UNIVERSITY OF GLASGOW.

Thesis Submitted for the Degree of M.D.

SOME VASCULAR ABNORMALITIES IN THE EYES OF YOUNG MEN OF SERVING AGE

With Illustrations.

By

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Introduction.

In the Spring of 1941 an Italian Civilian interned in Egypt was admitted to a British General Hospital after having been shot through both Orbits by a Rifle bullet. He was found to have choroidal haemorrhages in the lower half of both eyes and a symmetrical constriction of both lower temporal arteries. A further three cases with somewhat similar arterial attenuation were seen and it was decided to follow up this investigation. Unfortunately the author was taken prisoner in June 1942 and the original case notes of these four patients were lost. However, during captivity in Italy, a fifth case was seen and followed up for a period of about a year. From October 1943 until April 1945 a large number of Allied Prisoners of War in Germany presented themselves for examination with a view to repatriation and it was possible to study the effects of gun shot wounds and blast on the vascular system of the eye. This study forms the first part of this Thesis. The second part of the Thesis is devoted to the vascular changes occurring in cases of Periphlebitis Retinae, Periarteritis, Acute Choroiditis and Heterochromic Cyclitis. Both parts have been illustrated with water-colour drawings of the fundi oculorum by the author. Wherever possible, X-ray photographs were taken and visual fields charted.

Part 1.

The Effects of Trauma on the Ocular Vascular System. Description of Cases.

For Descriptive purposes the cases have been divided into five groups.

1. Choroidal Haemorrhages and Traumatic Exudative Choroiditis.
11. Ruptures of the choroid.
111. Lesions of the macula.
1V. Abnormalities in the Intraocular Pressure.
V. Miscellaneous Traumatic Lesions.

Group 1. Choroidal Haemorrhages and Traumatic Exudative Choroiditis.

It was found that cases of choroidal haemorrhage developed the traumatic exudative choroiditis (of Lagrange). Hence these cases are grouped together. The first four are described from memory and the first case illustrated from memory (Fig.1)

Case (i)

V.C., aged about 45, an Italian internee, was shot through both temples by a rifle bullet. He was first seen about an hour after the accident in a very excited state. Extensive/

Extensive subconjunctival haemorrhages were present in both eyes and ophthalmoscopic examination revealed extensive choroidal haemorrhages in the lower halves of both eyes. A few days later as the patient became less excited, constriction of both lower temporal arteries at identical distances from the optic disc was seen (Fig.1 Right Eye). The haemorrhages were somewhat more widespread in the left and involved the macular area. Visual acuity was greatly reduced and the patient at first asserted that he was blind. This, however, was apparently due to excitement and his visual acuity was ultimately found to be something in the nature of 6/60 in each eye. Examination of the fields of vision showed no well-defined altitudinal hemianopia as might have been expected, but a generalised contraction of the peripheral fields involving the fixation point on the left side. The left external rectus muscle was paralysed on admission but this soon recovered. The fundus picture gradually changed to one of fibrous tissue formation in the lower half of the retina (exudative choroiditis of Lagrange) but the temporal arteries never regained their normal calibre.

Case (ii).

M.B.M., a Libyan prisoner of War, was admitted to hospital several/

several weeks after injury with loss of the right eye, infection of the right lachrymal sac and defective vision in the left eye. He was stated to have been injured by a rifle bullet which appeared to have passed through both orbits, probably from right to left. He denied having had an operation on the right eye but it is possible that this had been enucleated rather than that it had been destroyed by the bullet. Ophthalmoscopic examination of the left eye once again revealed extensive dark choroidal haemorrages over the lower half of the retina but, in this case, both the lower nasal and the lower temporal arteries were much constricted. It was not possible to measure the visual acuity accurately but from the way in which the patient moved about, it was obvious that the acuity was much reduced. Examination of the visual field again showed a generalised contraction but in this case it was possible to define a greater deficiency in the upper nasal field than elswhere. Extirpation of the right lachrymal sac was performed on account of its septic state.

Case (iii)

A young New Zealand soldier was examined several weeks after injury complaining that he could not see the upper half of objects with his right eye although he could see the lower half/

half. He had been wounded by a small piece of shell fragment which was lodged in his right lower eyelid and which was easily removed by the hospital Medical Officer from under the skin. It is, however, possible that some other fragment entered his orbit but unfortunately he was transferred to another hospital before further investigation could be made. Ophthalmoscopic examination of the right eye showed once more the extensive choroidal haemorrhages and constrictiom of the lower nasal and temporal arteries, charting of his visual fields would certainly have demonstrated loss of the upper visual field.

Case (iv)

A Polish Sergeant Major aged about 40 was struck on the head by a metallic splinter which was found later to be intracranial. In addition to this, he was found to have a metallic foreign body under the skin of the left temple with a small wound of the left lower eyelid, demonstrating a possible route for a projectile to enter the left orbit and thus to cause concussion of the eyeball. The patient was mentally confused and bed-ridden and it was not possible to test the visual acuity accurately or to chart the fields of vision. Ophthalmoscopic examination revealed once more extensive choroidal haemorrhages in the lower half of the retina and constriction of both the lower arteries. He was ultimately transferred/

transferred to another hospital where he died.

Case (v)

Pte. H.C. aged 27 was wounded in March 1943 by a rifle bullet which entered his right orbit at a point 2 cms. behind the external orbital margin and emerged from the left orbit at a point 5 cms. behind the external orbit margin. He complained of defective vision in the right eye, numbness of the right cheek and inability to open his mouth wide. Examination performed 3 or 4 weeks after the injury in a British prisoner of war hospital in Italy showed that the visual acuity in the right eye was reduced to the ability to count fingers in the lower half of the field of vision only. V.A.L. was 6/18 with + 0.5 Sphere and - 1.0 Cyl. axis 180 = 6/6 The right palpebral aperture was narrower than the left and there was some enophthalmos of the right eye. These two signs were presumably due to cicatric-:ial contraction behind the eye. The right pupil was semi-dilated and reacted very sluggishly to light directly but briskly consensually. The reverse was the case in the left eye, the pupil of which was of normal size. The right eye could not be raised above the horizontal. Examination of the right fundus revealed once more the wide spread choroidal haemorrhages in the lower half of the eye. The lower temporal artery was much attenuated/

attenuated and a small fibrous band was visible on its temporal side. This was first thought to be a fibrosed vessel but further examination (Fig.11) suggested that it was a congenital anomaly. The temporal half of the optic disc was pale and a small choroidal tear could be seen nasally. As time progressed, large plaques of fibrous tissue formed in the area originally occupied by the haemorrhages (Fig.11) and the ocular movements greatly improved. A small haemorrhage could still be made out temporal to the disc (Fig,11). The refraction of the left eye gradually improved and, by October 1943, it was 6/9 with + 0.5 Cyl. axis 90^{C}. Examination of the peripheral field of vision of the right eye showed that the lower temporal field only was preserved (Chart 1). The left visual field was normal. The gradual improvement in the astigmatism of the left eye suggests that it is possible for a temporary astigmatism to be caused by concussion.

Case (vi)

Sergt. Major N. a New Zealander aged 29 was injured in 1941 by a rifle bullet which struck the lower orbital margin on the right side 1 cm. from the line of the infero-lateral angle and 2 cms. below the lid margin, and fractured the bony margin and emerged at a point 2 cms. behind the infero-lateral angle. When examined in January 1944, visual acuity of the right eye was found to be reduced to the perception of hand movements in

the lower nasal field. The right pupil was semi-dilated and reacted only very slightly to light directly but more briskly consensually. The reverse obtained in the left eye, in which the visual acuity was 6/4.5. Ophthalmoscopic examination of the right eye revealed one small punctate lens opacity peripherally and a few small vitreous opacities. The fundus picture was normal in the upper half but the lower temporal area was occupied by dense fibrous tissue similar to that in case (v)(Fig.11)and much pigmentary disturbance which ceased towards the periphery (Fig.111). The lower temporal artery was of normal calibre but became lost in the fibrous tissue and could be seen again peripheral to the fibrous tissue. The lower temporal vein, however, was reduced to a thread up to its junction with the lower nasal vein. A little sheathing was visible in that portion of the vein immediately inferior to the junction. It might be argued that this vein only appeared to be constricted owing to its being buried in fibrous tissue but it must be noted that the vein formed by the nasal and temporal tributaries and the nasal vein itself both lie in the fibrous tissue for a small part of their course and are not constricted.

Case (vii)

Gdsm. J.W., aged 21, was injured by a mortar-bomb explosion in April 1943. The skin over the inner 1/3 of the lower/

lower orbital margin on the right side was pierced by a fragment of the metal and the right eye blinded. There was no apparent external injury to the eye but the right pupil was semi-dilated and reacted sluggishly to light directly but briskly consensually. The reverse obtained in the left pupil which was of normal size. The right eye was divergent. V.a.R. was perception of Light in the lower half of the field only and V.a.L. was 5/9. with + 0.5 Cyl. axis 165^0, 5/5. Ophthalmoscopic Examination of the right fundus oculi (Fig. IV) revealed an extensive area of fibrous tissue below the optic disc and stretching outwards to the macular area with a little pigmentery disturbance here. All the vessels, both arteries and veins, appeared to be constricted, but careful examination suggested that the lower temporal artery was not involved so markedly as the others which was borne out by the appearance at the temporal end of the fibrous placque of a looped artery of normal calibre. It is difficult to explain this curious looping of this artery as there does not seem to be any visible connection between it and the shadowy lower temporal artery further up. Some fine vessels on the anterior surface of the fibrous placque appear to have had the function of collaterals. X-ray examination of the right orbit was negative.

SUMMARY OF GROUP 1.

Seven cases are described of choroidal haemorrhages followed by exudative choroiditis. Five were seen in the stage of choroidal haemorrhage (Cases i - v) of which three(cases i,ii and v) were followed to the stage of exudative choroiditis.

In/

In all seven there was attenuation of the vessel-calibre: In one or more arteries (Cases i - v) or a vein (Case vi) or both artery and veins (Case vii). All seven cases were injured by high velocity projectiles: A rifle bullet (Cases i, ii, v and vi) or a met alic splinter (Cases iii, iv and vii) and in all the injury was to the orbit and only indirectly to the eye.

Group 11 Ruptures of the Choroid.

The occurrence of vascular attenuation in the above cases led to an investigation into the possibility of such being present in other types of injury. Group 11 comprises cases of Rupture of the Choroid.

Case (viii)

Pte. T. aged 19 was injured on 7.1.44 by a bullet from an automatic rifle fired from above. The wound of entry was 8 mm. below the mid-point of the left lower eyelid. The bullet had been removed on 18.1.44 by a German surgeon from behind the left angle of the lower jaw. When examined about six weeks later, the left eye was almost blind and there was anaesthesia of the left half of the face. Considerable difficulty was experienced in opening the jaw widely. Ophthalmoscopic Examination of the left fundus oculi showed (Fig. v) two choroidal ruptures below. The lower artery was greatly attenuated and the lower nasal artery disappeared entirely/

entirely shortly after its commencement. These two arteries along with the lower temporal artery all showed sheathing and the disappearance of the lower nasal artery might be attributed to oedema of the surrounding retina. The lower nasal vein was represented by a very fine cord and one tributary of the lower temporal vein which joined the main trunk anterior to the more temporal of the two choroidal tears was represented by a fibrous cord. The macular area showed patchy yellow discoloration and another small yellow crescentic patch was discernable below the optic disc. A band of pigment stretched in a half-moon shape from the macular area, below the disc to a point the same distance from the disc on the nasal side giving rise to the classical picture of rupture of a posterior ciliary artery in addition to the choroidal tears. The Perimeter (Chart 11) showed limitation of the upper and temporal field.

Case (ix).

Pte. M. aged about 23 was wounded by a metallic splinter which entered below the left mastoid process and apparently emerged through the left cheek on 19.1.44. Another fragment injured his nose and this had been repaired by a wire by a German surgeon. On admission to Hospital on 18.2.44. swelling of the left parotid gland was observed and it was seen that the lower jaw had been wired. X-ray examination revealed a fracture of the left zygomatic bone involving the floor of the orbit/

orbit and opacity of the left antrum. When examined on 8.3.44 V.A.R. = 4/12 c - 0.75 Cyl. - 180° = 4/45 and V.A.L. = 4/18 c - 0.5 Sphere/ - 0.5 Cyl - 180° = 4/12. The left upper eyelid was split and the cornea showed a small scar on the nasal side. Examination of the left fundus with dilated pupil next day revealed (Fig. V1) marked pigmentary disturbance down and in from the optic disc. The diagram was drawn three weeks later but at first a few haemorrhages were visible in the same area. Two small choroidal ruptures could be seen amongst the pigmented area. The lower nasal artery could be seen to be about 2/3 the diameter of the lower temporal artery and also about 2/3 the diameter of the lower nasal artery in the right retina suggesting that, although there is frequently found a disparity between the nasal and temporal arteries, there is here a genuine attenuation. Perimetric examination of the visual fields was not very convincing as the fields of the uninjured eye varied after four weeks interval (Charts 111 and 1V) but the change is in the direction of a generalised contraction of the peripheral field.

Case (X)

Pte. R. aged 24 was injured in the right eye by an explosive/

explosive bullet in August 1942. The portion of bullet was removed by a German surgeon 7 - 10 days later. When examined on 11.4.44 V.A.L. was reduced to ability to count fingers at 6 inches. A Scar was visible in the right lower eyelid centrally and a small symblepharon was present between the conjunstival surface of the lid and the bulbar conjunctiva about 2 cm. from the limbus indicating that the bullet had penetrated the eyelid and at least impinged upon the eye. The right pupil was larger than the left and the pupil reacted very sluggishly to light direct-:ly but more briskly consensually. The reverse obtained in the left eye. Ophthalmoscopic Examination of the right eye revealed a few vitreous opacities still present. The fundus oculi showed (Fig. Vll) a cloudy yellowish area replacing the macula and several choroidal tears surrounded by pigment below the optic disc towards the periphery. The largest of these was the most peripheral which probably corresponded with the posterior end of the part of the globe directly injured by the bullet. The lower nasal artery was found to be constricted at its outset and much narrower than the upper nasal artery and about 2/3 the diameter of the lower temporal artery . The lower papilliary artery was 2/3 the diameter of the upper papillary artery. X-ray examination of the right orbit was negative. Perimetric examination of the right visual field revealed (Chart V) a constriction of the upper and temporal areas and a large central scotoma corresponding to the dusky yellow area at the macula.

Case xi.

Sergt. M.E. a Tunisian aged 24 was injured on 27.1.44. by a bomb splinter which penetrated his left cheek and caused defective vision in both eyes, but more especially the right. An X-ray photograph showed a metallic foreign body at the apex of the right orbit about the size of the little finger nail. (This was repeated to make certain that the foreign body was not, in fact, in the left orbit). In addition damage could be seen to the orbital floor. On examination three weeks later the V.A.R. was found to be ability to count fingers at 6 inches and V.A.L. = 4/24 partly with -0.5 Cyl. axis 90° = 4/9. The right pupil was dilated and almost inactive to light directly but more brisk consensually. The right eye could not be abducted, due possibly to injury to the right external rectus from contre-coup, and the left eye could not be elevated due, presumably, to mechanical interference by scar tissue. Ophthalmoscopic examination of the right fundus oculi (Fig. VIII) revealed a large circular choroidal rupture up and in from the optic disc. This was surrounded by an area in which the choroidal vessels could be seen but were covered by numerous spots of pigmentation. The retinal vessels running over this area were seen to be broken up and a large haemorrhage could be seen at one point. Above the injured area a complicated network of blood vessels could be seen, apparently in an attempt to establish collateral circulation and several haemorrhages could also be seen. The upper nasal vein down to its/

its junction with the upper temporal vein was thread-like. The arteries were apparently unaffected. Three months later (Fig. 1X) the broken up vessels on the surface of the injured area had disappeared and a curious little leash of blood vessels could be seen at the periphery possibly in an attempt to establish a collateral circulation with the markedly attenuated upper nasal vein. In addition to these changes the macula was replaced by dusky pigmentation and the macular veins were markedly tortuous and cork-screwed. The optic disc was very pale from the outset. Examination of the left fundus oculi showed oedema of the retina in its lower half without any signs of haemorrhage or detachment of the retina. Perimetric examination of the peripheral fields of vision revealed (Chart V1) only slight generalised contraction in the left eye but complete absence of the upper field and only a small amount of the other parts of the field in the right eye. No attempt was made to remove the foreign body from the right orbit. It was not found possible to follow this case up for a sufficient length of time to ascertain whether the astigmatism in the left eye was becoming less as was the case in Case (V).

