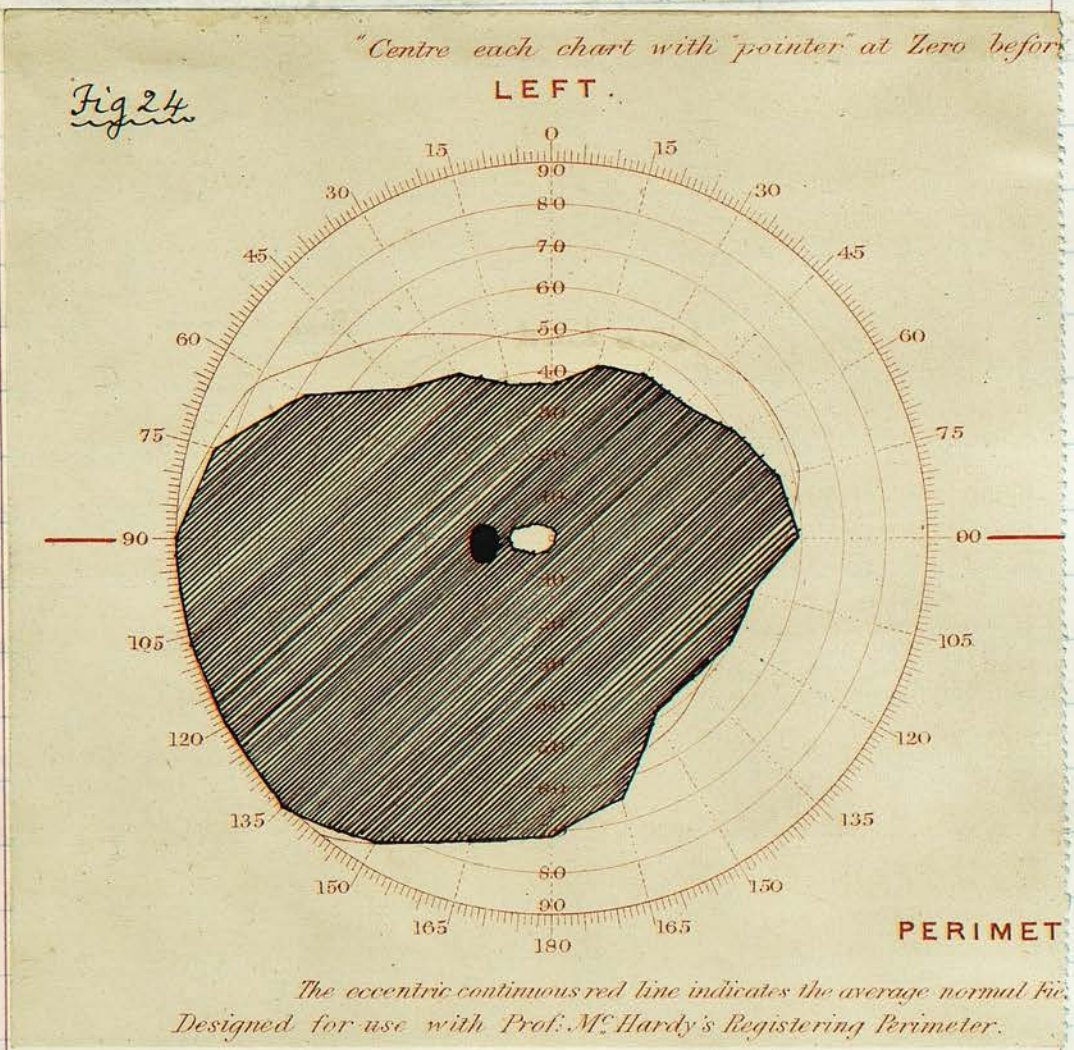
that is usually a rough one while by $\frac{3}{2000}$ the defect for form can always be demonstrated & its characters at once clear up the doubtful points in the diagnosis.

This will be readily seen on comparing the charts of the cases here quoted with those given under the head of Toxic Amblyopia. Accordingly I consider this method of immense practical value for on the one hand the prognosis in Toxic cases is good as the majority always recover good vision & the few remaining cases recover a certain amount & do not become worse whilst on the other hand the prognosis in these cases is less hopeful & should be guarded for though a certain proportion, not quite a half probably, & these the more acute & less severe cases, recover in a few weeks from the onset of the disease yet of the rest some go on to complete blindness & others are left with an absolute central scotoma & the ophthalmoscopic appearances of Optic Atrophy (see Case XIII)

Case 18. (fig 23) James D. Commercial traveller & exposed to the changes of the weather. Dimness of sight in O.D. that has lasted some years. $V = \frac{6}{9}$ in O.D. $V = \frac{6}{6}$ in O.S. For Bjerrum's Types $V = \frac{6}{36}$ in O.D. $V = \frac{6}{24}$ in O.S. — they appear more washed out looking to the right eye. Ophthalmoscopically the disc in O.D. is paler than normal & paler than that of O.S. while the retinal vessels are tortuous. By $\frac{10}{300}$ the visual field is contracted slightly on the outer side but no central defect is demonstrable. By $\frac{3}{2000}$ we can map out a central scotoma irregular in shape & running above the papillo-

Fig 24.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macular area from slightly to the inner side of the point of fixation to the blind spot. Colour perception is abolished within the scotoma but is perfect outside it except above it at which place all colours seem gray. In coming within the area of the scotoma all colours appear gray & as they approach the centre they disappear from view altogether.

Case 19. (fig 24) James L. Bath attendant. Exposed to sudden changes of temperature 31/12/89. Twelve months ago sight began to fail till three months ago he was no longer able to read Used to smoke 3 oz tobacco per week No alcoholic element $V = \frac{6}{60}$ in O.D. $V = \frac{5}{60}$ in O.S. A -2.50 Cyl Axis Vertical gives a very slight improvement For Bjerrum's Types $V = \frac{2}{60}$ in O.D. $V = \frac{1}{60}$ in O.S. Note here the difference in the degree of amblyopia in the two eyes Ophthalmoscopically the fundus appears normal By $\frac{10}{300}$ the visual field is contracted above & to the inner side slightly Elsewhere normal no central defect demonstrable. By $\frac{3}{2000}$ there is found a central scotoma for form & colour which does not extend up to the optic disc as in Toxic cases & encroaches slightly on the inner side of the point of fixation
15/3/90 $V = \frac{6}{60}$ in O.D. $V = \frac{3}{60}$ in O.S. so that it is slightly worse in O.S. Red & Green are recognized on the inner but not on the outer side of the visual field where they appear of a dark hue that becomes darker within the area of the scotoma until they totally disappear at its centre

Case 20 (fig 25) Andrew T. Sailmaker. 1/3/90 Dimness of sight existed for four months & is gradually increasing $V = \frac{6}{60}$ in O.S. For Bjerrum's Types $V = \frac{3}{60}$ Fundus apparently normal

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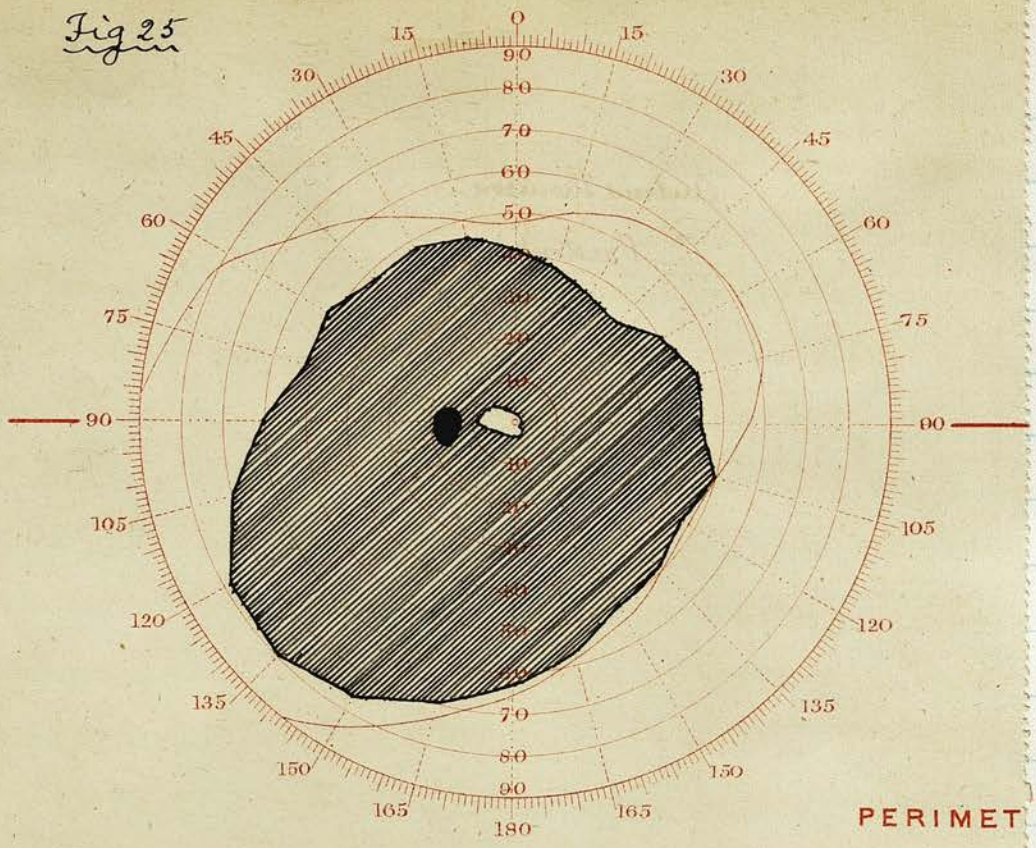
Fig 25



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

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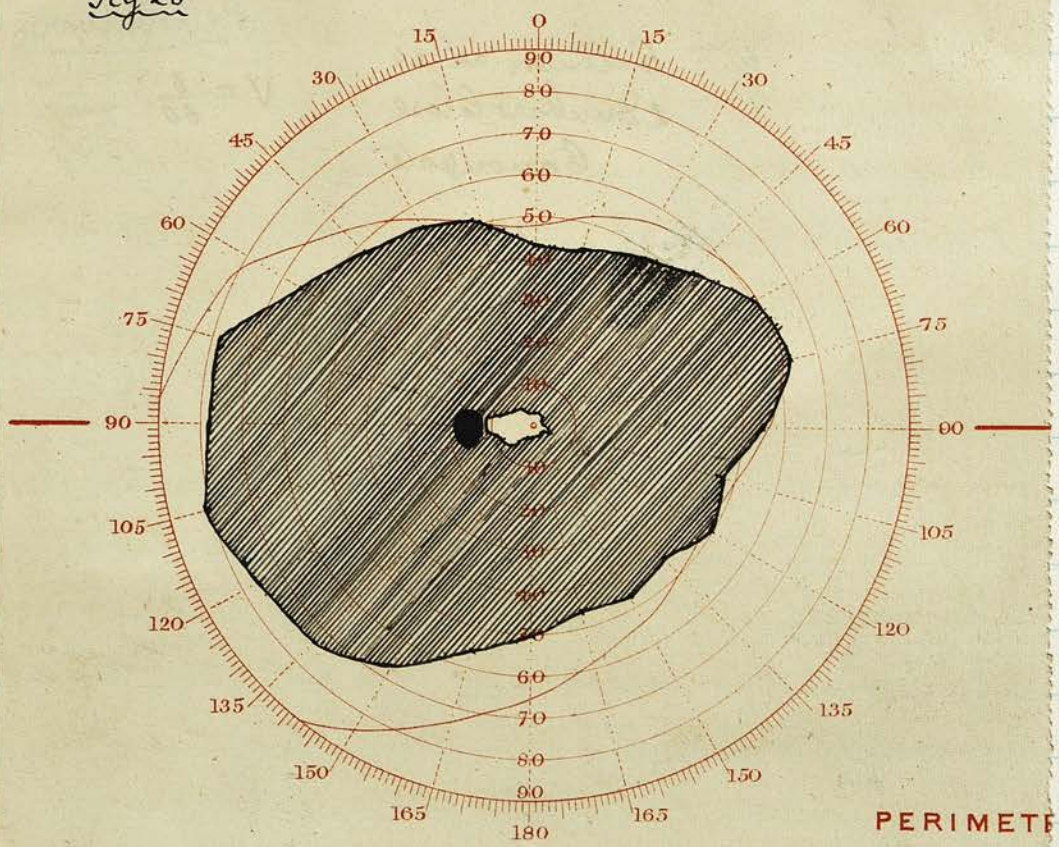
Fig 26



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By $\frac{10}{300}$ there is a slight concentric restriction of the visual field, being most marked to the outer & inner sides. Central scotoma not demonstrable By $\frac{3}{2000}$ there is found a central scotoma, irregular in shape, extending 4° to inner side of the point of fixation & though involving the macular area yet not reaching to the blind spot. Red & Green are recognized on the inner but not on the outer side of the visual field where they appear of a dark hue that becomes darker within the area of the scotoma till near its centre they disappear altogether. Blue also disappears near the centre of the scotoma & is not recognized within its boundaries while outside the scotoma it is readily distinguished everywhere. When this patient was seen four weeks later an improvement had taken place as $V=\frac{6}{18}$ in O.S. V for Bjerrum's Types still $\frac{3}{60}$

Case 21. (fig 26)

William C. Compositor. In Sept^{r} 1889 sight gradually began to fail - no pain - no inflammatory symptoms. Origin probably due to cold as he worked in a draught & had intense neuralgia at that time in face & head. No venereal taint. In Feb^{y} 1890 $V=\frac{4}{60}$ in O.D. $V=\frac{2}{60}$ in O.S. For Bjerrum's Types $V=\frac{2}{60}$ in O.D. $V=\frac{1}{60}$ in O.S. Though bilateral note the greater failure in one eye, O.S. Ophthalmoscopically in both eyes the discs are very pale & the vessels diminished in size In O.S. there are signs of peri-vascular inflammation as we see a white tag coming from the centre of the disc, bifurcating & running upwards & downwards on the retinal vessels. By $\frac{10}{300}$ the visual field is diminished all round slightly except on the inner side. There is a suspicion of a central scotoma as the test object is very dimly seen near the fixation point (a partial scotoma)

By $\frac{3}{2000}$ there is a well marked absolute central scotoma that extends 5° to the inner side of the fixation point & also reaches the blind spot & is slightly irregular in outline. Green is not recognized as such on either side of the visual field but appears of a dark gray colour that is darker on the inner side of the field & within the area of the scotoma disappears near its centre. Red & Blue are recognized on both sides of the field outside the scotoma but are darker on the inner side They are not recognized within the scotoma & appear of a darker hue as they approach towards its centre where they are totally lost This colour defect is suggestive of Optic Atrophy.

The fourth class of cases is that of "Toxic Amblyopia" with which as already stated the latter are often confused. It is a common affection & the symptoms are a gradual failure of sight to much the same extent in both eyes, absence of any restriction of the visual field or indeed of any interference with the functions of the peripheral portions of the retina; & the existence of a negative oval scotoma for form & colour (most marked for the latter) extending from the point of fixation, which it involves, to the blind spot. Most cases occur at or beyond middle life though some occur in young folks. It is rather rare among women as most cases are men. Those suffering from this disease are almost invariably smokers & generally they have smoked for many years before becoming affected while many also chew tobacco. The exceptions to this

occur chiefly in a very few patients in whom a similar kind of amblyopia is hereditary, is liable to affect the female as well as the male members & may come on much earlier in life, the etiology of such cases being obscure & in some few of them there being no evidence of heredity (Nettleship, Diseases of the Eye, p 235) Probably they are due to optic neuritis as we usually see evidence of well marked perivasculitis (Berry, Diseases of the Eye, p 308)

The amount smoked is usually not less than 3 oz or 4 oz per week & often much more though in women a smaller quantity is sufficient. Usually when the affection begins no change has recently been made in the amount smoked & so the cause is seldom suspected by the patients who indeed often smoke more on account of the worry caused by failing sight. They often smoke on an empty stomach first thing in the morning or else late at night or during the night in the case of bad sleepers. Hence the reason why it is more common in the working than in the educated classes. Alcohol is also said to be a cause & certainly many drink to excess yet the same symptoms never occur on excessive use of alcohol alone while many cases are total abstainers (vide Cases XXII, XXIII, XXIV, XXVIII). Probably alcohol acts indirectly by undermining the constitution. The fact of tobacco gaining the upper hand at some particular time though the patient has smoked as much & often more at other times points to there being some recent

diminution in the power of resistance though often it is not easy to discover it. Sometimes it is slight indisposition, dyspepsia, loss of blood, sleeplessness or anxiety & trouble while often it can only be ascribed to loss of nerve tone or some senile loss of vigour. The poison is more apt to take effect when any predisposing cause is present if indulged in at a time when there is no counter stimulus of food. Differences exist also in individual tolerance of tobacco also. The scotoma is negative i.e. the patient is not conscious of a limited defect in his field of vision, & it is oval in shape with its long diameter horizontal & extends from the outer side of the blind spot to slightly to the inner side (barely 2°) of the point of fixation. It corresponds to the external projection of that portion of the retina lying between the optic nerve & the outer margin of the macula & which is supplied by a special bundle of nerve fibres, the papillo-macular bundle, as shown by recent investigations. The fibres in this papillo-macular bundle are central at the hinder end of the nerve but lie on its outer (temporal) side close to the eyeball (vide Transactions of the Ophthalmological Society, Vol III, p 174)

Within the scotoma the form sense is abolished as well as the colour sense, the defect being most marked for red & green. It is interesting here to note the relation between the form & colour sense. When testing the form sense with a small white object as soon as you pass within the margins of the scotoma, the object is at once

lost to view while on the other hand the colour is at once unable to be distinguished but appears of a gray or neutral tint that becomes dimmer as it approaches the centre of the scotoma where it disappears totally, this being the area of greatest saturation. This fact is well brought out in the cases recorded in this paper & the only mention I have seen of it in books is by Dr. Berry in the Ophthalmic Review, Vol IX, p 109 whose observations I am thus able to confirm. The scotoma for form can usually not be demonstrated by $\frac{10}{300}$ so they fall back on the test for colours in order to map out the area in which the colour sense is deficient. I wish to emphasize the fact that by this method, however, you can always map out the scotoma both for form & colour when its extent & regularity in outline will at once differentiate it from that of a case of Retrobulbar Neuritis. When a case, originally one of this nature, goes on to genuine neuritis the scotoma will be found to extend its boundaries & become irregular in outline. These cases (Toxic) often complain of seeing worse in strong light, owing apparently to some existing hyperaesthesia of the retina. They see white letters on a black background better than ordinary black ones on a white ground. This is due to the defect in light sense which always exists in Toxic cases for patients are unable to perceive slight differences in intensity as tested by Bjerrum's Types while the minimum amount requisite to produce a