Case (xii)

Pte. W. aged 29 was injured by a hand grenade which exploded/

exploded immediately in front of him in January 1943. When examined one year later he was found to have two scars round his left eye; one which extended from the outer extremity of his eyebrow to the level of the external palpebral ligament and a second, smaller one below the inner end of the same eyebrow in the upper eyelid. V.A.R. was 4/12 and V.A.L. Perception of Hand movements. The left pupil was larger than the right and reacted more sluggishly to light than the right pupil. Ophthalmoscopic Examination of the left fundus (Fig. X) revealed a large choroidal rupture up and in bordered by pigmentary disturbance. All veins were of normal calibre but the upper nasal artery was reduced markedly in size from its commencement. Perimetric examination of the peripheral fields certainly demonstrated contraction of the lower temporal field as would be expected but there was, in addition, contraction of the upper nasal field.* The visual acuity was, however, so markedly reduced that it is possible that the patient's responses were not reliable. It was not possible to X-ray this patient but the method of production appeared to be by blast.

* See Chart VII..

Case (xiii)

F/Sergt. W. aged 23 was brought down in the Zuyder Zee on 14.5.43 at 07.00 hrs. At 23.30 hrs. on the same day he was in a German Hospital conscious, orientated in time and place and without psychological disturbance. He had a flesh wound/

wound about the size of a sixpence 2 cms. above the right eyebrow and 1½ cms. from the mid-line and a haematoma of the right eyelids. There were small superficial wounds of the right upper and lower eyelids. On 18.5.43 the German oculist reported V.A.R. = Perception of Hand movements at 50 cms. V.A.L.= Perception of Light. The Right Eye showed Subconjunctival haemorrhage. Several small foreign bodies could be seen in the cornea and the pupil was dilated. Examination of the fundus oculi revealed blurring of the disc margins; the veins were dilated and the whole temporal half of the retina was occupied by massive exudates and haemorrhages. At the extreme periphery at 3 o'clock a white exudate could be seen which extended into the vitreous. The left eye showed a perforating scar paracentrally at 12 o'clock. The pupil was dilated and the lens could be seen to be grayish-white, opaque and swelling. A localisation X-Ray showed a metallic foreign body in the lower anterior part of the eye. Trial by magnet was negative in both eyes on two occasions. On 27.6.43 the left lens was extracted and a note on 27.8.43 reported V.A.L. as 6/12 with a + 12.0 Sphere and on 19.10.43 V.A.R.= 6/36 with - 2.0 Cyl. axis 45° and V.A.L. = 6/6 + with + 11.0 Sphere.

When/

When examined on 27.6.44, V.A.R.= 4/36 and V.A.L.= 4/24 with glasses. By this time he was quite disorientated in time and place and, although he could answer questions, his memory was quite gone and he was not apparently responsible for his actions. It was therefore quite impossible to chart his peripheral fields of vision and the recording of his visual acuity may have been entirely innaccurate. Examination of the right fundus (Fig. Xl) showed a very large rupture of the choroid temporal to the macula bordered by pigment. A small artery and vein could be seen disappearing into this white area. The vein showed a small localised constriction after it had been joined by the macular twigs which seemed to be somewhat more dilated than normally. All the arteries were narrow but, in this case, the optic disc was very pale and atrophic and the narrow arteries were consistent with an optic atrophy of this duration. The macula was replaced by a macular "hole" surrounded by somewhat dusky retina. Bright radiating lines of light reflex could be easily seen round the macular area. X-Ray showed a gun shot wound of the right frontal bone involving the right frontal sinus but not the right orbit. multiple metallic foreign bodies were present the majority extracranial but the largest appeared to be in the orbit near its roof. The gun shot wound had healed over but the brain could be seen pulsating beneath the skin. Some secondary cataract was present in the left eye.

Case (xiv)

Corpl. B. aged 24 was injured by the explosion of a shell on 22.3.43. He was first examined on 7.5.44 when V.A.R. was found to be ability to count fingers in the lower half of the visual field only and V.A.L. 5/6, with + 1.0 Sphere, 5/4.5 Three small circular scars were present in the medial half of the right lower eyelid. The right pupil was semi-dilated and reacted only sluggishly to light directly but briskly consensually. The left pupil was of normal size and reacted briskly to light directly but sluggishly consensually. Ophthalmoscopic Examination of the right fundus oculi (Fig. XII) revealed a macular "hole" with a dusky area surrounding it. In the lower half of the retina could be seen extensive pigmentary disturbance and both lower arteries, nasal and temporal, were seen to be constricted to about 2/3 of the diameter of the upper arteries. The two final tributaries of the upper temporal vein were seen to have marked constrictions and dilatations in their course and several small haemorrhages could be seen in the vicinity indicating that the vessel was thrombosed. The patient was examined again five months later (Fig XIII) when the haemorrhages had become absorbed and the irregularities of the upper temporal vein were not so evident except where they were constricted by the upper temporal artery and its branches. The light reflexes formed a triangular cap above the macular area. Below, towards the periphery, several small choroidal ruptures could

be/

be seen in the area of pigmentation. X-Ray examination of the right orbit was negative. Perimetric examination of the peripheral fields of vision showed (Chart VIII) that the right upper temporal field had largely disappeared and the upper nasal field was severely restricted. Central vision was also absent.

Case (XV)

Spr. R.K. aged 27 was injured by a metallic splinter in his left eye in April 1944. He was standing about five yards distant from another man who was chopping sticks with a small axe when he felt something strike his left eye. When examined in October 1944, he was found to have a minute healed wound of the sclera at 3'clock about 1 m.m. from the limbus. V.A.R. was 6/6, and V.A.L. 6/9. The left pupil was widely dilated and fixed. Ophthalmoscopic Examination of the left fundus (Fig. XIV) revealed a metallic foreign body piercing the retina and choroid peripherally at 7 o'clock. This was surrounded by an area of pigmentary disturbance. The lower temporal vein was reduced to a fine thread which apparently arose from this pigmented area. All the arteries were reduced in calibre on comparison with those of the right eye (Fig.XV) The lower temporal artery showed some sheathing shortly after its commencement and then disappeared. The same occurred with a small branch which proceeded almost vertically downwards.

From/

From the appearance of the surrounding retina, it is probable that this was due to oedema or cloudy swelling of the retina. X-Ray examination of the left orbit was Positive for a metalling foreign body and he was transferred to another hospital in Breslau and was not seen again owing to the Russian siege of that town. Perimetric examination of the peripheral fields of vision (Chart IX) showed a generalised contraction of the field of the left eye, but rather more marked above than below, suggesting that the arterial constriction may be due to some degree of optic atrophy in spite of the good visual acuity. This is supported by the relative pallor of the optic disc. A curious little swelling could be seen on a small tributary of the upper nasal vein in the left eye (Fig. XIV) upper limit of drawing) which suggests that thrombosis had occurred but this is so doubtful that it must be discounted.

Case (xvi)

Spr. T. aged 43 was injured by bomb-blast in April, 1941. The bomb burst about five yards away, spattering his face with small metallic particles and rendering him unconscious for twelve hours. He stated that his skull was fractured. When first examined on 1.11.43 the V.A.R. was found to be ability to count fingers at 1 foot and the V.A.L. 6/6 partly . The Right pupil reacted sluggishly to light directly but briskly/

briskly consensually. The reverse was the case with the left pupil. Opthalmoscopic Examination of the right fundus revealed (Fig. XVI) pigmentary and colloid-body like disturbances at the macula. Below and far peripherally could be seen a few very small choroidal tears associated with pigment. No arterial or venous changes could be made out but it must be pointed out that the choroidal tears occurred in an area in which no large vessels were present and they were small compared to some of the other tears described. Perimetric examination of the peripheral fields of vision showed an almost normal field but a large central scotoma could be made out (Chart X). More accurate delineation of this scotoma was possible on the Bjerrum Screen (Chart Xa).

Case (xvii).

Pte. L. aged 37, a French soldier, was injured on 17.6.40. by the explosion of a hand grenade about 10 metres away from him. He was rendered unconscious and, on waking, found that he could not see with his left eye. He was first examined on 20.3.43. when V.A.R. was 6/6 and V.A.L., Perception of Hand movements only. The left pupil was larger than the right and was not circular, an irido-dialysis being present on the temporal side. The left pupil only reacted very slightly to light directly but more briskly consensually. The reverse was/

was present in the right eye. Ophthalmoscopic Examination of the left eye revealed a fairly extensive cataract on the temporal side which unfortunately, obscured the fundus details, but it was possible to make out (Fig. XVll) three concentric ruptures, one close to the optic disc, one almost at the macula and one larger one further temporally. All three were accompanied by pigmentary disturbance. No changes in the vessel calibre could be made out. These choroidal ruptures corresponded more closely to the classical type which is normally illustrated as typical of the condition in text-books in that their position is concentric with the disc. This case is one of the few in the series which exhibited external signs of injury in the form of an irido-dialysis and traumatic cataract. The visual acuity did not allow of charting of the fields of vision. An X-Ray had been done but it was not found possible to repeat this and the patient did not know the result.

Summary of Group 11.

Ten cases of various types of choroidal ruptures are described and alterations in the vessel calibre occurred in eight of them. In one, however, (Case Xiii, Fig. Xl) there was present a well marked optic atrophy and this is almost certainly the cause of the constriction. Of the two cases/

cases in which no constriction could be seen, one of them (Case XVi, Fig. XVl) showed only small ruptures peripherally and in the other, of the three ruptures which were present, two occurred in the papillo-macular area and the third was situated in an area where no large vessels were present.* In one case (Case ix, Fig. Vl) it must be admitted that the alteration in the lower nasal artery was very slight and could be accounted for by physiological variation. It must be pointed out, however, that here, as in Case xvi (Fig. XVl) the tears are small. In one case (Case xiv, Fig. Xll) organic obstruction could be seen in the upper temporal vein and this is the only case in this series in which such could be seen. Perhaps the most instructive case in this group is Case (XV, Fig. XlV) which demonstrated a metallic foreign body in situ and a thread-like vein running upwards from it. In case (xi) Fig. Vlll the broken-up remnants of blood vessels could be seen in the area of the choroidal rupture which disappeared in time (Fig. lX).

The method of production of the choroidal ruptures was very varied in this group and some appeared to have been caused by blast alone which was not the case in Group l where four out of the seven sustained injury by a rifle bullet. In Group ll, on the other hand, only two (Case viii, Fig. V and Case X, Fig. Vii) were so injured. To summarise, constriction of the vessel calibre may occur in cases of rupture of the choroid/

* Case xvii, Fig. XVII.

choroid.

Group lll Lesions of the Macula.

In this Group are described 4 cases of injury to the macula. Six cases in Group ll showed macular changes in addition to ruptures of the choroid (Cases viii, (Fig. V.), X, (Fig. Vll) xi (Fig. lX) xiii (Fig. Xl) xiv (Fig. Xll & Xlll) and xvi (Fig. XVl) and have already been described. In this Group are placed those cases which show macular damage only.

Case (xviii)

Sergt. J. aged 30 was injured by the explosion of a mortar shell which blew up the anti-tank gun which he was manning. He was struck by the blast and sustained a wound of his right eyebrow close to its medial end. The explosion blinded him at first but he thought that this was probably due to the presence of powder etc. under his eyelids. His eyes were bandaged for ten days, and when the bandages were removed, he foumd that the right eye was defective. When examined on 21.8.44 the V.A.R. was 2/24 and the V.A.L. 4/3.5. The right pupil was slightly larger than the left and reacted more sluggishly to light directly than did the left pupil. The consensual reflex was brisk but was sluggish in the left eye. Ophthalmoscopic Examination of the right fundus oculi (Fig. XVlll) revealed a macular "hole" surrounded by a dusky area and several small yellowish-white spots above this. Up and out

from/

from the macula could be seen a small spot of pigment. No abnormality in the vessel calibre could be made out. Examination of the peripheral fields by means of the perimeter showed (Chart Xl) a practically normal right visual field and the Bjerrum screen revealed a small central scotoma (Chart Xll). X-Ray examination of the right orbit was negative.

Case (xix)

Pte. L. aged 25 was injured by a football in April 1944 which struck his right eye. When examined on 30.10.44, V.A.R. was 1/18 and V.A.L. 4/45. The right pupil was slightly larger than the left and reacted very sluggishly to direct light but briskly to light consensually. The reverse was present in the left eye. Ophthalmoscopic Examination of the right fundus oculi (Fig. XlX) revealed a deeply pigmented oval patch surrounded by a white somewhat star-shaped area replacing the normal appearance of the macula. The optic disc was slightly pale and no vessel abnormalities could be detected.

Case xx.

Sergt. A. aged 29, a member of the R.A.F. was injured on 18.12.44 when the plane in which he was flying was struck by an anti-aircraft shell. Both eyes were blackened and the left was inflamed. He noticed that his vision in the left eye had been affected and did not improve.

When/

When seen on 31.1.45 V.A.R. was 4/3.5 and V.A.L. 4/24 (1 letter). Pupils both reacted normally and were equal in size. Ophthalmoscopic Examination of the left fundus oculi revealed (Fig. XX) pallor of the optic disc and a pale oedematous area involving the macula and the area between disc and macula. Two small petechial haemorrhages were present in this area and two further ones could be seen above and below. On the course of the upper temporal vein temporal to the macula could be seen a larger haemorrhage. Six weeks later (Fig. XXl) the changes were confined to the macula area, the haemorrhages having cleared up. Examination of the peripheral fields of vision by means of the perimeter showed (Chart Xlll) a practically normal left field but the Bjerrum Screen revealed a large central scotoma, (Chart XlV).

Case xxi.

F/Sergt. H. aged 24, a member of the R.A.F. was injured by a fragment of anti-aircraft shell on the left cheek on 23.4.44. He was rendered unconscious and removed to a German hospital where a splinter was removed from the left cheek. In June 1944 a British Ear Nose & Throat specialist operated on his left maxillary antrum and removed the button of his oxygen mask therefrom. Since the injury he had been unable to feel objects touching his left cheek, he had lost his sense of smell and his visual acuity had been seriously affected in the left eye. Examination on 17.3.45/

17.3.45 revealed some enophthalmos of the left eye, ptosis of the left upper eyelid and slight restriction in the upward movement of the left eye. A healed wound was present on the left cheek in line with the outer orbital wall at the lower border of the zygoma. The left pupil was slightly larger than the right and did not react to light directly although the consensual reaction was present. The reverse obtained in the left eye.

Ophthalmoscopic Examination of the left fundus oculi revealed (Fig. XXll) a pale atrophic disc with constriction of all arteries. In view of the accompanying pallor of the disc this constriction may be attributed to optic atrophy. Below, towards the periphery, could be seen a minimal amount of pigmentary disturbance. The macular area showed a mottled appearance with one small petechial haemorrhage in its upper part. The visual acuity of the right eye was 5/5 and of the left eye perception of hand movements eccentrically. Accurate perimetry was impossible but the visual field of the left eye appeared to be reduced to the extreme temporal and nasal periphery whilst the entire central field was lost. X-ray examination revealed a medium-sized metallic splinter in the left orbit and four smaller splinters in the same area.

Summary of Group lll.

Four cases of macular damage are described. The causative agent is varied. In one (Case xix, Fig. XlX) it was/

was a blow from a football. In two (Case xviii, Fig. XVlll and Case xx. Figs. XX and XXl) it appeared to be blast and in one (Case xxi, Fig. XXll), multiple metallic splinters. Taking into consideration the cases belonging to Group ll a common lesion was the macular "Hole" (Cases xiii, Fig. Xl; xiv, Fig. Xll and Xlll; and xviii, Fig. XVlll). In five others there was noted simply some irregularity at the macula (Cases viii, Fig. V, ix, Fig.Vl ; xvi. Fig. XVl; and xxi, Fig. XXll) and in one an oedematous appearance could be made out (Cases xx, Figs. XX and XXl). One case showed a scar at the macula. (Case xix, Fig. XlX). In two cases only (Cases xi, Fig. lX & xiii, Fig. Xl) could any vascular changes be seen in the macular area. These took the form of tortuosity of the veins draining the macular area. The other cases belonging to Group ll showed vascular changes in other parts of the fundus where there was choroidal damage but not in the macular area. The tortuousity of the veins in Case xi (Fig. lX) suggests that these have enlarged in an attempt to absorb the damaged tissue at the macula. In the four cases of Group lll no vessel changes were noted at all.