Fig 24

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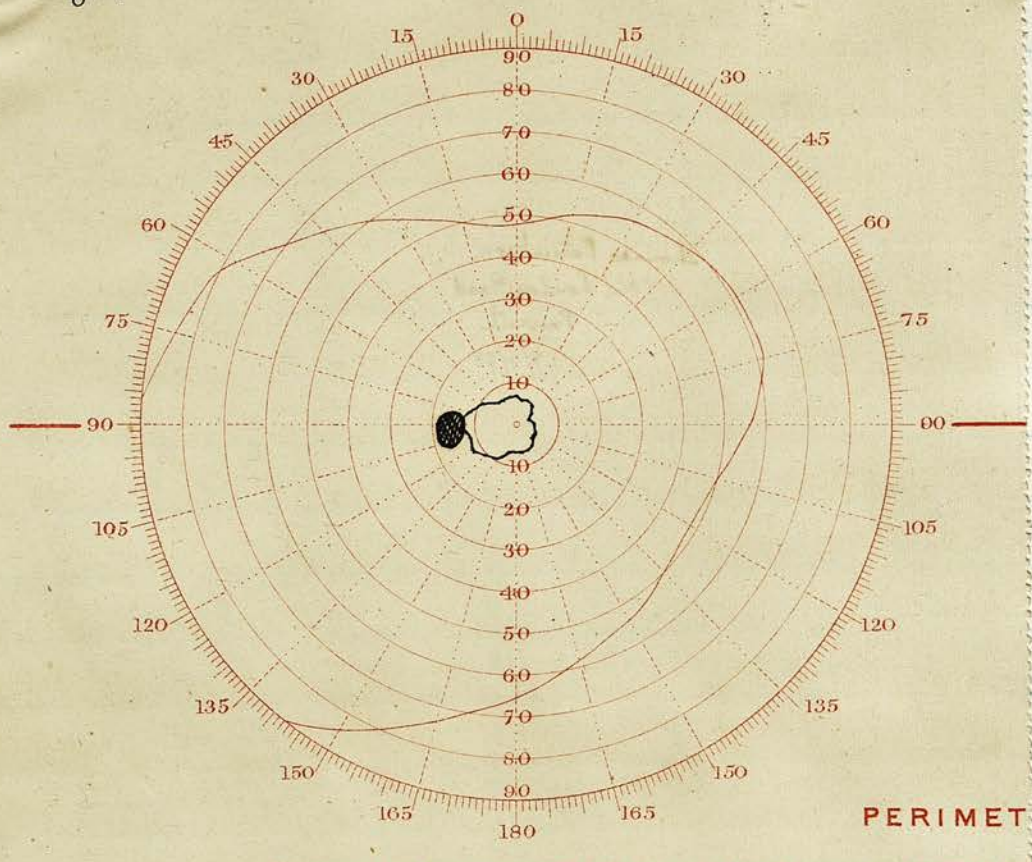

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sensation of light is not perceptibly lower than normal (see cases quoted). As the patient recovers the scotoma for form disappears & he may be able to read $\frac{6}{9}$ Snellen but still this defect in light sense & colour sense remains. (see Case XXIX).

Case 22. (fig 27) Thomas P. Millwright. 7/4/90 Dimness of sight lasting fifteen months but since New Year has increased almost daily No alcoholic element. Smokes 4 oz tobacco per week & also chews Had much mental anxiety lately. V = fingers counted at 12 ft No improvement with lenses For Bjerrum's Types V = $\frac{1}{60}$ & they are very washed out looking. Discs a little pale but not beyond the normal - well marked physiological cup - fundus normal. A well marked central scotoma for form & colour. Red distinguished all over the field except in the area of the scotoma & it is brighter on the inner than on the outer side of the field Within the scotoma it appears of a dark hue & gradually deepens as you approach its centre where it totally disappears. Green appears red all over the field, being brightest on the inner side of the field Within the scotoma it becomes a dark hue that deepens towards the centre where it vanishes altogether. Blue is distinguished on the inner side of the field but not on the outer where it seems a dark colour that becomes darker within the scotoma till near the centre it also disappears.

Case 23. (fig 28) Joseph H. Finisher in dye works. 7/4/90 Dimness of sight has gradually come on during the last three or four months No alcoholic element. Smokes 3 oz tobacco per week V = fingers counted at 16 ft Hypermetropic No improvement

Fig 28

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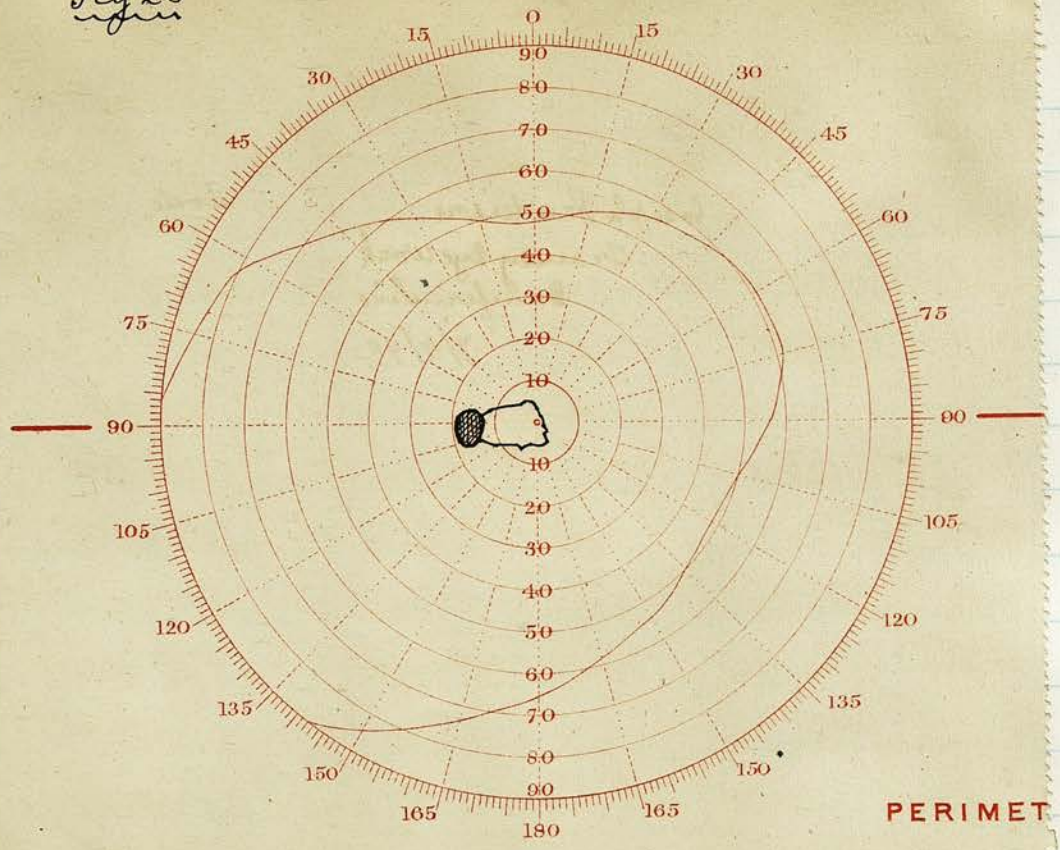

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Fig 29

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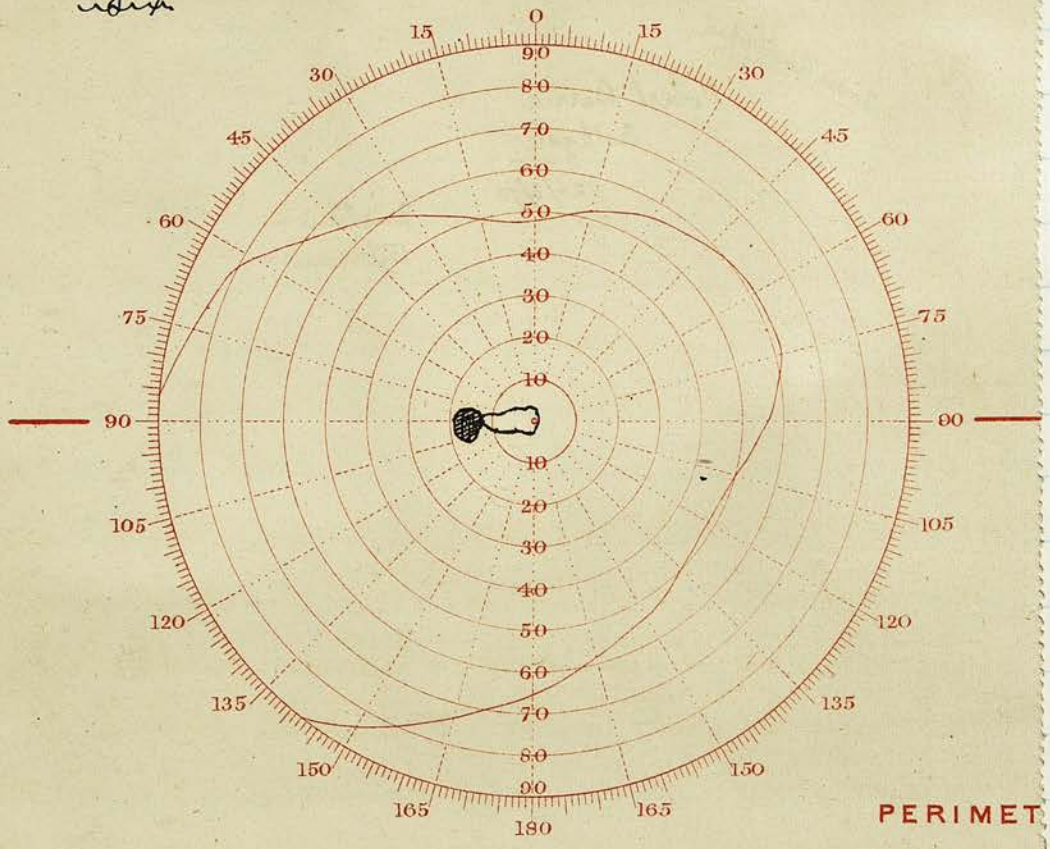

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with Lenses. V for Bjerrum's Types = $\frac{1}{36}$ Discs healthy & well marked scleral rings - fundus normal. Central scotoma for form & colour. Red & green distinguished all over the field, being brighter on the inner than on the outer side Within the scotoma they appear as a dark tint that deepens as they approach the centre where they totally disappear Blue is recognized all over the field, being brightest on the inner side. Within the scotoma it wholly disappears throughout its whole extent.

Case 24 (fig 29) Robert B. Miner. At new Year his sight gradually began to fail & continued to steadily deteriorate. At this time while still weak from the effects of an accident he had just resumed work. No alcoholic element. Smokes 3 oz tobacco per week. V = $\frac{6}{36}$ in both eyes. Hypermetropic With +1.50 Sph ○ +1.0 Cyl Axis Vertical he gets $\frac{6}{18}$ both eyes & with +4.50 Sph reads No 3 J. For Bjerrum's Types V = $\frac{2}{36}$ in both eyes Discs & fundus normal. A central scotoma for form & colour. Blue, red & green are all recognized over the whole field without the scotoma, being brightest on the inner side but becoming of a dark tint within the scotoma & this tint gradually deepens until near the centre the colours are totally lost to view.

Case 25. (fig 30) Alexander W. Sailor. 21/3/90. Sight began to fail four months ago until he could not tell friends across the street. A history of syphilis & malaria. Smokes 1 oz tobacco daily & drinks heavily V = $\frac{6}{36}$ in OD. V = $\frac{6}{24}$ in OS. Myopic astigmatism With -2.50 Cyl Axis Horiz = $\frac{6}{12}$ in both eyes

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Fig 30

RIGHT.

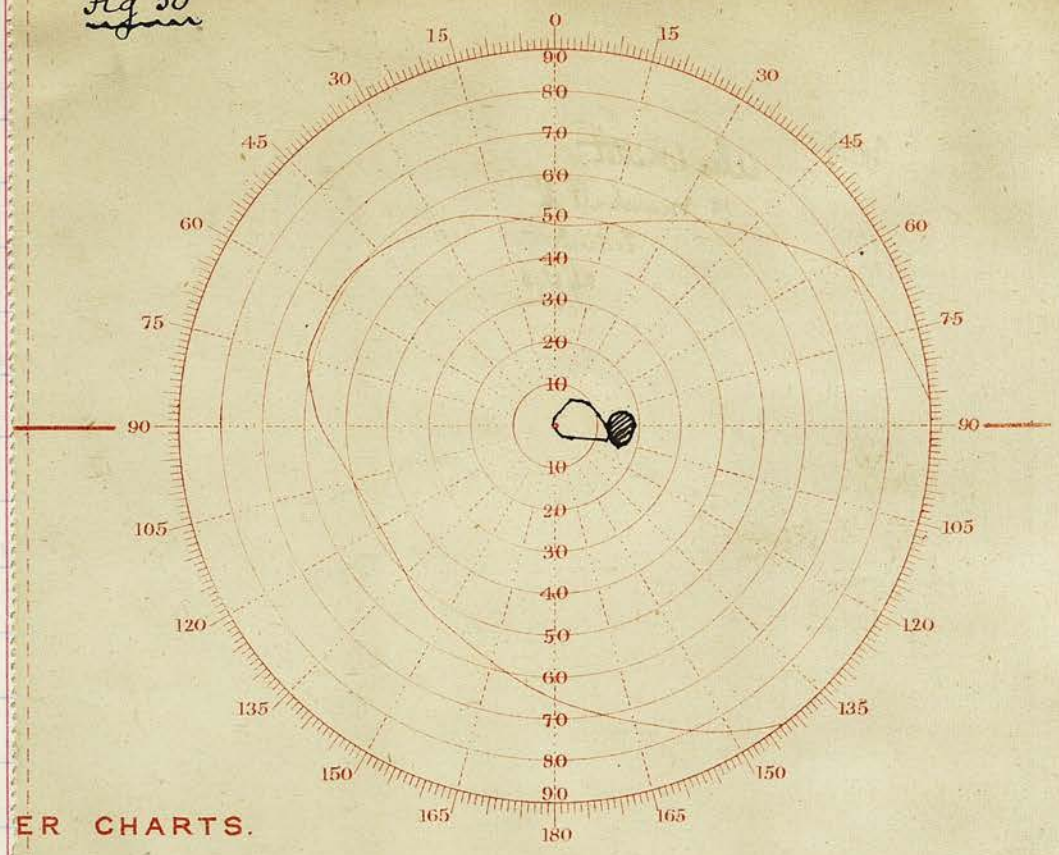

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Fig 31

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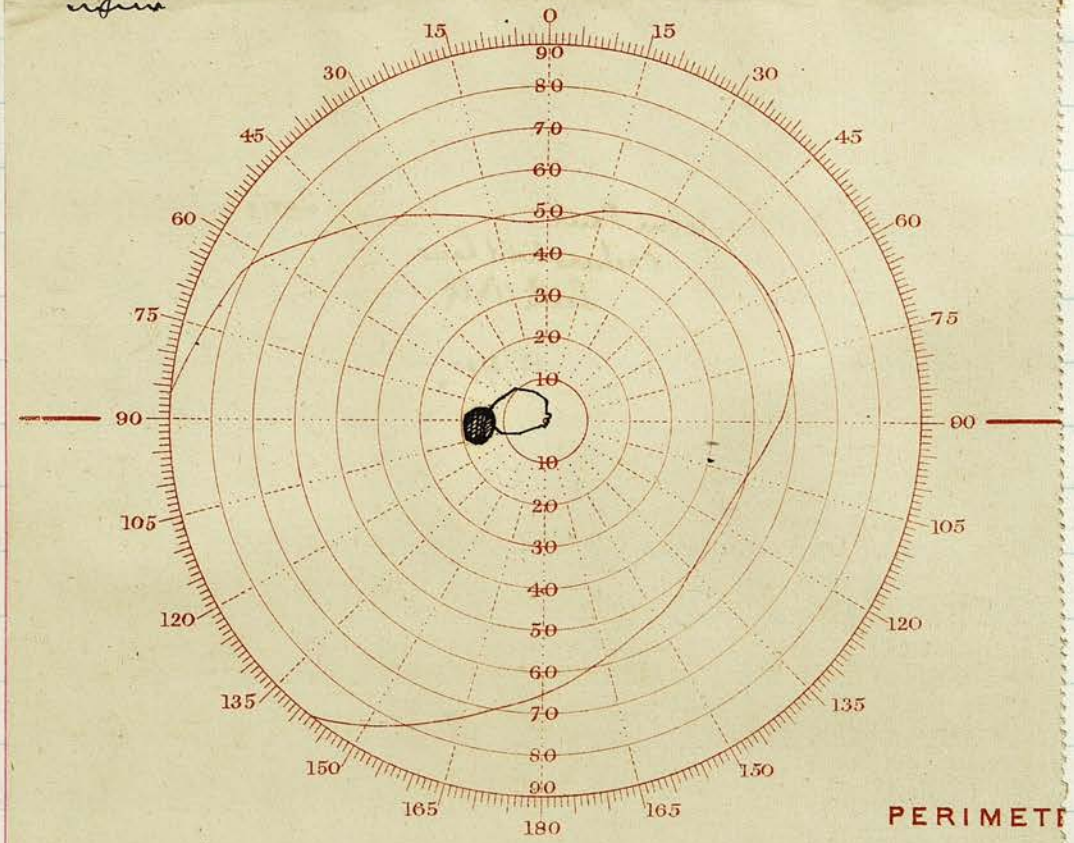

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& reads No 4 J. Fundus normal & venous pulsation well seen on the discs. For Bjerrum's Types $V = \frac{2}{60}$ in both eyes. A central scotoma for form & colour. Blue, red & green are all distinguished on the inner side of the field but not on the outer where they appear of a dull tint that becomes darker within the scotoma as they approach the centre until near the centre they vanish altogether. When I saw the patient three or four weeks later his vision was much improved, being $\frac{6}{18}$ in both eyes. I had no opportunity to examine the scotoma

Case 26. (fig 31) John P. Riveter. In March 1890 after a good deal of family trouble, sight began to fail. Drinks heavily & smokes 4 oz tobacco per week Fundus apparently normal. $V = \frac{6}{60}$ in both eyes Lenses effect no improvement. For Bjerrum's Types $V = \frac{1}{60}$ in both eyes A central scotoma for form & colour. Blue, red & green are distinguished all over the field, being brightest on the inner side. They become of a dark tint within the scotoma & darker as they approach the centre where they wholly disappear. He had a so called Coloboma of the optic nerve sheath in both eyes.

Case 27. (fig 32) Alexander A. Clerk. In middle of March 1890 a dimness of sight came on suddenly & increased daily. Is dyspeptic Moderate drinker. Never smokes less than 3 oz tobacco per week. $V = \frac{6}{60}$ in both eyes Hypermetropic No improvement with lenses For Bjerrum's Types $V = \frac{2}{60}$ in both eyes. Fundus apparently normal. Central scotoma for form & colour.

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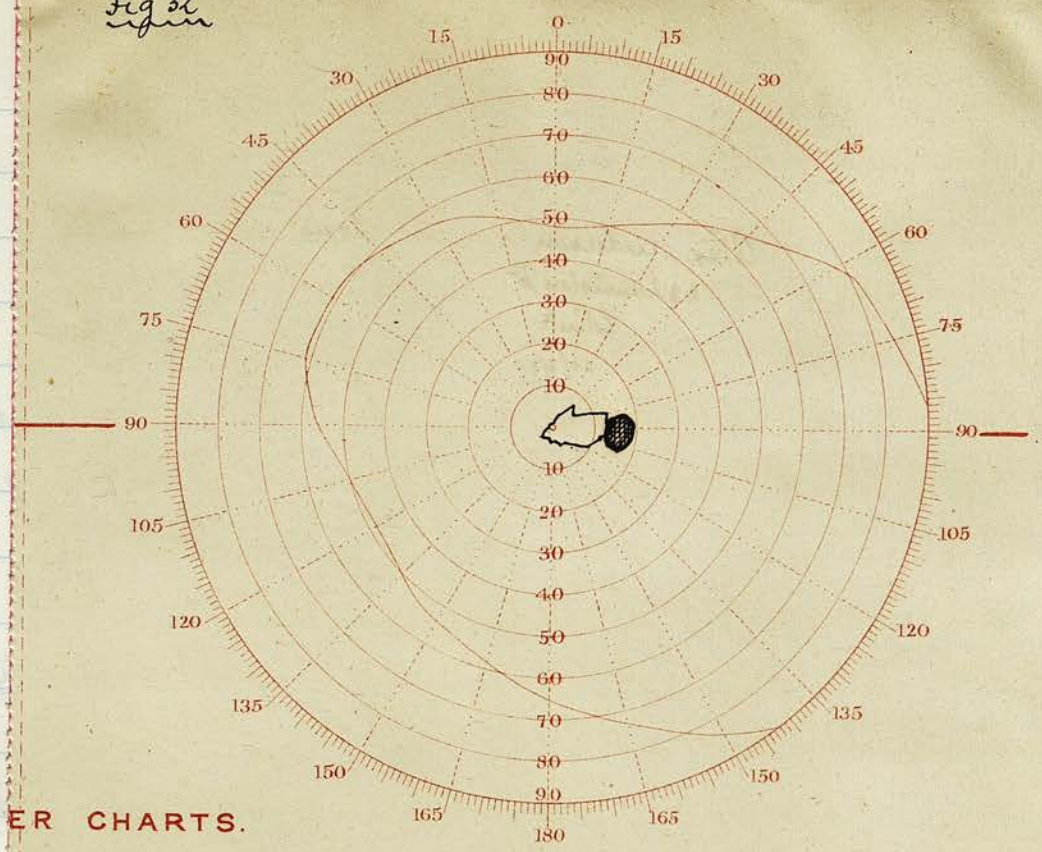
RIGHT.