Group lV. Abnormalities in the Intraocular Pressure.

Five/

Five cases of changes in the intraocular pressure following injury are described, four of glaucoma and one of hypotension. One case was complicated by a choroidal rupture but it was impossible to assess the arterial calibre in the vicinity and the case has been included in this group. Three of the four cases of glaucoma show vascular constriction along with well marked optic atrophy. In the fourth the fundus could not be seen.

Case xxii.

Gdsm. H. aged 24 was injured by the explosion of a mortar bomb in May 1940. He was blown against a truck and was unconscious for about five hours. On regaining consciousness he noticed that his left arm and left leg were weak but this passed off in a few minutes and he continued fighting. In June, 1940 the vision in the left eye began to fail and the eye became red and painful. He also suffered from headaches. The eye was treated by drops and the sight recovered. About three years later, in September 1943, deterioration in the visual acuity again began. He had no headaches or ocular inflammation and saw no haloes round the lights. Night blindness, however became very marked and he had to be led about at nights. He was first examined on 28.10.43 when V.A.R. was 4/9 and V.A.L. ; ability to count fingers at three feet. Externally the right eye appeared to be/

be normal. Ophthalmoscopic Examination of the right fundus oculi revealed two small patches of pigment peripherally at 12 o'clock and 8 o'clock. A few vitreous opacities and a few spicules and club-shaped opacities in the lens could also be seen. The optic disc had a healthy appearance. Externally the left eye showed a small staphyloma above. The cornea was slightly steamy and the pupil was dilated and fixed. The anterior chamber was shallow. Ophthalmoscopic Examination of the fundus oculi revealed (Fig. XXlll) a very pale atropic deeply cupped optic disc with arterial pulsation at the disc edge. Towards the periphery of the fundus were well-marked pigmented patches widely distributed. All the arteries were narrow and showed sheathing. The intraocular tension was ††† to fingers. X-Ray examination of the left orbit revealed no signs of a metallic foreign body. Examination of the peripheral fields of vision (Charts XV and XVl) showed a normal right field and a progressive general contraction of the left field. The Bjerrum screen confirmed the absence of signs of glaucoma in the right eye. On 11.11.43, in spite of treatment with Gutt. Eserine, he developed an attack of subacute glaucoma and on the following day a trephine operation was performed with complete iridectomy. The eye settled down and the trephine opening appeared to be functioning properly but the intraocular pressure remained elevated and he was treated by Gutt. Eserine Salicyl, b.i.d. until he was repatriated in September 1944. In view of the pigmentary disturbances and the night blindness, a/

a search was made for a septic focus on the grounds of the possibility that the condition might be primarily choroido-retinitis or retinitis pigmentosa with secondary glaucoma (which, however, is rare). The W.R. was negative. B.S.R. was 1.5. X-ray examination of the lungs showed scattered areas of healed Tuberculosis in both upper zones. Examination of the fundus after operation was simpler and marked attenuation of all arteries was noted on 12.2.44. (as in Fig. XXlll), marked sheathing of the arteries could also be made out. This attenuation, however, was most probably the sequel to optic atrophy. Pigmentary disturbances are known to follow chronic glaucoma in the late stages and it is more likely that the glaucoma was the primary condition than that it was secondary to some choroido-retinal disturbance.

Case xxiii.

Cpl. C. aged 40 was injured in action in August, 1942. His anti-tank rifle exploded in front of his eyes. At the same time two land-mines exploded close to him and the exact explosion which caused the injury could not be determined. The left eye felt normal until December, 1942 (i.e. four months later) when the sight began to fail. On examination sixteen months after the injury, V.A.R. was 4/4.5 and V.A.L. no Perception of Light. The right eye was of normal appearance. The left eye showed a somewhat shallow anterior chamber and some incipient lens changes,/

lens changes, which interfered somewhat with examination of the fundus details. However, it could be ascertained that the optic disc was deeply cupped and atrophic (Fig. XXlV). All arteries were narrow and far down and out towards the periphery could be seen a choroidal rupture surrounded by pigment. Owing to the narrowness of all arteries and the presence of lens opacities, it was found impossible to estimate the calibre of the arteries in the vicinity, but it must be admitted that the arteries and veins in the vicinity showed no striking changes. The intraocular pressure was +++ but, as the eye was quite blind, no operative interference was called for. The intraocular pressure felt normal in the right eye and the Bjerrum screen revealed no scotomata. The possibility of this case being one of chronic glaucoma simplex had to be borne in mind in view of the man's age, but the presence of a choroidal rupture indicated that the eye had been damaged. An interesting feature in this case is the long interval (four months) between the initial injury and the failure of vision.

Case xxiv.

Dvr. A. aged 26 was injured by a blow on the left eye by a cricket ball in 1936. The vision in the eye was affected to some extent at the time of the accident but gradually became worse. When examined on 10.8.44 V.A.R. was 4/6 and V.A.L. Perception of Light in the temporal field only. /

only. The left eye was divergent and the left pupil was larger than the right and reacted to light consensually but not directly. The right pupil reacted directly but not consensually. The left anterior chamber was very shallow and a well marked cortical cataract concealed all view of the fundus. The intraocular tension felt +++ to the fingers. The tension did not respond to treatment by Gutt. Eserine Salicyl. 1% t.i.d. and a trephine operation was performed on 23.8.44. The pressure did not return to normal and the eye required Gutt. Eserine Salicyl. 1% t.i.d. until the patient was repatriated in January 1945. The right eye had a normal external appearance apart from the absent consensual light reflex. The right fundus appeared to be normal and no abnormality could be found in the peripheral or central field of vision. As the cataract in this case was the usual complicating one in the posterior cortex, it is thought unlikely that the glaucoma was secondary to a swelling lens.

Case (xxv)

Cpl. W. aged 44 was injured by blast from a bomb which exploded about thirty yards from him in May 1941. Both eyes were blinded at the time but the right eye returned to normal in about a week. The left eye, however, did not improve. When examined 2½ years after the injury, V.A.R. was 4/9 and with glasses/

glasses, 4/6. The V.A.L. was no Perception of Light. Examination of the left eye revealed a small perforating wound and the absence of a small portion of iris up and out. He asserted that no operation had been performed on the eye. The anterior chamber was of normal depth. Ophthalmoscopic Examination of the left eye showed a few lenticular opacities. In the fundus oculi could be seen (Fig. XXV) some pigment round the disc, probably of congenital origin and a deeply cupped, atrophic optic disc. No abnormalities could be made out in the calibre of the blood vessels. The intraocular tension felt normal to the fingers. The right eye was of normal appearance and the Bjerrum screen revealed no scotoma in the right central field. The right intraocular tension felt normal. In view of the apparently normal intraocular tension in the left eye it might be argued that this is a case of cavernous optic atrophy with secondary cupping but the absence of vascular construction is against this. It is regretted that a tonometer was not available as a small rise of pressure might have been present in the left eye which was imperceptible to the fingers.

Case (xxvi)

Pte. S. aged 21 was injured by a fall of coal whilst working in a coal-mine on 22.11.43. His right eye was struck and/

and the visual acuity was found to be seriously affected. There was no subsequent improvement. He was first examined on 6.12.43 when the V.A.R. was 2/60 and the V.A.L. 4/12, with + C.5 Cyl. axis 90^{o} = 4/9. No improvement could be made on the vision in the right eye with lenses. The right eye showed multiple folds in Descemet's membrane and marked lowering of Intraocular Pressure. The fundus was normal. The left eye presented a normal appearance. The folds in Descemet's membrane disappeared after a few days and the Intraocular Pressure returned to normal in a few weeks but the visual acuity remained stationary.

Summary of Group IV.

Four cases of Glaucoma and one of Hypotension are described, all of which followed injury. Two of the cases of glaucoma were under thirty years (cases xxii, Fig. XXIII and xxiv) and two were forty years old or over. It must be admitted that these last came into the age-group in which glaucoma simplex is a definite possibility but one (Case xxiii, Fig. XXIV) showed a choroidal rupture in addition and in all four cases careful examination revealed no signs of glaucoma in the better eye. In one (case xxii), (Fig. XXIII) the possibility of the glaucoma being secondary to a condition such as Retinitis Pigmentosa had to be entertained in view of the pigmentary disturbance and the history of night blindness. Glaucoma is, however/

however, an unusual complication of Retinitis Pigmentosa and, in addition, only two minute particles of pigment could be found in the fundus of the good eye. One case of Hypotension is described (Case xxvi).

Group V Miscellaneous Lesions.

Six Cases are included in this group, four with traumatic lesions and two with opaque nerve fibres simulating a fundus lesion. These latter are included as controls as in one case it was at first thought that the eye had been injured by a metallic foreign body.

Case xxvii.

Pte. H. aged 42 was injured by a bomb explosion on 17.6.41. The bomb exploded a few yards away and his face was sprayed by earth and clay. He was struck just above the left eye which caused considerable bruising for five weeks. When the swelling had subsided he noticed that the visual acuity of the left eye was defective and no improvement has occurred. He was examined three years after the injury. The left eye was divergent. V.A.R. was 6/18 and with glasses, 6/12. V.A.L. was Perception of Light only, not improved with glasses. The right eye had a normal appearance. Examination of the left eye revealed a few lenticular and vitreous opacities. The fundus showed (Fig. XXVl) wide spread pigmentary disturbances peripherally/

peripherally of which the predominant shape was half-moon. The macula was replaced by a dusky yellowish area. Peripherally the choroid showed extensive thinning. The calibre of all arteries except the lower nasal was normal. The latter was only 2/3 the diameter of the other arteries and its branches ½ the diameter of the original. The venous calibre appeared to be normal throughout. It is difficult to appreciate the reason for the construction of the lower nasal artery when the disturbances are so wide-spread. Once again the possibility of the presence of a non-traumatic cause for the pigmentary disturbance must be borne in mind but, as before, the fundus of the better eye was of normal appearance and Retinitis Pigmentosa may be ruled out on that account.

Case xxviii.

Pte. M. aged 30 was injured by a football on 30.10.44. The ball bounced and struck his right eye at 11.45 hrs. He was seen ten minutes later when he complained that he could not see objects in the lower half of his visual field. He attributed this to swelling of his lower eyelid but this was not apparent. Homatropine was instilled and he was again examined at 14.15 hrs. By this time he was beginning to see objects in the lower half of his visual field but they were blurred. Ophthalmoscopic Examination of the right fundus revealed/

clearly
revealed (Fig. XXVll) multiple small fairly/defined yellow areas above the macula peripherally. No changes could be made out in the arteries or veins. The peripheral field of vision was by this time full (Chart XVll). He was treated by Gutt. Atropine and examined the next day at 10.00 hrs. The eye felt perfectly normal and the visual acuity and fundus appearance were both found to be normal. The explanation of these small yellow areas is difficult but they had the appearance of multiple small foci of cloudy swelling or oedema. This possibility is strengthened by their transient nature.

Case xxix.

Pte. S. aged 22 was injured by a mortar-bomb splinter on 12.9.44. His face and left shoulder were freely spattered. The visual acuity of the left eye was affected and the left ear became deaf. He was first examined on 10.11.44 when V.A.R. was 6/18 partly and V.A.L. was Perception of Hand movements in front of the eye. The left pupil was eccentric downwards and examination of the left fundus revealed an extensive vitreous haemorrhage with complete obscuration of the fundus details. On 15.11.44 an X-ray plate of the orbits showed a metallic foreign body lying in the left orbit inferiorly . This could be palpated deep to the outer 1/3 of the left lower eyelid. On 10.1.45 V.A.R. : 6/18 : with -0.5 Sphere / - 2.5. Cyl. axis 180°/

180° = 6/6. The left eye was treated with Gutt. Atropine and Dionine and the visual acuity gradually improved. On 18.1.45 V.A.L. = 6/24 with - 1.0 Cyl. axis 180° = 6/9 partly. On 30.1.45 it was 6/9 with glasses. The vitreous opacity gradually cleared and the fundus became visible. At no time was any lesion of the fundus seen and no variations in the vessel calibre could be made out, (Fig. XXVlll). Charting of the peripheral fields required to be done on an improvised Bjerum screen measured out on the wall with the result shown in Chart XVlll. This demonstrated a generalised constriction of the field more marked on the temporal side and due, presumably to interference from vitreous opacities. The left eardrum was found to have a large inferior perforation and on 10.1.45 hearing was reduced to ability to hear the spoken voice at two metres and the whispered voice at one metre.

Case xxx.

Pte. M. aged 44 was injured in the right eye by a piece of steel from a lathe in April 1941. The foreign body was removed the same day by magnet but the visual acuity was affected and has remained so. When examined on 11.4.45 (i.e. four years after injury), V.A.R. was ability to count fingers at one foot and V.A.L. was 6/75 = with-0.5 Cyl. axis 30° = 6/6 partly. The right pupil was irregular, the iris being adherent to the anterior surface of the lens below. Both pupils reacted normally to light and accommodation except, of course/

course, in the area of the posterior synechia in the right eye. A rough test of the right visual field by hand was all that was possible and this demonstrated a defect in the upper and temporal fields whilst the lower field was normal. The intraocular tension felt soft to the fingers. Ophthalmoscopic Examination of the right eye revealed (Fig. XXIX) two small areas of choroidal thinning one below the disc and one to the nasal side of the disc. A small area of pigmentation was present in the former and from the latter a thin band of scar tissue extended forward into the vitreous. Contraction of this scar tissue has apparently detached the retina as a shallow detachment of the retina was present in the lower nasal quadrant. No tear could be seen in this detachment. The arterial and venous calibre was normal with the exception of that of the lower nasal artery which was very slightly narrower than the other arteries. Such a slight disparity, however, is seen in normal eyes and must be discounted as of pathological significance.

Case xxxi.

Lieut. M - Z. aged 28. an officer in the Polish Underground Army was injured on 13.8.44 by a piece of exploding mortar-shell during the battle for Warsaw. The splinter struck his right mastoid process, passed upwards and forwards and came to/

to rest on the floor of the left orbit where it could later be palpated through the lower eyelid. He was first examined on 12.12.44. The right facial nerve and the mandibular branch of the right trigemenial nerve had been damaged, the former being evident by paralysis of the orbicularis oculi and frontalis muscles and the latter by anaesthesia of the right half of the lower lip, the right half of the chin and the right side of the tongue for touch and taste. The motor branch of the trigeminal nerve was intact as shown by unimpeded action of the Temporalis, Masseter and Pterygoid muscles. In the left cheek the sensation of the skin was possibly slightly affected, a slight sensation of tingling being elicited on testing. This was probably due to the fact already mentioned that a large metallic foreign body was lying on the left infraorbital canal in the orbit. The patient complained of deafness in the right ear but he could hear the whispered voice at four metres and the condition was apparently due to some otitis externa. V.A.R. was 4/9 and V.A.L. 4/4.5. Examination revealed that the right pupil was slightly larger than the left and not quite so active to light directly and consensually. Ophthalmoscopic Examination of the right fundus revealed (Fig. XXX) a striated yellowish-white area down and out from the disc on the course of the temporal artery and vein. This had the appearance of a group of opaque nerve fibres but was not so strikingly white or quite so ragged at the extremities. A possibility was that there had been an arterial blockage with swelling of the surrounding retina but the arterial calibre was quite unaffected and it must be concluded/

concluded that the appearances are those of an isolated patch of opaque nerve fibres. Just temporal to the upper extremity of this patch there is a curious little fusiform swelling on a venous twig which is probably of no significance. An X-Ray plate showed a large metallic foreign body lying on the floor of the left orbit where, as previously mentioned, it could be palpated. The patient was admitted to Hospital and on 15.1.45 the foreign body was removed through an incision in the left lower eyelid. At operation a bony splinter was encountered on the lateral side of the foreign body prised up from below like the lid of a box showing that the foreign body had entered the orbit from below. At the conclusion of the operation a probe could be passed into the left maxillary antrum. The path of the splinter was therefore from the right mastoid process upwards forwards to the left through the left maxillary antrum. This consideration makes it very unlikely that the right orbit could have been damaged by this splinter. The visual acuity, however, gradually improved in the right eye. On 17.1.45 it was 4/6 partly and V.A.L. was 4/4.5. On 19.1.45 V.A.R. was 4/6 partly (With + 0.75 Cyl. axis 180°, 4/3.5 partly. The right pupil remained larger than the left but all reactions were brisk. On 7.2.45 V.A.R. was 4/4.5 not improved by glasses and the pupils appeared to be equal. This improvement in the visual acuity and diminution in the astigmatism is interesting and has/

has been noted previously (Case No. v) suggesting a possible concussion and deformity of the globe which has returned to normal. Examination of both peripheral fields revealed no abnormality but a scotoma in the upper nasal field for 5/1000 white was noted on 18.1.45 (Chart XlX). This examination was repeated on 7.2.45 for 2/1000 white when the scotoma was found to merge with the periphery and this remained unchanged (Chart XX). In spite of the slightly dilated pupil and the apparently temporary astigmatism, it must be concluded that the appearances are those of an isolated patch of opaque nerve fibres and the case is included as a control.