Fig 32



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

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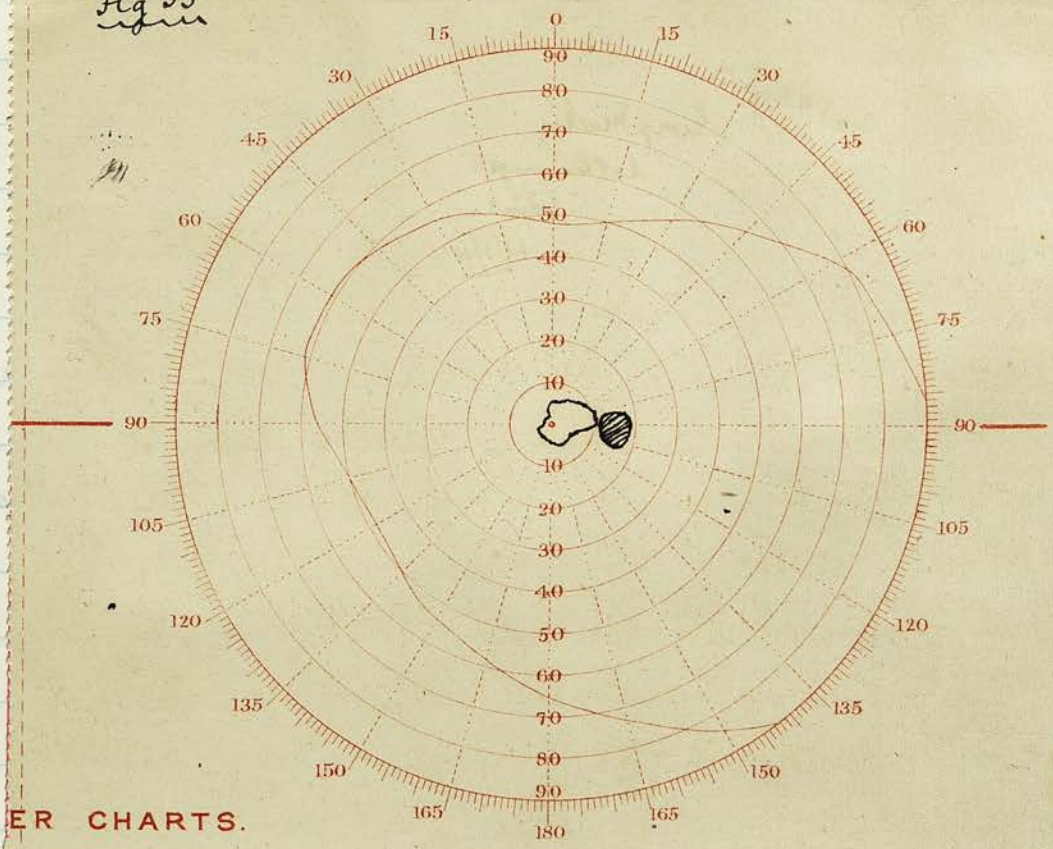
Fig 33



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Blue, red & green distinguished over the whole field except in the scotoma & are brightest on the inner side. Red appears orange just around the margins of the scotoma while green appears yellow at the same place. Within the scotoma they all appear of a dark hue that deepens as they approach to the centre where they wholly disappear.

He stopped smoking & began tonics & turkish baths & on examination a week afterwards no scotoma for form was able to be detected even at a distance of 3 and 4 metres though the light & colour senses still remained defective.

Case 28. (fig 33) George McK. Joiner. Has had Diabetes for fifteen months but is slowly improving. 21/3/90 Six weeks previously he noticed a mist before his eyes that slowly increased until he could not see to do his work. Smokes 4 oz tobacco per week & also chews. Does not drink. He is carefully dieted but sleeps badly. $V = \frac{4}{60}$ in both eyes No improvement with lenses For Bjerrum's Types $V = \frac{1}{60}$ in both eyes. Discs a little paler than usual but the fundus is otherwise healthy. Central scotoma for form & colour. Blue, red & green are distinguished over the whole field outside the scotoma, being brightest on the inner side. They assume a dull tint within the scotoma & this gets duller as they approach the centre where they wholly disappear.

These cases of Diabetes with central scotomata often have the latter becoming permanent & post mortem neuritis & degeneration of the papillo-macular bundle of

nerve fibres is found. This is probably due to the fact that the tissues have their vitality so lowered in Diabetes that continued irritation, due to the influence of a poison upon them, is sufficient to set up inflammation & death of the parts affected. In the Transactions of the Ophthalmological Society, Vol III, p. 176 it is stated that "of double equal amblyopia in non-smoking diabetics' 'no indisputable case seems to have been put on record" while again on p. 178 of the same volume they urge that "it may eventually appear that Diabetes acts' 'only as one of many other influences in predisposing' 'those who smoke to that particular disease of the' 'optic nerves of which central amblyopia is the clinical' 'expression".

Case 29. David H. Lumper. Had been affected for nearly three months & it began as a dimness of sight that gradually increased. Smoked heavily & was a moderate drinker. Also chewed. When first seen he had very defective vision & a well marked central scotoma for form & colour In the first week of April 1890 V = $\frac{6}{9}$ in both eyes. Hypermetropic slightly. With +0.50 Sph obtained $\frac{6}{6}$ in both eyes & with + 3.50 Sph read No 1 J Fundus normal' For Bjerrum's Types V = $\frac{4}{36}$ in both eyes. There was at this time no scotoma for form to be detected on a most careful examination. Blue, red & green were recognized all over the field except in the papillo-macular area, being brightest on the inner side. Within the papillo-macular area they became a dull tint that deepened till at the centre

they wholly disappeared. I introduce this case to show that though there may be no defect in the form sense & the patient be able to see $\frac{6}{6}$ Snellen yet the defect both for the light sense & colour sense still remains & takes much longer to be recovered from.

That this form of central scotoma is <u>due to the toxic influence of tobacco</u> I am certain from the following points:- (1) The remarkable frequency of this affection in smokers. (With few exceptions as already mentioned almost all are smokers while many also chew tobacco. (2) The comparative rarity of this scotoma in cases of other poisons or diseases. Carbon Bisulphide is the only other known poison that produces the same results while Diabetes is about the only disease in which it is found & even then it is rare & as already stated the patients are usually smokers (3) The curious selection of the seat of lesion which is always confined to the papillo-macular area (4) The fact that while one part is affected, another is at the same time in a state of healthy activity. Thus while the macular area of the retina is affected & direct vision impaired, the peripheral portions are unaffected & indirect vision is as good as normal there being no restriction of the boundaries of the visual field. (5) The affection does not occur at once but comes on slowly (6) The symptoms come on when the general vitality is slightly below par e.g. in dyspepsia, after mental

anxiety, sleeplessness &c & in elderly people
(7) Pathological changes have been observed in some cases in the nerve bundles (8) The symmetry of the affection. It is extremely rare if it ever does occur unilaterally. (9) Spontaneous recovery is the rule This usually occurs on stopping the tobacco, a fact which is strongly suggestive of the gradual weakening & final disappearance of a toxic effect. Moreover in some cases we get recovery without any change in the habits as regards tobacco. (10) Analogy. Vapour of Carbon Bisulphide when inhaled cause central scotomata & if long continued leads to haze & pallor of the discs.

There are <u>three situations</u> in which the lesion may be situated (1) The peripheral nerve terminations. There is no evidence whatever in favour of this & moreover Nicotin in the amounts in which it is probably absorbed into the system tends rather to excite than to depress the activity of sensory nerves.
(2) The nerve trunks. Degenerations have been seen in these but owing to the fact of cases of Toxic Amblyopia usually recovering it is very difficult to obtain post mortem examinations The fact that recovery is the general rule is rather against this idea as well as the nature of the central scotoma which as I have already shown differs widely from that in cases of neuritis
(3) The central terminations in the brain. This is the

most likely situation though anatomical evidence is wanting owing to the rarity of post mortem examinations. The clinical evidence however rather favours this view. Thus the symmetrical distribution, the equal degree of the amblyopia in both eyes, the absence in most cases of any pathological changes in the retina or optic nerve, the nature of the colour blindness, the regularity of the scotoma, the frequent toxic etiology & the curative nature of the amblyopia all distinctly point to a central origin.