Case xxxii.

Pte. S. aged 40 is also included as a control as his fundus appearances are very similar to those of Case xxxi (Fig. XXX). He was a medical case suffering from Trigeminal Neuralgia and "Nervous" Hypertension. As will be seen from Fig. XXXl the patch of opaque nerve fibres is in much the same position as that in case xxxi, on the course of the lower temporal artery and vein but is more wedge-shaped and whiter looking. Variations in the arterial calibre and constriction of the veins at the arterio-venous crossings gives evidence of arterio-sclerotic changes and, in addition, four small punctate haemorrhages can be seen. A diagnosis of "Nervous" Hypertension does not, therefore/

therefore, seem justified. Examination of the central visual fields on 23.4.45 showed a curious result (Chart XXl). The scotoma appeared to be continuous with the blind spot and not, as one would have expected, in the upper nasal field. Examination, however, was not very satisfactory as the patient was a Jugo-Slav and the examination was carried out in the English language.

Summary of Group V.

Six miscellaneous cases are described. The last two (Cases xxxi, Fig. XXX and xxxii, Fig. XXXl) are not traumatic cases but cases of congenital abnormality showing patches of opaque nerve fibres. One (Case xxix, Fig. XXVlll) showed no fundus lesion at all but a large vitreous opacity: One (Case xxx, Fig. XXlX) showed retinitis proliferans with a resulting retinal detachment but no constriction of the vessel calibre: One (Case XXVlll*) showed transient yellow patches which are presumed to be due to small collections of fluid and one only (Case xxvii, Fig. XXVl) showed constriction of an artery. This was accompanied by macular irregularity and widespread peripheral pigmentation.

Discussion.

The above considerations give rise to the following subjects for discussion.

(1) High Explosives may give rise to choroidal haemorrhages and/

* Fig. XXVII.

and, later, exudative choroiditis with constriction of the retinal arteries or veins or both in the same area. (Group 1).

(2) Ruptures of the choroid may also be caused by high explosives with similar constriction of retinal arteries or veins or both in the same area (Group 11).

(3) Where the macular area only has been involved, no arterial or venous changes have been seen (Group 111).

(4) Disturbances in the intraocular tension, either in the shape of hypertension or hypotension, may be caused by high explo-:sives.

The fact that injury to the choroid may produce constriction of the retinal vessels points to a co-ordination between the choroid and the retina. It is well known that there is an anatomical and physiological connection between the choroid and the retina in that the deeper layers of the retina are nourished by the vessels of the choroid and the macular area is devoid of any retinal vessels at all being entirely dependent upon the choroid for its nourishment. Injury to the choroid, therefore, may give rise to death of the overlying retina and we are faced with the question as to whether the vascular constriction is a reflex nervous reaction or is simply secondary to atrophy of the corresponding area of retina.

Let us consider first the question of reflex nervous reactions./

reaction. This is intimately bound up with the question of axon reflexes. Experimental work has shown that these axon reflexes may be of two types - either via the trigeminal nerve as an ordinary sensory nerve response or via the sympathetic nerve supply to the blood vessels of the eye. The response in the former case is the response to trauma and consists of dilatation of the capillaries locally and also in the neighbouring area, increased capillary permeability and increased intraocular pressure (1) In the latter case it consists of capillary constriction and decreased intraocular pressure and constitutes a defense against haemorrhage. (2) These axon reflexes have been studied in relation to the uveal tract (3) where stroking of the iris has produced the "triple response of Lewis" (i.e. a primary and local dilatation of the minute vessels, a local increase in permeability of the capillary walls, and a widespread dilatation of neighbouring arterioles) (4). This triple response is produced chemically by the outpouring of Histamine and has been noted after injury to the cornea, painful subconjunctival injections and, more important to the present discussion, contusion of the globe.(5). In his experimental work on animals, Leplat noted an augmentation of the albumen in the aqueous humour and a rise in the intraocular pressure of both eyes following contusion of one eye. Certain vascular phenomena were also noted. The eye became congested, with injection of the conjunctival, pericorneal and iris vessels. In man Leplat noted a difference in the intraocular pressure after/

after trauma when lying and when sitting, it being higher when lying. This he attributed to a lesion of the intraocular vaso-motor nerves, the regulation of the ocular tonus being normally independent of cephalic congestion due to the lying position. Leplat also quoted a case of increased intraocular tension, following cupping and scarification of the temple which he likened to vaso-motor reactions due to subconjunctival injections, of Sodium Chloride or inhalations of Amyl Nitrite. The rise in the intraocular pressure and the increase in albumen in the opposite eye he explained by a reflex vaso-motor phenomenon through the sympathetic nerve supply. In discussing the hyper-albuminosis, he argued against this increase in albumin being due to cyclitis on the grounds that it followed a rise in the intraocular tension, was seen also in the other eye and could be suppressed by Adrenaline and assumed that it must be due to vaso-motor disturbances which caused congestion of the intraocular blood vessels and consequent transudation.

Somewhat similar results were obtained by Magitot (6) who found that, on ligaturing the vorticose veins of a cat, a rise of intraocular tension could be produced. He also discussed the effect of paracentesis on the human eye. The albuminous content of the aqueous humour was raised and the new aqueous humour was found to be a mixture of serous exudative and aqueous humour. The sudden decompression, he stated, instigated a hyperaemia which modified the osmotic processes and favoured the/

the passage of blood serum into the aqueous humour. Variations in the intraocular pressure occurred after paracentesis and Magitot sought for an explanation of these phenomena. If the carotid artery were ligatured, no change occurred in the intraocular pressure and from this he assumed that the local circulation was at fault and that a considerable vaso-dilatation was produced when the anterior chamber was emptied and this dilatation did not remain stationary but fluctuation occurred and the intraocular tension thus varied. Experimental work on the cat had shown that the eye was soft immediately after pressure upon it but in ten minutes became harder than normal. Ophthalmoscopic Examination of the cat's fundus at this stage showed actual vascular spasm and it was probable that, if a spasmodic state were produced, this would be followed later by turgescence. Schülter was quoted as confirming this and blaming the cervical sympathetic. He found that in an anaesthetised dog excitation of the sympathetic caused a fall in the intraocular tension from 18 mm. to 15 mm. Pressure was then exerted on the globe and the intraocular tension fell to 10 mm. and then rose to 30 mm. Stimulation of the sympathetic reduced it again to 10 after which it rose again to 25 mm. In short, animal experiments all point to the intervention of the nervous system in the results of trauma.

The effect of this nervous intervention, however, is not/

not so easily worked out in the case of the retinal arteries as many different results have been obtained by experimental workers. Magitot & Baillart (7) concluded that in the cat the cervical sympathetic contains vaso-constrictors and that excitation of the cervical cord or of the superior cervical ganglion caused a slight narrowing of the arterial calibre, section of the sympathetic caused a slight dilatation. In the case of the iris the same effects were evident but were more pronounced. These authors state that alone of all workers, Morat and Doyon in 1892 found that, in the cat and dog, excitation of the sympathetic caused an augmentation of the retinal circulation. The reverse, however, was obtained in the case of the rabbit. Duke-Elder, (8) however, quotes Wöllflin as having found in man that stimulation of the cervical sympathetic led to constriction of the retinal vessels in one case whereas in another it had no effect. Duke-Elder sums up, however, by saying that it may be taken that stimulation of the cervical sympathetic leads to an augmentation of the tone and a constriction of the blood vessels in the uveal tract accompanied by a fall in the intraocular pressure while its section leads to their dilatation but that the position of the retina is not so clear. In most cases, he states, the effect appears to be the same but it is by no means/

means invariably so. A reverse effect may be seen and frequently no change can be observed (8).

In the foregoing account of the experimental work, however, it must be noted that Magitot (6) observed vascular spasm in the fundus of a cat after pressure had been exerted upon it. This would lead one to compare the results of the present series of cases with this experimental finding. That axon-reflexes occur in the eye is undoubted (9) but it will be noted that, in the case of the uvea, they result in vaso-dilatat-:ion whereas in the present series the changes are all in the direction of constriction. In no case was there any sign of vaso-dilatation. In a few cases the arterial calibre was unaffected (Case xvi, Fig. XVl: xvii, Fig. XVll: xviii, Fig. XVlll: xix, Fig. XlX: xx, Fig. XX: xxv, Fig. XXV = xxvi = xxviii, Fig. XXVll = xxix, Fig. XXVlll= xxix, Fig. XXVlll & XXX, Fig. XXIX) but the majority showed some form of constriction. The question then arises as to whether this constriction could be due to a nervous reflex in the shape of arterial spasm or whether there is some organic basis for it.

If the constriction is due to spasm then we should expect to find arterial spasm occurring in other parts of the body following gun-shot wounds. This does, in fact, ocurr. In the Hunterian Lecture of April 1943, Cohen (10) stated that the "lash" injury to a vessel was the main cause of the vessel of a limb going into spasm but there is little chance of such occurring in the eye, the retinal vessels of the latter being

encased/

encased in a rigid sclera. The important point is, however, that spasm can occur in a limb and may therefore, occur in an eye. The permanence of the constriction in the present series, however, is a point against it being of spasmodic origin but Cohen (10) points out that in John Hunter's experiments on the vessels of a severed placenta the vessels did not open up until the third or fourth day and that, in his own experience, arterial spasm had been noted in the amputated limb. These points would indicate that the spasm may at least be of long duration.

Turning now to Cohen's opinion on the causation of arterial spasm, we find that he attributes it not to a sympathetic nerve reflex but to the muscle inherent in the arterial wall which has natural contractile properties. The type of injury of which he is speaking is the "near miss" of an artery in a limb which bears some resemblance to the type of injury in the present series. In the eye, however, there is another important point, namely that the constriction occurs in the arteries and veins of the retina whereas the choroid is apparently the structure primarily involved and it is difficult to avoid the conclusion that a nervous reflex must be involved. Sympathetic fibres have been traced to the retinal vessels via the nerve of Tiedemann but, as already stated, their physiological action is not so certain and the triple response of Lewis to injury seems to point to dilatation rather than constriction as a normal response. One cannot, however, deny the close anatomical and physiological relationship between/

between the choroid and the retina, the deeper layers of the latter depending on the former for their nourishment, altho' there is no anastamosis between the circulation of the two layers apart from a problematical anastamosis round the optic disc. This further strengthens the case for a nerve reflex being involved. Turning again to Cohen's work on arterial spasm in the limb, there is an interesting account of the effect on the cutaneous circulation when a limb is damaged. He states that the sympathetic nerves have the important function of keeping the cutaneous circulation closed in order to divert as much blood as possible to the site of injury and thus avoid ischaemic contracture in the damaged muscles. If there is an intimate connection between the circulation of the choroid and that of the retina, one might postulate a contraction of the retinal circulation in order to try to divert as much blood as possible to the damaged choroid.

The evidence, therefore, appears to point to the intervention of a nervous reflex in the changes in the retinal vessels described. If we are right in this assumption, then lesions of the macula ought to be in a somewhat different category owing to the absence of retinal vessels at the macula and this is borne out by the present series. It is unfortunate that no case in the present series showed extensive enough damage to the choroid in the macular region to produce a choroidal tear when it might have been possible to trace constriction of the/

the macular twigs. There is, of course, the possibility that the reflex might work in the opposite direction in those cases, such as a macular "hole" occurring, in which the underlying choroid appears to be quite healthy, but there is no means of telling, short of microscopic examination, whether the choroidal vessels are constricted or not. If, however, the reflex works from choroid to retina then it must be of very local extent since the visible macular twigs are not apparently involved which does not fit in with the discription of the triple response of Lewis in the uvea where there is a neighbouring flare as well as a local dilatation.

Turning to the question of changes in the intraocular tension, it has already been noted that experimental work has proved the trauma exerts an important effect on the intraocular tension (1) (5) (6). Duke-Elder (11), having reviewed the various theories as to the causation of traumatic glaucoma concludes that only one theory is possible stating that "The Primary mechanism is undoubtedly an upset of the local nervous control of the circulation, any disturbance of which is generalised over the entire uveal tract by axon reflexes". In favour of this theory he quotes the behaviour of the uninjured eye following trauma to one eye described by several authors. As has already been described, Leplat (5) found a rise in tension in the opposite eye after contusion. It has been found experi-:mentally by Duke-Elder that paracentesis of the rabbit's cornea also causes a rise in pressure about ten minutes to half/

half an hour later followed by a fall after a further twenty minutes and then a gradual rise during the next two hours (12). He also quotes Magitot and Baillart and others as having studied similar effects following the application of pressure to the globe which is precisely the type of injury with which we are dealing in the present series. The first rise in intraocular pressure Duke-Elder ascribes to capillary dilatation and a very rapid transudation of fresh plasmoid intraocular fluid. This rise falls fairly rapidly and the second slower rise in pressure is due to difficulty in obtaining adequate circulation through the drainage channels. As these are the normal reactions to paracentesis and application of pressure to the globe in rabbits he sees no reason to suppose that, in this exceptional case, the permanent state of increased pressure in man need be due to anything but the same mechanism. On the question of hypotony, Duke-Elder is equally dogmatic (13). He quotes Magitot as having found experimentally that the hypotomy is preceded by a rise in pressure but points out that in clinical conditions either a rise or a fall may occur. Just as a rise in pressure has been observed in the uninjured eye following a rise in pressure in the injured eye so has Magitot (14) found hypotony occurring reflexly in the uninjured eye when the injured eye is also in a state of decreased intraocular pressure. Duke-Elder once again reviews the various theories of causation but concludes that "It is probable that the most potent cause is a disturbance of the circulation through the/

the local nervous axon-reflex mechanism, which governs the condition of the small vessels and is regulated by axon reflexes through the ganglionic nerve system in the choroid, a factor which accounts not only for the upset in the tension of the contused eye but also explains the similar reflex phenomena in its fellow". It is interesting to note the mention here of the ganglion cells in the choroid since the choroid is apparently the structure mainly damaged in Groups 1 and 11 of the present series.*

Let us consider now the train of events which must follow an injury such as is described in the present series. When pressure is applied to the eye from without, intra-ocular fluid is forced out of the eye through the canal of Schlemm and the pressure falls. As we have already seen, however, paracentesis causes a rise in pressure following this fall and a slight decrease later on, the pressure varying somewhat. In the case of a high velocity projectile such as a rifle bullet, there must be a phase when the pressure exerted on the eye as the projectile crosses the orbit causes very high pressure within the eye with which the Canal of Schlemm cannot deal all at once. It is possible that this pressure may be sufficient to exsanguinate the eye and the arteries may collapse and not recover. This would account for the constriction of certain retinal arteries but it is difficult to understand why this would account for the constriction of one artery which is in the same area as a choroidal rupture or choroidal haemorrhages.

It/

* With the exception of Case XV Fig. XIV.

It might, however, account for the constriction of the lower nasal artery in one case (xxvii, Fig. XXVl) in which the retina appeared to be extensively damaged and in which only one artery was constricted.

The question to be decided now is whether a theory of axon reflexes with vascular spasm is sufficient to explain the pnenomena. The reaction of the retinal vessels to trauma has not been exactly determined but vaso-constriction may be the result. This, however, must be in the nature of spasm and the question of the permancy of this constriction again arises. It is, however, quite conceivable that a spasm, once it has become fully established for sometime, could cause a permanent vaso-constriction.

A review of various drawings of choroidal injuries due to gunshot wounds shows that Würdemann (14) illustrates one case of "Ruptura Choroidalis et retinae, lesio contussionis" in which the inferior temporal artery is obliterated. There is an extreme choroidal rupture involving the foveal region and retinitis proliferans in the lower part of the optic disc. He does not, however, comment on this case in his text. Lagrange also (15) illustrates concussion lesions in which the vessels appear to be constricted but he does not comment on the fact in the text. Four illustrations in particular (Plate lll Fig. 3, Plate lV Figs. 1 and 3, and Plate V. Fig. 3) showed marked vascular constriction.

It/

It will be seen, therefore, that there are objections to the theory of axon reflexes causing this vascular constriction, notably that the response to trauma in the anterior uvea is one of vaso-dilatation rather than vaso-constriction and it is necessary to discuss other theories of causation.

In his lecture on arterial spasm due to gunshot wounds, Cohen (10) stresses the importance of dilatation of the deep arteries in the repair of muscle trauma rather than dilatation of the capillaries to the skin, pointing out that it is the function of the skin capillaries to become constricted and so divert more blood to the injured muscles. One of the features of the present series of cases is the fact that the part of the eye which is primarily injured is the choroid and that the vascular constriction occurs in the arteries or veins of the retina. It would be an attractive theory to suppose that the retinal arteries or veins become constricted in order to divert more blood to the damaged choroid. Where the retina only is involved as in detachment of the retina (e.g. Case XXX Fig. XXIX) there is found no arterial or venous constriction but, again, in cases of choroidal atrophy following choroiditis no arterial or venous constriction is in evidence except in the later stages when optic atrophy has supervened. On this account, this theory becomes of less value.

Another/

Another feature of the present series of cases is the almost complete absence of evidence of an organic vascular obstruction. One certain exception is Case xiv (Fig. Xll). In this case there is a visible blockage of the superior temporal veins with haemorrhages along their course. One other case (xv, Fig. XlV) demonstrates a curious little somewhat fusiform swelling on the course of a small tributary of the upper nasal vein. The origin of this swelling is doubtful but it might be due to thrombosis. One objection to this statement might be that it is far removed from the site of injury, but it must be pointed out that the obstruction in the first case is also somewhat remote from the choroidal ruptures although much closer to the macular hole. In the vast bulk of the cases, however, the classical picture of venous thrombosis with its dilated, tortuous veins and its multiple haemorrhages or of arterial embolism with its retinal oedema, narrow arteries and cherry-red spot is absent. (But see ref. 26.) Objection might be taken to the last statement in view of the fact that arterial embolism leaves little trace after some months except optic atrophy and arterial constriction but it must be repeated that the structure primarily involved is not the retina but the choroid and it is difficult to imagine now a rupture of the choroid or a choroidal haemorrhage could cause embolism of the artery. We have, however, in at least one Case (xiv, Fig. XII) evidence of thrombosis of a venous tributary and it is necessary now to see whether sucha lesion/

lesion has been found in any other part of the body followin- blast. Pinnock & Wood (17)have described examination of a number of men injured in the water by a depth charge. Haematemesis and bleeding from the rectum occurred in some cases and at necropsy in one case, the appearances suggested thrombosis of a mesenteric vein. Zuckerman (18) in his experimental work on the effects of blast on the rabbit does not describe thrombosis in any part of the pulmonary circulation but describes pulmonary haemorrhages. The tubes and alveoli are filled with blood and the alveoli are ruptured. He states that in man the findings are somewhat different. There is intense arteriolar and capillary dilatation with exudation of fluid into the alveoli. It seems, therefore, that thrombosis is an exception following blast injury but that it can occur as shown by the single case in this series and the appearances at necropsy of the man injured by the depth charge.

It will be noted, however, that Zuckerman (18) has noted appearances similar to those obtained in the anterior uvea on stroking the iris, namely arteriolar and capillary dilatation and exudation of fluid into the alveoli. Somewhat similar appear- ances have been noted in the case of the ear. Korkis (19), describing the effect of blast on the human ear, described blast hyperaemia of the tympanic membrane and the possibilities of lesions of the inner ear. In the latter there is no visible macroscopic pathology of the middle ear and the deafness is of

the/

the nerve type. He conjectures that haemorrhages occur in the inner ear or a tissue oedema is produced. He suggests that histamine is responsible for local vasodilatation with resulting fluid exudation into the tissue spaces of the inner ear consequent upon the increased permeability which is part of the triple response.

Here again it must be noted that the changes are all in the direction of vascular dilatation and not constriction.

A further possibility which must be considered is that the vascular constriction is secondary to the death of a part of the retina. It must be admitted, however, that few of the present cases showed evidence of optic atrophy. A possible exception is Case v, Fig. II in which there was well marked temporal pallor without nasal pallor. In some cases the whole nerve showed pallor (Case xi , Fig. VIII, Case xiii, Fig. XI, Case xx, Fig. XX, and Case xxi, Fig. xxii). It is well known that vessel changes occur in optic atrophy such as is caused by head injury and it is interesting to note that Duke-Elder (20) regards the efferent fibres of the optic nerve as vaso-motor in function. Commenting on this, Rodger (21) discusses the presence of these fibres in the optic nerve and inclines to the view that the fibres run as a solitary bundle but expresses no views upon their distribution in the retina. If, however, they should become atrophied with the other efferent fibres, one would expect a dilatation of the arteries and not a constriction.

Rodger/

Rodger is, however, of the opinion that it is more likely that the vessel changes in optic atrophy are the result of a post-degenerative gliosis, the glia proliferating and constricting the lumina of the neighbouring vessels. It is possible that such is the case in the present series, but it does not explain the sudden constriction seen in Case i, Fig. I. nor does it explain why, in Case xv , Fig. XV the vein leading from the foreign body is constricted and yet the field of vision does not show a sector defect, but rather a generalised constriction of the field. Rodger further comments on the absence of changes in the veins, the constriction being confined to the arteries. Several cases, notably vi, Fig. III and xv, Fig. XV, in the present series, however, demonstrate that a well-marked venous constriction may occur. It has already been stated that there is no indication as to how the efferent fibres of the optic nerve are distributed in the retina but Rodger mentions the possibility that degenerating nerve fibres might irritate these vaso-motor fibres, if vaso-motor they be, and thus cause vascular constriction. On the same assumption localised degeneration might cause localised vascular constriction but vascular spasm from an axon reflex appears to be a simpler explanation.

In a search for any other ocular condition which might give rise to a similar appearance of vascular constriction associated with a choroido-retinal lesion, a similarity was found in drawings of cases of angioma and glioma retinae treated with radon seeds. Foster Moore (22) has described the obliter-

ation/

obliteration of the tortuous vessels in an angioma of the retina. Griffith (23), however, describes the cure of a fresh growth of glioma by radon seeds but states that no sclerosis or thrombosis of vessels could be seen. On the other hand, Stallard (24) has shown that necrosis may occur in sarcoma of the choroid when treated by radium accompanied by obliteration of the blood vessels. In the vicinity however there is dilatation of the blood vessels due, he states, to temporary paralysis of the cells of Rouget. On the subject of Glioma of the Retina, however, Stallard (25) shows that at the conclusion of treatment the arteries supplying, and the veins draining, the affected area are obliterated. In angiomatosis retinae also he describes the sealing off of the dilated tortuous vessels probably, he states, by obliterative endarteritis. He does not, however, discuss the possibility of vascular spasm. Here, however, we have a strong chemical action on the blood vessels and the conditions are not really comparable unless one compares the action of histamine which is released when an axon reflex comes into operation, in which case the result is vaso-dilatation and not vaso-constriction.

It has already been stated that the picture of embolism of the central retinal artery has not been seen in the present series, Doherty (26), however records a very interesting case in this connection. The patient was struck on the eye by a metal spring and complained of defective vision. A drawing was made 24 hours after the injury and reproduced in Doherty's article. It shows a typical cherry red spot at the macula with oedema of the surround-

ing/

surrounding retina but no vascular changes are visible. Below towards the periphery a small haemorrhage is seen. The Picture gradually changed to one of traumatic exudative choroiditis in place of the haemorrhage and a macular hole. The interesting feature of the case is the cherry-red spot unassociated with any vascular change, strongly suggesting a spasm of the artery which has become relieved. If this is so, then the association of spasm with the traumatic exudative choroiditis supports the theory of axon reflexes in the present series.

SUMMARY OF PART I.

Thirty two cases of injuries to the eye are described. In the great majority some form of vascular constriction is present. In some cases glaucoma or a hypotony has supervened. It is postulated that the cause of these vascular phenomena and disturbances in intraocular pressure is a local axon reflex set up by the injury.

PART II.

SOME VASCULAR DISEASES OCCURRING IN BRITISH AND ALLIED PRISONERS OF WAR DURING CAPTIVITY.

In Part II six cases are described and discussed. Three are of Periphlebitis Retinae, one of Periarteritis, one of Acute Choroiditis and one of Heterochromic Cyclitis.

DESCRIPTION OF CASES.

Case xxxiii.

Pte. C., a New Zealander aged 28 was captured in May 1941 and first began to have symptons on 12th November, 1943, when he woke up to find that he could not see with his left eye. He was examined four days later when V.A.R. was 6/6 and V.A.L. ability to count fingers at two feet. Examination of the left fundus was not possible owing to a large vitreous haemorrhage. Two days later ophthalmoscopic examination of the right fundus revealed a small circular haemorrhage far out to the nasal side with a small vein above and below. These two veins were joined by a tortuous, cork-screw-like new vessel. A few smaller haemorrhages were visible far out on the temporal side towards the periphery (not shown in the diagrams). A few details of the left fundus could be made out in the form of tortuous veins and some haemorrhages. A diagnosis of Periphlebitis Retinae was made and general investigation carried out. Blood Pressure, Blood Count, Blood Sedimentation Rate, Chest Screen, Ear Nose and Throat, Urine, Wassermann Reaction and Mantoux Test were all normal. In short, no pathological changes/

changes could be observed in any other part of the body. The vitreous of the left eye gradually cleared but haemorrhages recurred on 23.2.44., 19.4.44., 27.5.44., 10.7.44., and 3.8.44. The right eye remained unaffected until 27.7.44. when a haemorrhage occurred into the right vitreous. On 20.8.44. a second haemorrhage occurred and a few days later the patient was repatriated to England. He wrote from there saying that his eyes were keeping very well but no medical report was available.

He thus gave a typical history of recurrent vitreous haemorrhage in both eyes.

The treatment given was a course of fourteen subcutaneous injections of Old Tuberculin extending over three months and a course of five subcutaneous injections of Karan (Vitamine K.) 1.0 c.c. per day for five days. It cannot be claimed, however, that any improvement occurred.

DETAILED DESCRIPTION OF THE LEFT FUNDUS.

As the first haemorrhage cleared, many retinal haemorrhages could be seen and a month after the onset, the picture presented was as shown in Fig. XXXII. The upper nasal vein had a normal appearance at the disc but the tributary which came down from above at "12 o'clock" showed a very complicated system of looping and branching as if the vein had divided into two and as if the temporal division had formed a small loop forwards. A similar looping could be seen just below, nearer the disc. Immediately above this system of looping was a single vein formed by the junction of two smaller veins. The nasally situated vein/

vein showed multiple small swellings in its course suggestive of thrombi but no such swellings could be seen on the more temporally situated vein. Where these two small tributaries joined could be seen a somewhat pear-shaped reddish swelling, suggestive of a haemorrhage but it will be seen later that this became another small system of loops and branches. From both sides of the vein and its tributaries at 12 o'clock numerous small twigs passed out to the two neighbouring arteries establishing at some points a collateral circulation.

Six days later (Fig. XXXIII) the picture had changed somewhat. The large system of looping and branching had become more elongated and more complicated. Immediately below it and hiding the small loop previously noted, the collateral vessels had become bunched together and were pushing forwards into the vitreous. The retinal haemorrhages were fewer in number.

Six weeks later (Fig. XXXIV) the picture had further altered. The system of looping and branching had become still more elongated downwards and the bunch of vessels forming the collateral circulation below it had become less bunched and was attached to its nasal side. The pear-shaped swelling above was now showing signs of dividing up into smaller branches and the collateral circulation was, on the whole, less.

Seven weeks later (Fig. XXXV) the whole course of the vein from the looping upwards was showing signs of pushing itself forwards into the vitreous in a fold. The system of looping and branching had become more elongated still and the looping

and/

and branching above had become more obvious. A second haemorrhage into the vitreous had occurred a month previously.

The lower nasal vein also showed abnormalities on its course (Fig. XXXVI). A large, somewhat fleshy looking area became apparent on its peripheral part. This was apparently composed of multiple small blood vessels and some branches of the upper nasal artery came down towards it, but whether they established any contact with the new vessel-formation is problematical.

The whole fundus picture can be seen in Fig. XXXVII which, along with the previous figure were drawn the day before the third haemorrhage into the left vitreous. It will be seen that there were three main groups of new-vessel-formation. The topmost one at 12 o'clock has been described already in its earlier stages but now it has pushed itself into the vitreous to a greater extent and the lower, larger system of looping and branching has become still more elongated. The upper, smaller system has become a little more complicated. The group on the temporal side at 2 o'clock has not been previously described but had a broad base in which small vessels could be seen coming forward into the vitreous. From its lower tip came a narrow black band which terminated below in a grayish-white fold. This fold was in turn connected by a second black band to another fold. No blood vessels could be seen in either of these folds. From the second fold, which lay below the disc at 6 o'clock a black band extended upwards, sent a small prolongation to the optic disc and terminated in a sausage-shaped black mass which had the appearance/

appearance of attempting to establish contact with the vascular system at 2 o'clock. The more nasally situated vascular system at 9 o'clock has already been described.

Four weeks later (Fig. XXXVIII), the vascular systems at 12 o'clock and at 2 o'clock had begun to join up and blood vessels had appeared round the sausage-shaped termination of the black band extending upwards towards the disc. Vessels had also appeared in the more temporally situated white fold below. The nasally situated vascular system at 9 o'clock had extended somewhat but not sufficiently to make contact with the other systems. The whole effect was of attempts at joining up of collateral circulation, the black bands previously noted apparently acting as scaffolding for the new vessels as they grew. Twelve days after Fig. XXXVIII was drawn, a fourth vitreous haemorrhage occurred. The fundus picture became increasingly complicated and two more haemorrhages occurred, but a month after the last one, the fundus was as shown in Fig. XXXIX. The blood vessels appeared to be reduced in number, although the whitish folds were more in evidence. The vascular system at 9 o'clock was more extensive and the black band at 6 o'clock was thicker but showed less vascularisation. The vascular systems at 12 o'clock and at 2 o'clock had a degenerate appearance but were still linked up.

The patient was repatriated shortly after this but it will be seen that the pictorial record extends over a period of almost nine months.

Such was the picture of the more seriously affected eye/

eye but the earlier changes could be followed in the right eye..

DETAILED DESCRIPTION OF THE RIGHT FUNDUS.

The lower temporal vein showed striking changes and the details will now be considered. Fig. XL shows looping of the vein. A loop is formed in the first place and the vein becomes doubled on itself. This is followed by a straight portion when another loop is formed but this time the vein is not completely doubled on itself. This is followed by a third rather complicated loop. The first and second loops are linked together by three small collateral blood vessels.

Further along its course the vein gives off a little tail somewhat cork-screw in shape and later divides into many small branches which join up again into one small vein which ends in a dark area like a haemorrhage.

Ten weeks later (Fig. XLI) the picture had changed somewhat. The original loops had all disappeared and multiple small vessels had taken theirplace as if the original vein had divided up into numerous smaller vessels. These had the appearance of trying to establish contact with the vein extending upwards. Further along the course of the vein, the cork-screw-like tail had become more globular in shape and the divisions which had originally joined up again into one vessel had become more elongated and more complicated.