The exact nature of the lesion is a matter of dispute & has been ascribed to (1) neuritis or else (2) to some functional disturbance

(1) That it is a partial neuritis. Thus Nettleship, Diseases of the Eye, p 236 states that "there is reason" "to believe the disease depends on a chronic inflam-" "mation of the central bundles of the optic nerve" "beginning at, or a short distance behind, the eye" & refers to cases quoted in Transactions of the Ophthalmological Society, Vol I. p 124 & Vol III, p 160. The great tendency in chronic inflammation of nerves is to go on to the formation of fibrous tissue in them & to cause permanent & often great interference in function through atrophy of the nerve bundles as is frequently seen in cases of Neuritis & Retrobulbar Neuritis. The great tendency to recovery in this disease in the majority of cases, the regular nature of the scotoma as to size & shape, the all but

invariable equality in the degree of amblyopia in the two eyes & the absence of any peripheral limitation of the visual field all tend to negative this view of neuritis as already described under the heading of Retrobulbar Neuritis. Moreover some rare cases occur in which recovery takes place although there has been no change in the habit of smoking & to these the Neuritis theory is scarcely applicable. More probably they are to be explained on the assumption of some functional disturbance occasioned by some lowered condition of the vitality of the system allowing the toxic influence of the tobacco to get the upper hand & which has been so far recovered from as to enable the individual to throw off the poison, notwithstanding the cumulative action which has taken place.

Further cases such as No 27 (Alexander A. p 47) can scarcely be explained as being due to neuritis as within a week the scotoma for form had entirely disappeared whereas neuritis cases as a rule are slow in recovery & according to Juler simple hyperaemia does not occasion any diminution of functional activity in itself.

(2) <u>That it is a functional disturbance.</u> This has been ascribed to vaso-motor agency & certainly Nicotin causes contraction of vessels & we know that ischaemia of the retinal vessels is able to produce amblyopia while the opposite condition has no tendency to do so. Moreover cases like No 27 already noticed rather

favour this view. However as we have here a cumulative action of the poison this contracted state of the vessels will tend to go on to dilatation since Lauder Brunton in his work on Pharmacology, Therapeutics & Materia Medica, p 282 states that Nicotin although it temporarily contracts vessels yet passes on to cause dilatation of them either by direct action on the vessel walls or by peripheral vaso-motor mechanisms within them becoming paralysed & this probably is further aided by paralysis of that part of the vaso-motor centre presiding over the blood vessels supplying the central terminations in the brain of the papillo-macular nerve fibres for Landois & Stirling in their Textbook of Human Physiology, Vol II, pg 58, state that Nicotin first stimulates & then paralyses the vaso-motor centre. Thus this view although probably accounting for a few cases such as No 24 in this paper & possibly the early stages of others yet will hardly hold good when dilatation of the vessels is likely to have occurred from the cumulative action of the poison.

Lauder Brunton in his work already referred to pp 68-69-70 & 72 states (a) that protoplasm possesses the power of taking up oxygen & assimilating it to itself & is also able to take up oxygen & give it off to other substances and (b) that Nicotin diminishes this oxidation power of protoplasm & further (c) that Nicotin also diminishes the oxidation processes in

the blood. i.e. it diminishes the amount of oxygen absorbed & the amount of carbonic acid given off in the blood. Thus it is extremely probable that Nicotin acting on the central terminations in the brain of the nerve fibres diminishes the oxidation power of the protoplasm of the nerve cells & this is aided by a diminution in the oxidation processes in the blood. The effect of this will be to lower the nutrition of these cells & hence diminish their functional activity which effect is also doubtless aided by the contraction of vessels that occurs at first due to the stimulation of the vaso-motor centre & of the vessels themselves as already mentioned Moreover although we do not know from what degree of degeneration it is possible for tissues to recover we can draw a distinction between primary alteration of structure & degeneration produced by the temporary action of a poison. Is it not more probable that the latter may be recovered from entirely than the former?

This view also explains the degenerations that have been found in the nerve trunks, leading to incomplete optic atrophy & permanent scotomata for it is very probable that if the impaired nutrition of the nerve cells of the central terminations in the brain be not relieved a degeneration of them is set up that causes a descending degeneration of the nerve fibres as the trophic influence of their centres

upon them is thus cut off. The general tendency to recovery & its gradual manner, the regular shape & extent of the scotoma, the absence of peripheral restriction of the field, the symmetry & the equality of the degree of amblyopia in both eyes all tend to favour this view which I consider to be the correct one.

A fifth class of cases to which I wish to refer is that of "Central Senile Choroiditis" which too often are confounded with Toxic Amblyopia.
Like the latter this disease begins with dimness of sight gradually increasing so that visual acuity is often reduced to $\frac{6}{60}$ or less & the presence of a central scotoma for form & colour while the boundaries of the visual field are about normal in extent.
Here however there is metamorphopsia, either micropsia, when recent, due to the exudation separating the retinal end organs so that a fewer number are stimulated than normal or else Macropsia from approximation of the retinal end organs due to shrinking in old cases so that more are stimulated than normal. This defect is often unnoticed due probably to the fact that the disease may be symmetrical & the patient not of an observant nature.
Ophthalmoscopically in the early stages the defect is so slight as to be often overlooked, the appearances being those of a reddish or reddish yellow patch, usually irregularly

oval in shape & often differing very slightly in colour from the rest of the fundus but always presenting a very definite outline & occupying the region of the macula. Later the patch assumes a more atrophic aspect & by the erect image we see numerous little sharply defined white dots while the edges become more irregular & bordered by pigment. It usually appears in both eyes & is as a rule hardly as large as the papilla though it may be larger.

Thus owing to the facts that metamorphopsia is often unobserved & that the ophthalmoscopic appearances are slight & often overlooked, that the disease is often symmetrical & the boundaries of the visual field normal, that visual acuity slowly deteriorates & that there is a central scotoma for colour (that for form not being detected by $\frac{10}{300}$) these cases often pass for Toxic Amblyopia. This method ($\frac{3}{2000}$) however always brings out well the scotoma both for form & colour & its nature will then at once arouse attention as it is confined to the area immediately surrounding the point of fixation & never involves the whole papillo-macular area or extends up to the blind spot.

This will at once lead you to test for metamorphopsia & to examine the fundus carefully by the direct image.

The value of this is great as the <u>prognosis</u> in the two classes of cases is so vastly different. Thus in Toxic cases the majority recover perfect vision while here central fixation is eventually abolished though

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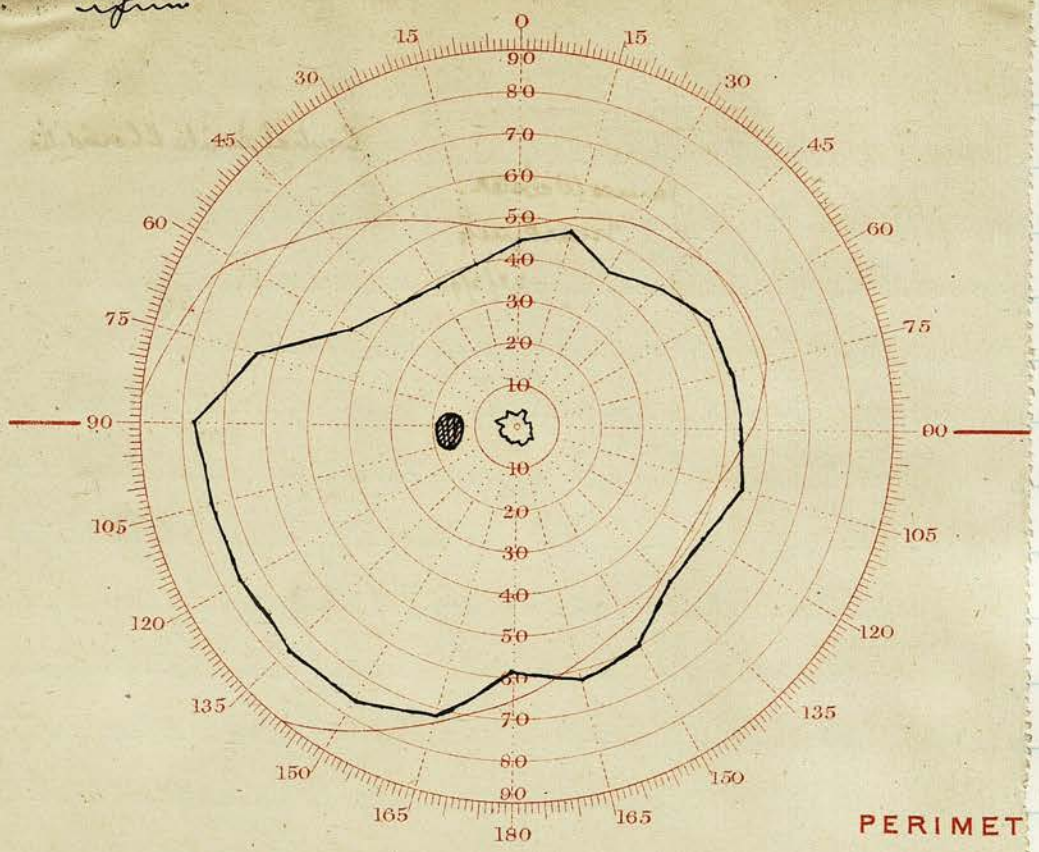
Fig 34

LEFT.