About eight weeks later the picture had not changed to any great extent (Fig. XLII).

About six weeks later, however, (Fig. XLIII), the branch-

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branchings had become more extensive and the changes further along the vein had taken on a new appearance. The little cork-screw area which had become globular now took on the appearance of a vascular loop, reminiscent of the original looping further nasally. Beyond this loop the vein appeared to give off many small vessels which collected together in a downward direction to form a small vein.

About six weeks later, (Fig. XLIV) the original branchings had the appearance of receeding from the vein going upwards and it seemed doubtful whether they were establishing contact at all. The loop further along the vein was a little wider and the branchings beyond were more bunched and circular.

Five weeks later (Fig. XLV), two weeks after the first vitreous haemorrhage in that eye, the picture was substantially the same but several large retinal haemorrhages could be seen in the vicinity of the temporal end of the vein.

The changes in other parts of the right fundus are shown in the next four Figures (XLVI to XLIX) which cover roughly the same period of time. Leaving out consideration of the lower temporal vein, which has already been described in detail, Fig.XLVI shows that the main changes were in the direction of retinal haemorrhages and splitting of the peripheral veins. A small vein at 12 o'clock showed small swellings on its course which strongly suggested multiple thrombosis. At 3 o'clock the lower nasal vein was beginning to split up into smaller branches and two small cork-screw vessels from its upper tributary appeared to be making efforts/

efforts to establish communication with the upper nasal artery. Five months later (Fig. XLVII) the retinal haemorrhages were less in evidence but two white areas had appeared above (Near the limits of the Figure). That on the temporal side coincided with the termination of a curious system of vessels which drained into the upper temporal vein. A series of small veins joined up to form a small trunk which immediately split into two and joined up again to drain into the superior temporal vein. The white area on the nasal side had a small vein emerging from its lower end which was connected to a small globular collection of veins. The lower nasal vein showed more extensive branching at its nasal end.

Seven weeks later, the picture had not altered materially but it could be seen (Fig. XLVIII) that a small terminal branch of the superior nasal artery had established contact with the globular mass of veins on the nasal side. Another new feature was in the more temporal of the two veins at 12 o'clock which showed a small tributary with a claw like termination apparently ending blindly in the fork formed by the two branches of an artery.

Five weeks later (Fig. XLIX), two weeks after the first vitreous haemorrhage in that eye, the picture was substantially the same but the various complicated systems of vessels showed further divisions.

SUMMARY OF CASE xxxiii.

The case is one of recurrent vitreous haemorrhages with periphlebitis retinae in both eyes. The age of the patient is/

is that usually associated with the disease. Search for a septic focus was unfruitful. W.R. was negative and he had no signs elsewhere of Thrombo-angeitis obliterans.

The left eye was more markedly affected than the right, having had six vitreous haemorrhages as opposed to two in the right. Moreover the changes were somewhat different in the two eyes. In the left eye the tendency was for complicated looping and branching to occur whereas in the right eye, branching only took place except in the case of the lower temporal vein. The various systems of branching and looping in the left eye showed a tendency to push forward into the vitreous but there was no such tendency in the right eye . The earliest changes were seen in the lower temporal vein where looping occurred first, followed by division of the original trunk into many smaller vessels with attempts at collateral circulation.

No treatment seemed to have any effect on the condition.

Case xxxiv.

Sergt. M., a New Zealander aged 26 was captured on 15th July 1943 and began to notice gradual blurring of the vision in the right eye from the nasal side outwards about March 1944. After two weeks the vision became quite blurred and remained so. He noticed no change in the visual acuity of the left eye at any time.

The patient was first examined on 6th June 1944 i.e. about three months after he first noticed the defective vision in the right eye. The V.A.R. was ability to count fingers at two feet/

feet and the V.A.L. 4/6. Ophthalmoscopic examination of the right fundus revealed multiple retinal haemorrhages and coarse vitreous opacities. The vitreous opacities cleared somewhat and allowed of a better view of the fundus but the visual acuity did not improve. He was admitted to Hospital and given Gutt. Atropin. Sulph. 1% once daily. A general examination revealed an elevated Blood-Sedimentation Rate of 19 in the first hour and 40 in the second and a slight elevation of the Blood Pressure to 170/88. The differential blood-count, cardio-vascular system and accessory sinuses were all normal. The Kahn, Citochol and N.K.R. reactions were all negative and the teeth were in good condition. Urine was normal. His past history was somewhat uneventful. He had measles, whooping cough and chicken-pox in childhood and jaundice and a poisoned leg when a boy.

In February 1944, i.e. one month before noticing his symptoms, he began to be troubled by swelling of his legs above the ankles which he attributed to physical training and rugby football. This swelling subsided after a short rest.

His family history was unhelpful. His father died at the age of 60 from cancer of the jaw. His mother, three sisters and one brother were alive and well but one sister had died aged 13 in 1944 from blood poisoning.

Physically the patient was a very fine specimen.

He was kept in Hospital for five months during which time there was a gradual deterioration of vision without any very dramatic change for the worse. In October, however, he complained that the vision seemed to be somewhat worse and ophthalmoscopic/

ophthalmoscopic examination showed some increase in the vitreous opacities which cleared up somewhat but on 2nd November, when he was discharged from hospital, the V.A.R. was Perception of Hand Movements only and the V.A.L. was 4/3.5.

DETAILED DESCRIPTION OF THE RIGHT FUNDUS.

In view of the vitreous opacities it was difficult to study the finer changes at first but it gradually became possible to gain some idea of the picture presented. Fig. L. shows the state of the fundus on 22nd July. Where one would expect to find the optic disc one could make out dense vitreous opacities with a complicated vascular swelling to its temporal side. As this was semi-transparent, it was presumed to be composed of blood vessels almost entirely. A small haemorrhage was present on its anterior surface. Five fair sized veins radiated from this swelling as centre, one at 10 o'clock one at 12 o'clock, one at 1 o'clock, one at 2 o'clock and one at 4 o'clock.

The vein at 10 o'clock showed a vascular "bud" on its course with an artery below it which had the appearance of connecting with it but which, in the light of later findings, almost certainly did not actually establish connection with it. The retina around this vascular "bud" was very pale.

The vein at 12 o'clock showed a larger vascular "bud" on its course and the vein could not be traced above this, its place being taken by multiple retinal haemorrhages whichprobably concealed its upper reaches. Between this vein and the vein at 1 o'clock,/

1 o'clock, lay an artery which forked at the same level as the vascular "bud". In this fork lay two haemorrhages and the more temporally situated limb of the fork had a curious interruption in its course where a small branch came off. It is suggested that this interruption was caused by some swelling of the retina which merely hid the vessel as the artery could be traced onwards above it.

The vein at 1 o'clock showed only a small vascular "bud" on its course.

The vein at 2 o'clock was made up of two tributaries, the upper one of which showed an extensive vascular "bud" joined by a small vein from the temporal side and above, which had a smaller vascular bud at its visible extremity. The lower tributary appeared to arise at its periphery from a dark circular haemorrhage and to be joined from above by a smaller tributary with swellings on its course. Above the dark circular haemorrhage was a smaller haemorrhage which appeared to have a small artery running to it, and a small artery could be seen below the larger haemorrhage having its origin from the lower nasal artery. To the nasal side of these two haemorrhages could be seen a band of smaller retinal haemorrhages stretching upwards and downwards.

The lower vein at 4 o'clock showed two vascular "buds" on its course and then, lower down, a small loop from which small collaterals appeared to join up with the lower "bud". Two small collaterals also stretched between the two extremities of the loop. Lower down the vein appeared to be split in two and then rejoin/

rejoin or else cross like a letter "X". The lower limb of the "X" on being followed downwards showed a sudden constriction and later a series of little loops which ended in a small kidney-shaped complete loop. These loops, particularly the larger one, were very reminiscent of the loops seen in the early stages in the right eye of Case xxxiii (Fig. XL).

Fig. L I shows the fundus picture on 14th August. The optic disc had now become visible and it could be seen that most of the veins were distended, particularly the vein at 10 o'clock which was also tortuous. The large vascular swelling to the temporal side of the optic disc had become more extensive and still semi-transparent. The vein at 10 o'clock when traced temporally also showed a more extensive vascular "bud" which was quite distinct from the artery below. It was impossible to say whether the "bud"established connection with the larger vascular swelling or not.

The vein at 12 o'clock could still not be traced beyond the vascular "bud". This latter had become more rounded in outline and somewhat resembled an open parachute. The interrupted artery which was originally on its nasal side now appeared above it and was still interrupted in its course. The small branch, however, showed a few little vascular dots round its extremity and two small tributaries of the vein at one o'clock opposite showed similar dots. These little dots were suggestive of aneurysmal dilatations on the course of newly formed vessels in an attempt to establish a collateral circulation.

The vein at one o'clock could now be traced further upward.

The vascular "bud" originally seen was substantially unchanged but above it could be seen two tributaries, the more nasally situated of which had a smaller vascular "bud" on its course from which a small tributary appeared to emerge to join the more temporally situated vein. This last appeared to make connection with the neighbouring artery but might simply have crossed it.

The vein at 2 o'clock was substantially unchanged apart from slight extension of the vascular "bud". The two dark circular areas which looked like haemorrhages appeared to have merged and to be connected with arteries both above and below.

The vein at 4 o'clock could not be traced very far owing to vitreous opacities but the vascular "buds" could not be seen and the visible extremity showed multiple branchings into tortuous dilated tributaries.

The left fundus remained normal throughout.

He was treated by six weekly injections of "Pyrifer", a German preparation which induced Protein Shock.

SUMMARY OF CASE xxxiv.

The case is one of unilateral haemorrhage into the vitreous with Periphlebitis Retinae. The findings differ from those in case xxxiii in that they were confined to one eye and did not show the same tendency for the haemorrhages to clear up and then recur. In addition, the changes in the veins were more marked centrally than peripherally, whereas in Case xxxiii they tended to be more marked peripherally. As in case xxxiii the/

the patient was a young healthy adult New Zealander of very good physique in whom no abnormalities could be found apart from his eye.

Treatment by Protein shock appeared to have no effect on the condition.

Case xxxv.

W.O.S., a New Zealander aged 29 was first seen on 10.11.44. with the complaint that two days previously the left eye had suddenly gone blind. When examined the visual acuity had already recovered to 6/12 partly. The right eye showed a visual acuity of 6/6. The left pupil was larger than the right but reacted normally to light. Ophthalmoscopic examination revealed a cloudy vitreous, particularly in the lower half which obscured any view of the lower blood vessels. No abnormality could be made out in the visible vessels. He was given Gutt. Atropin. Sulph. and the vision improved until 17.11.44. when sudden blindness recurred in the left eye. He was examined four days later when V.A.R. = 6/6. partly and V.A.L. = ability to count fingers at one foot. Ophthalmoscopic examination of the left eye revealed a large vitreous haemorrhage in the left eye and he was admitted to hospital on 23.11.44. The visual acuity rapidly improved with Gutt. Atropine and Dionine and on 27.11.44. V.A.L. = 4/6 and on 4.12.44. V.A.L. = 4/4.5. By this time it was possible to obtain a good view of the left fundus which was as shown in Fig. LII.

DETAILED DESCRIPTION OF THE LEFT FUNDUS.

The only abnormal vessel appeared to be the lower temporal vein/

vein. On tracing this down from the disc a dark elliptical haemorrage could be seen overlapping its course. Below this two tributaries could be seen, the upper of which was crossed by a white area containing multiple small branching veins beyond which the vein split up into multiple fine tributaries. From the lowest point of this white area a cork-screw-like vessel emerged apparently to make connection with the lower tributary. This lower tributary appeared to divide and then re-unite. A second white area bearing a few blood vessels lay below this tributary connected with it by a few fine vessels. In the vitreous at 6 o'clock could be seen a grayish-white opacity. Above and below the two tributaries could be seen a small branch from neighbouring arteries. No definite connection could be seen between them and the veins.

Three weeks later (Fig. LIII), the picture had become somewhat more complex. The elliptical haemorrhage had become much smaller. The two white areas did not appear to be so vascular but the terminal branchings of the two tributaries were much more complex. The two small arteries were still present above and below but no connection could be made out with the veins. The vitreous opacity was unchanged.

Two weeks later (Fig. LIV) the affected area was showing signs of pushing forward into the vitreous and had now the appearance of having folds on its surface. The ellipitcal retinal haemorrhage had disappeared and the tendency for more complicated branchings and anastomosis was more marked. A small dark area on these anastomoses suggested a possible/

possible haemorrhage. The two white areas and the grayish-white vitreous opacity were unchanged. The right fundus remained normal throughout. General investigation once more proved negative and family history was normal. Chest Screening was negative for Tuberculosis. Kahn, Citochol and N.K.R. tests were negative. Nasal air sinuses were all normal and the Blood Sedimentation Rate was 1st hour 2, 2nd hour 10. A course of subcutaneous injections of Tuberculin was started but not completed owing to the War situation and the patient was moved elsewhere.

Summary of Case XXXV.

The case is one of early unilateral recurring haemorrhage into the vitreous with Periphlebitis Retinae. Two haemorrhages occurred with only nine days between them and no recurrence had taken place for two months. The periodicity was similar to that in Case xxxiii and the complete absence of abnormalities elsewhere in the body allies this case to the two previous ones, xxxiii and xxxiv. Like the two preceding cases he was still in the twenties and a New Zealander.

The fundus lesions resemble those seen in Case xxxiii more than those in Case xxxiv in that the tendency is for branchings rather than vascular "buds", and the vascular areas tend to push forward into the vitreous.

DISCUSSION.

These three cases bear a resemblance to one another in the occurrence of vitreous haemorrhages and the evidence of abnormalities/

abnormalities in the veins. For these reasons they have been diagnosed as cases of Periphlebitis Retinae.

A consideration of the three cases brings out several points.

(1) The changes were confined to the veins with the exception that

(2) Neighbouring arteries were seen to send out branches to try to re-establish the circulation in all three cases.

(3) Haemorrhages occurred into the vitreous on more than one occasion in cases xxxiii and xxxv and on one occasion in case xxxiv with a doubtful recurrence.

(4) All the patients were under thirty years of age and all came from New Zealand.

(5) No familial or congenital factor could be made out.

(6) No evidence could be found of Tuberculosis, Syphilis, focal infection or vascular disease elsewhere.

(7) Treatment did not appear to affect the course of the disease.

One striking feature in Case xxxiv was the complete absence of signs of disease in the left eye in spite of the advanced state of the changes in the right eye. Case xxxiii showed changes in both retinae but case xxxv was unilateral, possibly because the disease was in its early stages.

Periphlebitis Retinae is a disease of young healthy adults, a statement which is substantiated by the present series. The generally accepted causes, if any, are according to Duke-Elder (27), (a) Tuberculosis, (b) Septic foci and (c) Thrombo-angeitis obliterans/

obliterans (Buerger's Disease), but he admits that many cases occur in which no cause can be found. Two of the present cases (xxxiii and xxxv) were treated by Tuberculin and one (xxxiv) by Protein Shock, but neither line of treatment appeared to have any effect on the course of the disease. Vitamine "K" was also administered to case xxxiii but, again, without any apparent benefit.

In the present series of cases, as already stated, the changes were confined to the veins, hence the diagnosis of Periphlebitis, but cases have been recorded, diagnosed as Perivasculitis, in which changes have been noted also in the arteries. The clinical and pathological details of one such case have been recorded by Ballantyne and Michaelson (28). The patient was a young adult who had been having fits for the past eight years. The left eye was the seat of recurrent vitreous haemorrhages and was excised on account of secondary glaucoma. Exudates were present along the vessels and the latter showed as white lines in the right eye. This case was interesting in that the earlier stages were present near the periphery and the later stages nearer the disc. This finding is in accordance with those in cases xxxiii and xxxv. The changes were preponderately in the veins although arteries were also affected. The microscopic examination of the retinal vessels showed that there was a well-marked cellular infiltration within the vessel wall which rapidly became annular until, in some cases, the lumen of the vessel was obliterated by the exudate without impairing/

impairing the endothelium. Finally the vessel became converted into a fibrous cord. In other vessels the endothelium proliferated and in others there was a direct invasion of the lumen of the vessel by the exudative cells.