PERIMET

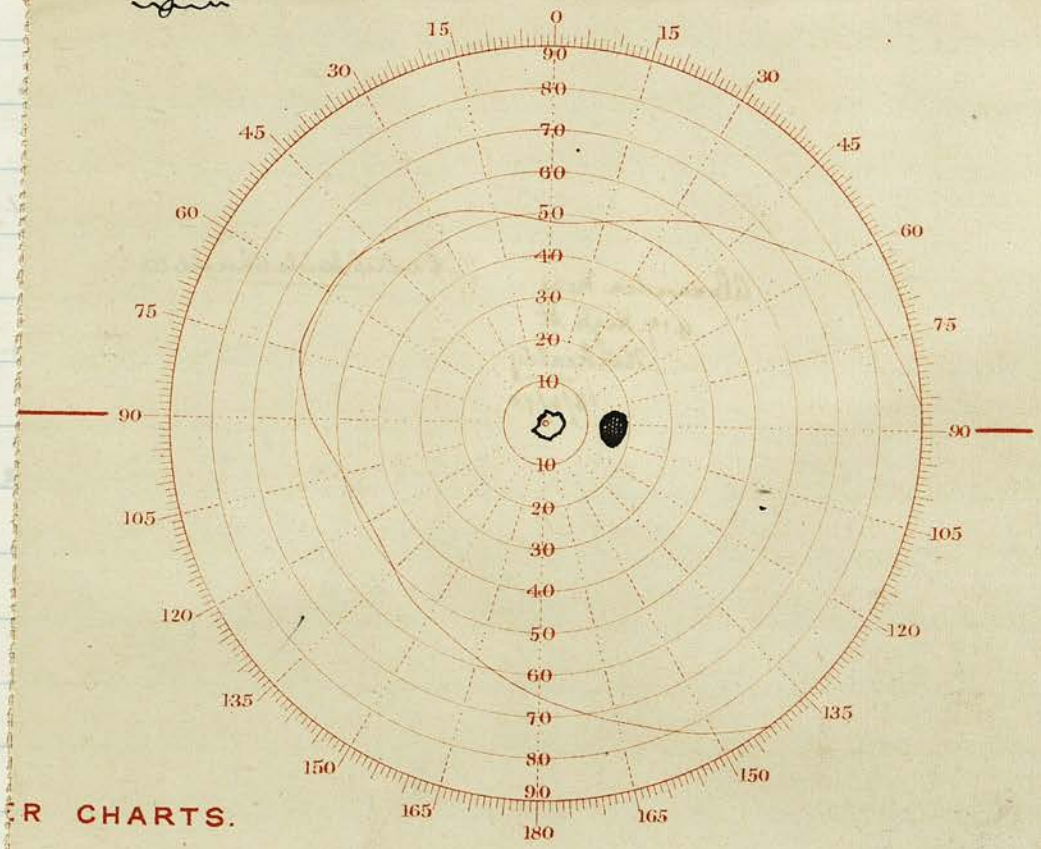
The eccentric continuous red line indicates the average normal fie

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

commencing to use the Automatic Registration.

Fig 35

RIGHT.



ER CHARTS.

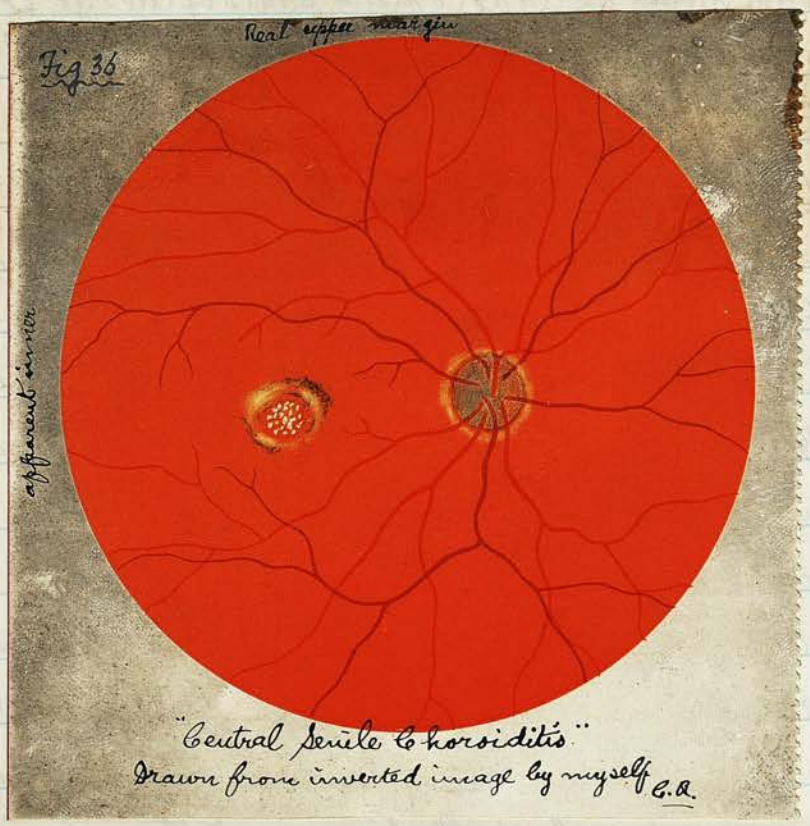
d of Indirect Vision: the small red circle the position of the blind spot.

Published by Messrs Pickard & Curry. 195. Gt Portland St. London. W.

the disease never spreads to other parts of the eye so that the patient never becomes blind though he will be unable to read ordinary print.

Case 30. (fig 34) James W. Dimness of sight had lasted for a year & was gradually becoming worse. Smoked 4 oz tobacco per week & was a moderate drinker. $V = \frac{6}{24}$ in both eyes. Myopic & lenses afforded very little improvement $-1{\cdot}0$ Sph $= \frac{6}{18}$ in both eyes For Bjerrum's Types $V = \frac{4}{60}$ in both eyes. A central scotoma for form & colour Diagnosed as Toxic Amblyopia until I mapped out the scotoma by this method & then careful testing revealed Metamorphopsia (micropsia) & by the erect image there was evidence of disturbance of the hexagonal retinal epithelium at the macula, the patch being fairly well defined.

Case 31. (figs 35 and 36) Alexander H. Dimness of sight in O.D. lasting for two & a half years $V = \frac{6}{60}$ in O.D. $V = \frac{6}{12}$ in O.S. Hypermetropic. $+5{\cdot}0$ Sph $= \frac{6}{6}$ in O.S. while O.D. was not improved by lenses For Bjerrum's Types $V = \frac{6}{60}$ in O.S. but only $\frac{2}{60}$ in O.D. A central scotoma in O.D. for form & colour. Diagnosed as a retinal haemorrhage since a dark reddish, definitely outlined patch was noticed at the macula. Lately after mapping out the scotoma, well marked metamorphopsia (micropsia) was elicited while by the erect image there was seen at the macula a well defined yellow ring surrounding it with some fine twigs of vessels running over its edges while within the ring



"Central Senile Chorioditis"
Drawn from inverted image by myself C.A.

there were numerous sharply defined, little white patches. The ring was bordered by pigment & was a little less than the diameter of the papilla. Around the disc there was a well marked scleral ring (see diagram, fig 36)

Bjerrum in a paper upon this method (nordisk Ophthalmologisk Tidsskrift, II 3, p 149 et seq) refers to another class of cases "Disseminated Choroiditis" & brings out the existence of scotomata which otherwise owing to their small size or to there being only a somewhat diminished functional activity in the corresponding areas escape detection by the ordinary method of examination. Ring shaped areas of diminished functional activity & of greater or less breadth seem to be the rule in this affection though often the defective area is not quite annular. These scotomata lie close up to the point of fixation sometimes involve it.

Moreover in another class of cases "Retinitis Pigmentosa" in which as is well known one occasionally finds ring shaped scotomata, the scotomata are not usually found close to the point of fixation but have their inner border about 20° from it. In this disease the small test objects often reveal a very marked concentric limitation which could not be detected in ordinary daylight.

In another class of cases "Circumpapillary Choroidal Atrophy" accompanying high degrees of Myopia we may or may not find an enlargement of the blind spot when measured with the small test objects. A large scotoma in this region stretching close up to the point of fixation is by no means a favourable sign even when the scotoma is not absolute. All these observations I have been able to confirm though owing to the pressure of time I have not been able to examine a sufficient number of cases to justify me in detailing them here at present.

Bjerrum further alludes to a class of cases in which Keratitis Punctata, Iritis with Synechiae & Vitreous Opacities were the objective signs & in these often a very pronounced concentric limitation was met with but of this class I cannot speak as I have not examined any cases. However an opacity in the transparent media does undoubtedly produce concentric limitation of the field as tested both in the ordinary way & also by small objects It therefore becomes necessary to determine with the oplithalmoscope whether the veiling of the fundus is sufficient to account for the limitation

A comparison of the two eyes may sometimes decide this point as we may find that the eye in which the fundus is most clearly seen presents the most marked functional defects, i.e. greatest

limitation of the field. The exploration of the field may thus show that the affection exists all over the fundus although there may be no ophthalmoscopic evidences of choroiditis & only a few vitreous opacities We thus see the value of this method in diagnosis.

Another group of cases in which an examination of peripheral vision made at a greater distance than is usually done would afford interesting information contains cases of Partial Hemianopia & the scotomata sometimes left after attacks of Megrim.

Dr. Berry in the Ophthalmic Review, Vol IX, p110 refers to a case of Megrim in which by this method he was able to demonstrate ascotoma that was bilateral, not much larger than the blind spot, very irregular in outline, not passing beyond the middle line & exactly similar in both eyes which facts are strongly suggestive of a central origin & could not be demonstrated in the ordinary way thus also showing the superiority of this method.

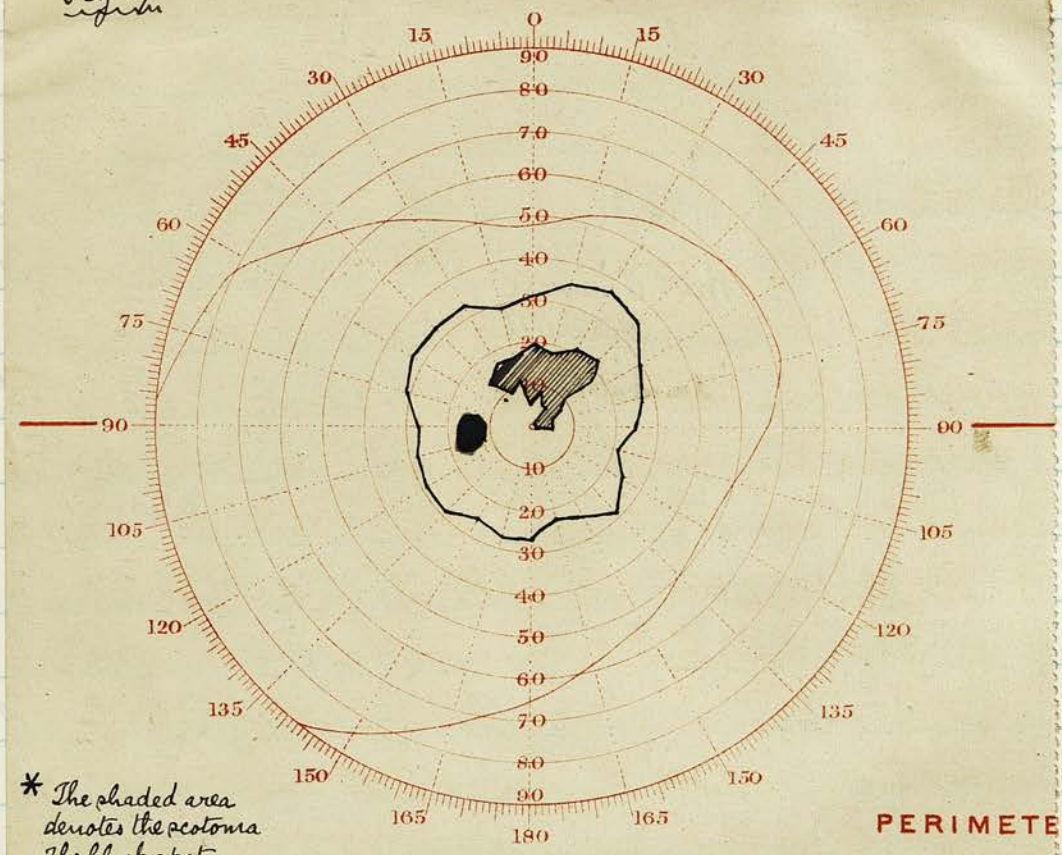
I wish here to bring forward another advantage afforded by this method & will illustrate the point by a couple of cases.