In this particular case, the onset and progress of the disease were extremely rapid and the authors were of the opinion that these features pointed to an infection of some kind as the cause.

Two other cases were described by Ballantyne (55) in which convulsions occurred along with retinal changes. One was diagnosed as Periphlebitis retinae due, possibly, to inherited syphilis but here there were choroidal changes in addition, and the venous changes were confined to perivascular sheathing. The second case showed white spots in the retina and far out to the temporal side a grayish area in the retina with sheathing of the neighbouring veins and one or two dark red areas on the veins which suggested globular dilatations. The latter are very reminiscent of changes seen in the present group of cases.

The apparent new-vessel formation is, in all three cases, profuse. Michaelson & Campbell (29) are of the opinion that many so called "new vessels" are, in reality, pre-existent capillaries which have become dilated. The extreme profuseness of the "new vessels" in these cases, particularly Case xxxiv, must cast a little doubt on this statement. The appearances in the three cases are somewhat different. In Case xxxiii the right eye shows very strikingly the numerous sub-divisions of the affected vein. It is as if the original vein/

vein had broken itself up into many smaller channels in order to try to re-establish the circulation. The lower temporal vein, which was followed in detail for some considerable time, showed first of all some looping (Fig. XL) as if the vein had become too long and had formed these loops in order to gain space for itself. It will be noticed that, even at this early stage, a few collateral vessels had formed. This looping gave way later to numerous small branches all anastamosing with each other (Figs. XLI - XLV). The changes in the left eye were somewhat similar but more advanced and here the looping was of a more complicated nature (Fig. XXXII) but gradually the vein split up in a similar way and showed the same appearance of wanting more room by pushing forward into the vitreous and causing detachment of the retina (Figs. XXXV to XXXIX). This feature occurred in other parts of the retina and there was a tendency for all these to join together. This tendency for retinal detachment was not seen in the right eye but the changes were at a much earlier stage in this eye.

In case xxxiv there is only one part visible, the lower nasal vein, (Fig. L) in which looping occurs similar to that seen in Case xxxiii and the tendency here is for vascular "buds" to form which, however, show little tendency to join together.

Case xxxv shows the same tendency as Case xxxiii to have multiple divisions of the vein which shows at first as brush-like veins (Fig. LII) which gradually join together into a complicated/

complicated network (Figs. LIII and LIV). The two white areas on this network suggest detachment of the vitreous but the main tendency is once more, as in Case xxxiii, for the retina to become detached in folds (Fig. LIV).

It is interesting to note that the visual acuity in Case xxxiii remained good between the haemorrhages in the left eye in spite of the very gross changes in the fundus.

SUMMARY OF CASES xxxiii - xxxv.

Three cases of Periphlebitis Retinae are described. The aetiology in all three is unknown. The changes are confined to the veins and in all three "new vessel" formation is a feature although this manifests itself in all three somewhat differently. No treatment had any effect on any of the three cases.

Case xxxvi.

In the preceding section, three cases are described in which the veins only were affected. Case xxxvi is one in which the pathological changes are confined to the arteries and a diagnosis of Periarteritis has been given to the condition.

W.O. C. aged 26 was first seen on 31.3.44. when he complained that he had been seeing black spots in front of both his eyes for the past two months. He had noticed no signs of inflammation in the eyes. Examination showed that V.A.R. was 4/4.5 and V.A.L., 4/4.5. Each eye had pigmented spots on the anterior surface of the lens and vitreous opacities. Ophthalmoscopic/

Ophthalmoscopic examination showed multiple small white areas on the retinal arteries (Figs. LV and LVI), more marked in the left eye. The retinal veins in both eyes were markedly dilated. An investigation was made to see whether there were any foci of infection elsewhere in the body. Blood Sedimentation Rate was first hour 3, second hour 15. Urine was clear. Blood Pressure was normal. Blood Count was normal. Wassermann Reaction was negative. X-Ray of Chest was also negative. An intradermal Tuberculin Test was negative. He had some nasal trouble in the form of stuffiness of the nose and was found to have mucopurulent discharge from the middle meatus of the left nares and a little less from the superior meatus. He was given treatment with Argyrol Packs and douches and later ephedrine and menthol drops. An X-Ray showed that both antra were probably opaque. The patient was later referred to a German Hospital for examination where a detailed investigation was again carried out including examination of the sputum for Tubercle Bacilli which was negative.

The clinical picture was therefore one of old Irido-Cyclitis with a few posterior synechiae and vitreous opacities along with multiple white placques on the retinal arteries and dilatation of the retinal veins.

The Fundi were examined at intervals and it was found that the white placques tended to become absorbed. Figs. LV and LVI show the appearances three weeks after his first examination. It will be seen that some of the white areas appear to encircle the artery/

artery like a ring whilst others appear to be applied to the side of the artery (Fig. LVI, upper temporal artery). Four months later (Figs. LVII and LVIII) the occurrence of fluffy pink areas on the course of several arteries could be seen. These were fainter in the left eye (Fig. LVIII lower nasal artery) and more obvious in the right (Fig. LVII lower nasal and temporal arteries). At the lower extremity of the lower nasal artery in the right eye a fluffy patch could be seen through which sheathing of the artery could be seen (Fig. LVII). No white plaques could be seen in the right eye but two were still visible in the left eye on the superior temporal artery in the disc area (Fig. LVIII). In both eyes small, bright circular dots could be seen on the course of the arteries at places roughly corresponding to the former position of the white plaques.

Seven weeks later the appearances tended towards recovery (Figs. LIX and LX) but a fresh patch of fluffy pink could be seen in the left eye on the course of the upper temporal artery (Fig. LX). In the right eye several small bright yellow circles could be seen outside the lower branch of the superior temporal artery (Fig. LIX).

A month later the pink fluffy areas had all disappeared (Figs. LXI and LXII) and the yellow circular areas had decreased in the right eye (Fig. LXI)

Five weeks later the yellow circular areas had become less in the right eye (Fig.LXIII) but the left eye showed two very small fresh white circular areas on the course of the lower temporal/

temporal artery (Fig. LXIV) It will be noted also that the white placques on the course of the upper temporal artery in the disc area were still present, although smaller, at this time.

Three weeks later (Figs. LXV and LXVI) the appearances were substantially the same although less in extent.

DISCUSSION.

Periarteritis is a much rarer condition than Periphlebitis and, in this case, appeared to be a complication of Irido-cyclitis. It is well known that Periphlebitis can occur as a spread backwards from Irido-cyclitis but Periarteritis is not such a common complication. As has already been stated, it is possible to have Periphlebitis and Periarteritis associated in the same eye, but, in the present case, the changes, apart from dilatation of the veins are confined to the arteries and no haemorrhages or attempts at collateral circulation can be made out.

A study of the changes which occur brings out the following points.

(1) The first changes seen were white placques, sometimes apparently encircling the artery like a cuff and sometimes apparently applied to the side of the artery.

(2) Small refractile yellow circular areas appeared apparently at the site of a former white placque.

(3) Pink fluffy areas appeared on the course of the arteries, some of which were succeeded by refractile yellow circles as before/

before.

(4) In the later stages in the left eye (Figs.LXIV and LXVI) small circular white fluffy areas appeared applied to the wall of the lower temporal artery.

(5) Although the majority of the white placques disappeared, there were still three small ones applied to the upper temporal artery in the optic disc area (Fig.LXVI) after an interval of 10 months.

The question of aetiology is difficult. In recorded cases, Tuberculosis has been regarded as a possible cause and Muncaster and Allen (30) have described a case which bears a very striking resemblance to the present case. Their case followed a Tuberculin test and it is interesting to note that it was associated with a generalised uveitis which, presumably, included Irido-cyclitis, as in the present case. The case referred to is illustrated in Duke-Elder's Textbook of Opthalmology (31) and it will be seen that the white placques are smaller than those seen in the initial stages of the present case but bear a striking resemblance to the fluffy patches seen at a later stage (Figs. LXIV and LXVI). In addition, a fluffy patch can be seen on the artery at 6 o'clock which bears some resemblance to the pink fluffy patches seen in the present case (Figs. LVII, LVIII, LIX and LX). Tuberculosis was considered as the causal factor of the Irido-cyclitis and the Periarteritis in the present case but all investigations/

investigations in this direction, as already stated, were negative.

The only other possibility was a source in the nasal air sinuses and here some stuffiness of the nose was found accompanied by muco-pus in the superior and middle meatus of the left nares. In addition an X Ray of the skull showed that both antra were probably opaque.

Although it is very difficult to determine the casual factor, it must be recorded that, under treatment of the nose with Argyrol packs and douches and later with ephedrine and menthol drops, the condition of both nose and eyes improved.

The alternative diagnosis, however, of tuberculosis must not be forgotten.

Changes in the fundus oculi in cases of periarteritis nodosa have been described by King (56) and Sampson (57) but in neither case have the changes resembled those seen in the present case of periarteritis retinae. In the former the left eye suffered from Iritis and secondary glaucoma and was excised but no very distinctive changes were found in the eye microscopically. The right eye showed papilloedema probably due to involvement of cerebral blood vessels. In the latter the patient had convulsions and ultimately died. During life both eyes showed transient retinal detachments and normal retinal vessels. In the right eye lateral to and above the macula were several areas of retinal detachment of small extent and, deep to the retina, grayish-white nodules. Post-mortem examination revealed extensive subretinal exudate/

exudate and healed arteritis in the choroidal vessels and Sampson is of the opinion that the exudate and the choroidal nodules were manifestations of active choroidal periarteritis nodosa. Johnson, Harley and Horton (58) have described ocular changes in two cases of arteritis and periarteritis of the temporal vessels. In one case defective vision was due to occlusion of the upper branches of the central retinal artery and vein in the left eye and in the right eye to ischaemic areas along the course of the upper temporal vessels. In the second case a vascular lesion, apparently retro-ocular, affecting the retina occurred on one side.

Case xxxvii.

The last four cases to be described have illustrated abnormalities in the retinal circulation without any apparent choroidal changes. Case xxxvii was one in which the initial lesion appeared to be a patch of acute choroiditis and in which changes occurred in the retinal circulation.

Pte. S. aged 23 was first examined on 17.12.43. when he complained of black spots in front of his right eye for the past three months. His left eye had been defective for as long as he could remember. V.A.R. was 6/6 with glasses and V.A.L. was less than 6/60 with glasses. Ophthalmoscopic examination of the right eye showed fine vitreous opacities and a large patch of acute/

choroiditis about 1½ D.D. in size above the optic disc. Above this was a complicated network of retinal blood vessels. The left fundus showed a large coloboma at the macula with pigment round the periphery. A general idea of the two fundi three months later can be obtained from Figs. LXVII and LXVIII. The left eye remained unchanged throughout. He was given Gutt. Atropine Sulph. for the right eye and admitted to hospital on 20.12.43. His family history showed that one brother and one sister wore glasses for short sight but his father only required reading glasses. The patient himself was slightly short-sighted. His past history had been uneventful apart from the usual childhood illnesses. A general investigation was carried out, including a Wassermann Reaction, which was quite negative apart from a leucocytosis of 19,100.

Fig. LXIX shows the fundus picture on 1.1.44. Three fairly large arteries can be seen emerging from the upper limit of the patch of choroiditis. Cross branches can be seen between them in an attempt to re-establish the circulation and a few branches appear to make contact with the neighbouring veins. Quite a few small haemorrhages are visible and one small arterial twig shows aneurysmal-like swellings on its course.

The visual acuity in the right eye remained normal until 3.1.44. when he complained of mistiness and V.A. was reduced to 4/9. Ophthalmoscopic examination showed that a large haemorrhage had taken place in the retina above the patch of choroiditis and the vitreous was more cloudy again.

The/

The following day the haemorrhage had almost cleared away and the V.A.R. was 4/6. He improved until 8.1.44. when the sight was again suddenly reduced, this time to the ability to count fingers at three feet. Ophthalmoscopic examination at this stage revealed a large haemorrhage into the vitreous. A course of 10 c.c. Calcium Gluconnate daily intravenously was started and given for eleven days. At the same time he had his remaining six teeth extracted. A further vitreous haemorrhage occurred on 10.1.44. and was given Gutt. Dionine 2% and Hot Bathings.

Conditions did not improve and a course of Old Tuberculin was begun on 29.1.44. and on 10.2.44. a course of Karan (Vitamin K.) was begun and continued for five days. On 10.2.44. a course of Eubasin (Sulphathiazol) was begun, but on 27.2.44. after a total dosage of 25 Grammes a rise of temperature took place to be followed by a brilliant generalised morbilliform rash and the drug was stopped. A gradual improvement however took place. Fig.LXX showed the appearances on the day of commencement of the Eubasin. It will be seen that there had been a considerable increase in the size of the haemorrhages and in the complexity of the vessel-pattern.

The vitreous gradually became clearer and on 13.3.44. V.A.R. was 4/24 and on 20.3.44. 4/12 partly. Fig. LXXI shows the fundus appearance on the following day when the lower edge of the inflamed patch could be seen and no continuation of the complicated arteries at the upper border could be seen. On 22.3.44. a fourth, smaller/

smaller haemorrhage occurred into the vitreous and V.A. was again reduced to less than 4/60. Improvement began, however, almost at once and on 1.4.44. V.A.R. was 4/18.

A picture of the whole fundus the day previous is shown in Fig. LXVIIalready referred to. It will be seen that there are two bands of retinal haemorrhage at the temporal and nasal extremities of the field, the larger one being on the nasal side. In addition, two small patches of healed choroiditis can be seen on the course of the upper temporal artery above the macula and a third patch can be seen to the nasal side of the optic disc on the course of a small vein.

The V.A.R. gradually improved and on 10.4.44. it was 4/6 and on 16.5.44. 4/4.5.

Fig. LXXII is an enlarged picture of the right fundus on 3.5.44. showing the white, healed inflammatory patch with the three arteries, two of them larger, stretching upwards. As these arteries are not continued below the lower border of the patch, it is suggested that they form an anastamosis with the choroidal circulation. Another artery further up can be seen to have no lower attachment with visible arteries. Many of the haemorrhages persist and at the top right hand corner of the figure a series of vessels can be seen which have the appearance of a series of small aneurysms.

DISCUSSION.

The occurrence of repeated vitreous haemorrhages in this case, suggested that it might be classified as one of Perivasculitis affecting/

affecting the arteries. There are, however, several points against such a diagnosis. Chief amongst these is the presence of a patch of acute choroiditis and three small patches of healed choroiditis in the right eye and a large coloboma of the macula of the left eye. This picture suggested a diagnosis of Congenital Syphilis but serological tests were negative. This raises the question of the reaction of the retinal blood vessels to inflammation in the choroid. It has already been seen in Part I that injury to the choroid may precipitate spasm in the retinal arteries and veins but here there is a different type of reaction, in the shape of what appear to be new blood vessels which give rise to haemorrhages in the retina and also, on four occasions, into the vitreous. As is well known, the deeper layers of the retina are nourished by the choroid and any inflammatory reaction in the latter must inevitably give rise to considerable disturbances in the overlying retina. One interesting point about this case is that no sector defect could be made out in the right visual field (Chart XXII) but only a scotoma below the optic disc (Chart XXIII).

The question therefore arises as to whether the visible vessels in this case could be forming an anastamosis between the retinal circulation and that of the choroid. Such anastamoses have been recorded but most of these are in congenital abnormalities. Mann and Ross (32) report such a case seen in later life and postulate a patch of choroiditis occurring in the fourth month which would involve the chorio-capillaries and probably also the external choroidal layer but not necessarily the intermediate layer which/

which is just beginning to develop at the fourth month. The inner layers of the retina, they say, might escape since they would be separated from the focus by the potential cavity of the primary optic vesicle. Bruch's Membrane, however, would be destroyed and the normal barrier between the mesoderm and the neural ectoderm would thus be broken down. Hence new arteries developing from the intermediate layer of the choroid could invade the overlying retina from its deep surface.