Case 32. (fig 34) Mrs L. 18/4/90 Has had dimness of vision in O.S. for the last four months. no swelling of feet or hands no headaches & no albumen in urine $V = \frac{6}{9}$ in O.D. but

Fig 37

"Centre each chart with "pointer" at Zero before

LEFT.



* The shaded area denotes the scotoma The black spot denotes the blind spot

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The eccentric continuous red line indicates the average normal Fiel

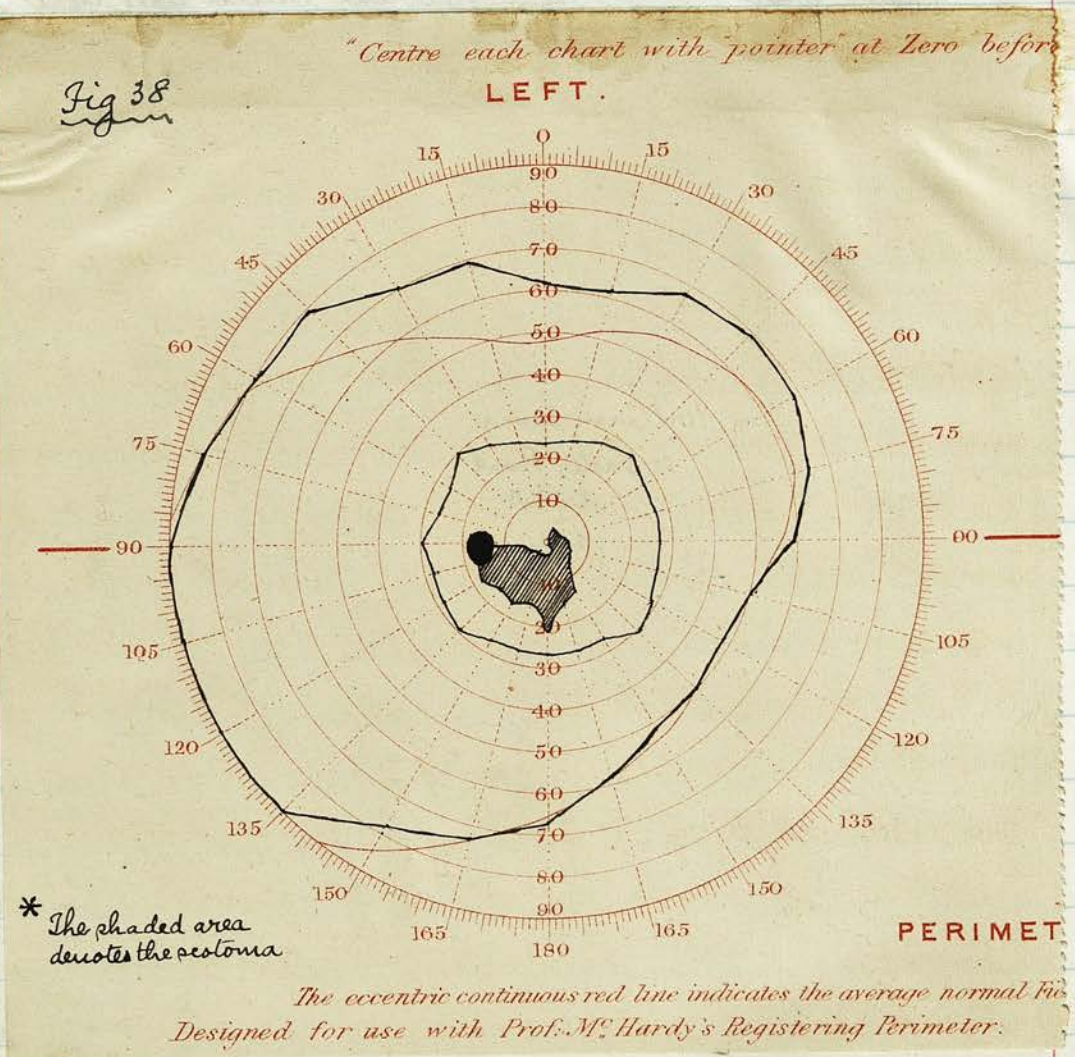
Designed for use with Prof. McHardy's Registering Perimeter.

in O.S. V is only $\frac{6}{60}$. Hypermetropic. With +2.0 Sph = $\frac{6}{9}$ partly in O.D. while in O.S. +2.0 Sph = $\frac{6}{18}$ partly. With +5.50 Sph reads No 1 J with O.D. but only No 5 J with O.S.
Ophthalmoscopically O.D. media clear & no exudation nor haemorrhages O.S. The vein running from the disc below the macula is blocked in two places & around it are several haemorrhages & a dense soft white cloudiness in diffuse spots in the deeper retinal layers due to an exudation & causing thickening of the retina From this level downwards there extend several round small superficial haemorrhages. No exudation at the macula, at the periphery or indeed anywhere else By $\frac{10}{300}$ no defect in the visual field could be detected By $\frac{3}{2000}$ there is no restriction of the boundaries of the visual field but above the macular area there is a somewhat T shaped scotoma. The cross piece of the T runs above the macula & extends about 18° to the inner side of the vertical diameter of the field while the upright piece descends to the inner side of the point of fixation which it involves.

This is evidently a case of Phlebitis of one of the retinal veins & the point that I wish to bring out by means of it is, that, by testing the visual field at intervals by this method, we can obtain a fairly accurate idea of the progress of the case which otherwise could not be obtained either by the ophthalmoscope or by the ordinary perimeter.

Case 33. (fig 38) William S. 26/4/90 Complains of a dimness of sight in O.S.

Fig 38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* The shaded area denotes the scotoma
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Designed for use with Prof. Mc Hardy's Registering Perimeter.

that has lasted for a week. Ten days ago he received a severe blow upon this eye. No albumen nor blood in the urine. No nervous symptoms in the slightest degree. V = $\frac{6}{9}$ in O.D. V = $\frac{6}{36}$ in O.S. Hypermetropic +1·0 Sph = $\frac{6}{6}$ in O.D. With +1·0 Sph = $\frac{6}{24}$ in O.S. For Bjerrum's Types V = $\frac{6}{12}$ in O.D. but only $\frac{1}{60}$ in O.S. Ophthalmoscopically = O.D. normal. In O.S. the disc is swollen & its margins hazy & indistinct especially above & on the outer side No haemorrhages Veins engorged & tortuous. Arteries about normal Parallactic movement present at the disc. Slight exudation around disc margins but none anywhere else. By $\frac{10}{300}$ the boundaries of the visual field are normal & no scotoma is to be detected on careful examination By $\frac{3}{2000}$ the boundaries of the field are about normal but a well marked scotoma both for form & colour is revealed. This scotoma extends from the blind spot inwards below, or very slightly encroaching upon, the papillo-macular area to ~~xx~~ 5° to the inner side of the point of fixation which it here slightly involves. Downwards & inwards the scotoma extends 21°. The case is evidently one of Retrobulbar Neuritis & the important point to note is that though $\frac{10}{300}$ reveals no scotoma yet this method does & by examining at intervals we are able to note the progress of the disease far more accurately than by the ophthalmoscope & the state of the visual acuity

Before concluding I wish to sum up briefly the chief points contained in this paper

(1) The nature of the test & the mode of application.

(2) The fineness of this test in comparison with those in ordinary use.

(3) That immediately surrounding the optic disc there exists a zone of nerve fibres in which both the form sense & colour sense are deficient as compared with the adjacent parts of the retina.

(4) The characteristic & diagnostic nature of the scotoma exhibited in Glaucoma.

(5) The proof that increased tension is the cause of this scotoma

(6) The evidence in favour of the theory that the increased tension acts upon the optic nerve.

(7) The value of this method in the differential diagnosis between Glaucoma & Optic Atrophy; also between the three classes of cases — Retrobulbar Neuritis, Toxic Amblyopia & Central Senile Choroiditis — & its effect upon the prognosis

(8) The evidence in favour of the theory that Toxic Amblyopia is due to a functional disturbance

(9) Some new facts regarding the nature of the scotomata in cases of Disseminated Choroiditis, Retinitis Pigmentosa, Serous Iritis & Megrim

(10) The value of this method in accurately ascertaining the progress of disease which the present methods of examination are inadequate to determine except in the roughest manner

In conclusion I would emphasize the fact that this method of examination & the points before mentioned are completely new additions to our knowledge of Ophthalmology & I trust that they will be of great service in furthering the means that are at present in use for the investigation of disease.

I have the honour to be,
Sir,
Your most obedient servant,

Charles Ramage M.B.C.M.