Lawson (33) and Beaumont (34) also record cases in the macular area which they regard as congenital in origin but Gradle (35) and Feingold (36) record cases which appear to have arisen later in life. Gradle's case occurred in a girl of 19 years who had had defective vision in the right eye since the age of 8 years when she had measles. Feingold reported 3 cases. The first was in a 14 year old boy who had a perforating injury. The second was in a 46 year old woman with a patch of healed choroiditis in the right eye. Her son had previously been treated for disseminated choroiditis of syphilitic origin but no details were given as to the serological reactions of the patient. The third was in an 18 year old girl who gave a history of seeing black spots in front of her eyes for three months. A patch of old choroido-retinitis was present in each eye with choroido retinal anastamosis. The second and third cases are open to the objection that they may have been present for some considerable time but it seems fairly clear that the anastomosis in the first case took place after the injury.

Bickerton (37) also reported a case of senile macular degen-

eration/

following haemorrhage. This case had been seen two years previously when there was only a haemorrhage visible at the macula. The choroido retinal anastamosis had apparently developed during the two years.

It cannot be doubted that the present case had an infection as the patch in the right eye had fuzzy edges, a leucocytosis was present and the case ultimately responded to teeth extraction and Sulphathiazol therapy. The further fact that haemorrhages kept occurring into the retina and vitreous supports the evidence of activity..

The coloboma at the left macula, however, was quite inactive and remained stationary throughout. It is interesting to note, however, that no choroido-retinal anastamosis occurred in the left eye although the focus was presumably congenital in origin and situated in a common site for such a lesion.

The question therefore arises as to whether there is actually a choroido-retinal anastamosis in this case. A study of Fig. LXXII will show that two fairly large branches and one smaller branch are visible at the upper extremity of the quiescent patch. Only one smaller branch can be seen at the lower extremity of the patch. The arteries must be receiving blood from somewhere and it is suggested that they form an anastamosis with the choroidal circulation although this cannot be seen. The repeated haemorrhages into the retina and vitreous are probably the result of blockage of the arteries by small infected emboli from the patch of choroiditis/

choroiditis or to thrombosis in the arterial wall from damage caused by infection from the same source.

SUMMARY OF CASE xxxvii.

A case of acute choroiditis is described in which a complicated system of retinal vessels made their appearance. Haemorrhages occurred from this system into the retina and the vitreous. The condition became quiescent after extraction of teeth and a course of Sulphathiazol. A large macular coloboma was present in the other eye which was thought to be congenital. In spite of the rarity of choroido-retinal anastamosis, it was thought that the retinal arteries established a communication with those of the choroid.

Case xxxviii.

This case is one of Heterochromic Cyclitis in which changes were present in the unaffected eye which lend support to the view that Heterochromic Cyclitis is the result of vaso-motor disturbance.

Tpr. C. aged 26 was first examined on 16.6.44. His history was that in 1938, whilst serving in the Army, he began to have defective vision for close work in the right eye. He was treated spasmodically but was never admitted to hospital. His father committed suicide but his mother was alive and well but wore glasses constantly. One sister, his only one, was alive and well. In childhood he had had jaundice, whooping-cough and measles at the age of 5. His tonsils had been removed in childhood but he had had frequent sore throats since then.

On/

On examination of the eyes the iris of the right eye was found to be pale blue-gray whereas that of the left eye was greenish-blue (Fig. LXXIII). V.A.R. was 3/24 under Atropine and V.A.L. was 4/4.5 undilated. Ophthalmoscopic examination showed that the right vitreous was somewhat cloudy and several spots of K.P. were present. The right fundus had a normal appearance (Fig. LXXIV). The left vitreous was quite clear and no K.P. were present but the left fundus showed grossly dilated tortuous retinal veins. (Fig. LXX V). The a/v. Ratio was 1/2.

He was admitted to hospital where a general investigation was carried out. The Kahn test was negative. The nasal sinuses were clear and the Blood Sedimentation rate was 1st hour 2: 2nd hour 8. Some focal sepsis, however, was found in the teeth and the left upper incisors were both removed, an apical abscess being present in one.

On 13.7.44. V.A.R. = 4/24 (1 letter) with + 2.5 Sphere = 4/9 partly. On 4.8.44. V.A.R. = 4/18 under Atropine and the latter was stopped. The visual acuity gradually improved. On 8.8.44. V.A.R. = 4/6. On 15.8.44. V.A.R. = 4/4.5 partly and on 1.9.44. it was 4/6. partly. On this latter date there was still some cloudiness of the vitreous and multiple K.P. were still present.

DISCUSSION.

Any case of chronic Cyclitis gives rise to a problem of aetiology and it is a common experience in cases of Heterochromic Cyclitis to find no septic focus and this, combined with the absence/

absence of inflammatory signs in the affected eye, has led many observers to suggest that the condition is not an inflammatory one but is in the nature of a vaso-motor phenomenon.

The subject was discussed fully in Holland in 1929 (38) . Bistis was of the opinion that the heterochromia whether it be congenital or acquired, if it does not arise from an inflammatory process is the result of paralysis of the sympathetic in the neck. The K.P. and the cataract (which accompanies this condition so frequently) are also the result of sympathetic paralysis and heterochromia can be produced experimentally showing actual atrophy of the iris pigment.

Poos, however, stated that this was a deception and the heterochromia was due to contraction of the pupil and stretching of the iris. Loewenstein and Bruckner supported Bistis.

Bistis further (39) described a case of a 38 year old man with heterochromia of the right iris which had been present for two years without apparent cause. K.P. were present and he also had Horner's syndrome in the affected eye. He described also a case of acquired heterochromia of the left eye. One year previously this eye had been bright yellowish and earlier it was brown like the other eye. Horner's syndrome was also present. At the anterior edge of the left sterno-mastoid muscle was a broad scar which extended up to the mastoid process. This was the result of an operation for removal of the sympathetic ganglion.

It will be seen, therefore, that there is considerable support for the view that heterochromic cyclitis is the result of sympathetic nerve paralysis. In the present case the bleaching of the iris is in the right eye with K.P. and cloudy vitreous (but no cataract) whilst the retinal vessels are of normal calibre. In the left eye, however, there is no bleaching of the iris or cloudiness of the vitreous but the retinal veins are grossly dilated and tortuous. No other vaso-motor effects could be made out in any other part of the body, but it seems reasonable to postulate that the manifestations of sympathetic nerve upset in the right eye are bleaching of the iris, K.P. and vitreous opacities whilst in the left eye the only manifestation is dilatation and tortuosity of the retinal veins.

SUMMARY OF CASE xxxviii.

A case of Heterochromic Cyclitis is described in which no septic focus beyond an apical abscess in a tooth could be made out. The retinal blood vessels in the affected eye were normal whilst the veins in the other eye were grossly dilated and tortuous. It is suggested that the two conditions are the individual responses to an upset in the sympathetic nerve system.

SUMMARY OF PARTS I and II.

In Part I an attempt has been made to show the effects of injuries to the choroid on the retinal blood vessels. These effects are probably brought about by the intervention of axon reflexes.

With one exception (Case xxiv) all these injuries occurred during/

during the War of 1939-1945 and 24 out of the 32 cases described were injured by high-velocity projectiles. In very few cases was there any external sign of injury to the eye. 28 were British or Allied Prisoners of War.

These changes occurred in an age-group in which one would not expect to find vascular changes.

In Part II six cases of abnormalities in the retinal circulation occurring in British Prisoners of War are described and discussed, three of Periphlebitis, one of Periarteritis, one of Acute Choroiditis and one of Heterochromic Cyclitis.

GENERAL DISCUSSION.

In both parts of this Thesis vascular abnormalities are discussed in an age group in which vascular changes are, on the whole unusual. In elderly people it is usual to find some degree of arterio-sclerotic change varying from variations in the arterial calibre to gross changes in the vessel wall accompanied by exudates and haemorrhages but such changes are rare in the age group concerned in the present series of cases. There are, however, certain features of these cases which bear some resemblance to early arterio-sclerotic changes.

Ballantyne (40) has described arterio-sclerotic changes in detail. He notes the occurrence of cork-screw veins in the periphery of the fundus, particularly in cases of Thrombosis of the Central Retinal Vein, and also in the macular area. In two cases in the present series (Cases xi Fig. IX and xiii Fig. XI) such a cork-screw like/

like appearance was observed in the macular area. The macula itself in the first case had become replaced by a dusky area and there was a large choroidal rupture in the upper nasal area of the fundus. In the second a macular hole had occurred. In another case (Of Heterchromic Cyclitis) there was dilation and tortuosity of all the retinal veins (Case xxxviii Fig. LXXV) This change, however, was thought to be in a different category as a vaso-motor influence was probably coming into play.

Ballantyne states also that parallel and pipe-stem sheathing are without doubt evidence of an advanced state of arterio-sclerosis. Three of the traumatic cases in Part I of this Thesis show such an appearance (Cases viii Fig V., xv. Fig.XIV and xxii Fig. XXIII and one case in Part II (Case xxxvi, Fig. LVII & LIX.) Further changes of a degenerative character may take place with the formation of glistening crystalline patches on the arteries. Such a condition can be seen in the case of periarteritis (Case xxxvi Figs. LVII to LXVI) along with a certain amount of pipe stem sheathing as mentioned above. A general examination of this case, however, revealed no rise in blood pressure or any other sign of arterio-sclerosis. The periarteritis was thought to be toxic in origin.

Ballantyne in the same article described the occurrence of aneurysmal terminations in the terminal twigs of unoccluded veins in cases of thrombosis of the central retinal vein. Such an appearance was not seen in any of the traumatic cases but in the cases of acute choroiditis in Part II (Case xxxvii. Fig.LXXII) and/

and periphlebitis retinae (case xxxiv, Fig. Ll) some of the small collateral vessels in the periphery had a definite beaded appearance which, it is suggested, was due to aneurysmal dilatations.

Changes somewhat similar to those encountered in arterio-sclerosis are seen in the fundi of diabetic subjects but Ballantyne (41 & 42) is of the opinion that arterio-sclerotic and diabetic retinopathies are quite distinct entities. He points out that, in a series of cases of diabetic retinopathy investigated by him, 50% had no elevation of blood pressure and that the changes tended to occur in the middle-aged rather than the elderly. One case, in fact, occurred in a boy of 19 years of age. He stresses also the importance of microaneurysms which may well be confused with punctate haemorrhages. Careful examination of the retina in bulk has, however, established that many of the so-called punctate haemorrhages in diabetic retinopathy are, in fact, microaneurysms connected to the blood vessels.

The earliest changes are in the veins in contradistinction to the changes in arterio-sclerotic retinopathy which are more marked and occur first in the arteries and Ballantyne describes in detail some changes observed by him in the veins in diabetics which bear a striking resemblance to those seen in periphlebitis retinae and described in Part II of this series (Cases xxxiii to xxxv). The veins become tortuous and beaded and complicated loops are formed. Venous stasis may, he states, be an important factor in the development/

development of diabetic retinopathy. Microscopically, he has found fatty droplets in the endothelium of the capillaries which may give way in the event of a block on the venous side.

The important differences between arterio-sclerotic and diabetic retinopathies lead him to state that, if a chemical agent is responsible for the vascular changes in the two conditions it must have an affinity for precapillaries and arterioles in the case of arterio-sclerosis and for capillaries and venules in the case of diabetes. In the present thesis, apart from the three cases of periphlebitis, there are few changes of a nature similar to those described by Ballantyne as characteristic of diabetic retinopathy. It is interesting to note, however, that the case of periarteritis (Case xxxvi, Figs. LV to LXVI) showed grossly dilated veins in both fundi although the primary changes appeared to be in the arteries. In this case the periarteritis was assumed to be toxic in origin. The case of heterochromic cyclitis (Case xxxviii, Fig . LXXV) showed grossly dilated veins in the unaffected eye, but, as already stated, this was thought to be a vaso-motor phenomenon. Amongst the traumatic cases any changes present in the veins were in the direction of constriction but two cases showed small punctate "haemorrhages" which might have been microaneurysms (Case xx, Figs. XX & XXI and Case xxi, Fig. XXII). In the former case, however, the "haemorrhages" cleared up in the course of six weeks, a point in favour of their haemorrhagic nature.

The most striking similarity in the changes lies in the appearances/

appearances of the veins in the three cases of periphlebitis retinae. A study of Figs. 27, 28 & 29 of Ballantyne's & Loewenstein's paper (42) shows the looping and tortuosity of the veins and also the splitting up of the veins into brush-like formations in places. General opinion points to periphlebitis retinae being a response to toxic absorption and Gifford (43), investigating a small number of cases, showed that Tuberculosis was a factor in seven out of twelve cases. Another factor, however, was found to be peripheral angiospasm and this will be discussed shortly.

It is interesting in view of the opinion of Michaelson & Campbell (29) concerning the so-called "new vessels" that Ballantyne has examined many of these "new vessels" microscopically and is of the opinion that many of the pre-retinal ones must be newly formed vessels.

The subject of angiospasm was for a long time a vexed one but recently it has been established as a clinical entity. As long ago as 1903 Sir William Gowers (44) suggested that the generalised arterial constriction in Bright's Disease was due to spasm but this view was not supported and Taylor (45) speaking to the Ophthalmological Society of the United Kingdom in 1913 on vascular and other retinal changes associated with general disease did not mention angiospasm. He did, however, recall four cases of obstruction of the central retinal artery of the retina in young people. Two of these were found to have no apparent source for an embolus and were aged 10 years and 19 years old

respectively/

respectively. Of the two others one had a history of rheumatic fever and chorea and the other had a heart murmur and it could be claimed that they had a possible source for an embolus. Taylor, however, did not consider spasm of the artery as a possibility but attributed the blockage to thrombosis. In the light of more recent work it might be suggested that angiospasm was the underlying cause.

At the same meeting, however, Louis Werner (46) spoke of repeated attacks of sudden temporary failure of vision and of peristaltic waves having been seen, passing along the arteries. One theory of such a wave was that, as the blood pressure falls, the vis-a-tergo is no longer capable of forcing the blood through the narrow diseased lumen. The second theory was angiospasm. The author, however, did not consider that there was sufficient evidence to support such a theory.

In the discussion following this paper, Priestley Smith said he thought that some cases could be attributed to angiospasm and Richardson Cross was of the opinion that some cases of so-called retrobulbar neuritis were due to spasm of the small arteries supplying the optic nerve behind the eye. Coats agreed that spasm occurred but Werner, in replying, said that angio-spasm was only an inference whereas arterio-sclerosis was an established fact and he thought that it was difficult to see how spasm could last sufficiently long to produce a permanent obstruction.

Later, however, Mylius in 1928 (47) described tetanic and spastic/

spastic contractions in women suffering from eclampsia which ceased on the termination of the pregnancy.

In 1930 Friedenwald (48) quoted Lange and Lange and Mylius as having regarded the constrictions of retinal arteries in hypertension as angiospastic in origin. Raehlmann, however, he said, differentiated localised constrictions due to endarteritis and generalised constrictions due to angiospasm thereby recalling the views of Gowers (44).

In the discussion which followed this paper Foster Moore observed that it seemed unlikely that a highly diseased artery should be capable of severe spasm.

The next year, however, Davenport (49) reported an undoubted case of spasm of the central retinal artery to the Ophthalmological Society of the United Kingdom. The patient was aged 57 years with pyrexia and cramping pains in the legs. The conditions were thought to be all toxic in origin including the arterial spasms, as, although his blood pressure was raised, there was no suggestion of generalised arterio-sclerosis or retinal endarteritis. The patient's fundi were examined repeatedly during attacks of temporary blindness and the discs were seen to be pale and the arteries very narrow. No cherry-red spot, however, was visible but the conditions always relieved themselves, the arteries regaining their normal diameter and the discs becoming pink again.

In 1940 Grimsdale (50) reported a personal experience. He had a sudden onset of a central scotoma in one eye and a colleague found a small contracted arterial twig which led to the macula. Acetyl Choline and Doryl were given and the scotoma disappeared

for the most part and the arterial twig refilled. In the same year Bruhn (51) recorded cases of traumatic retinal angiopathy occurring in war injuries. These, he described as usually a distance effect of skull injuries or thoracic compression and instanced the case of an airman who fell from his aeroplane and complained of a central scotoma in the right eye. The ophthalmoscope revealed oedema of the macular area and other foci around the disc. As a cause he suggested compression of the thorax causing sudden congestion in the peripheral parts of the body.

In 1941 a similar condition to the above but not of traumatic origin was described by Loewenstein (52). This he referred to as Retinopathia Centralis Angiospastica and Serosa allergica and consists of oedema of the macular area. He wrote of the analogy between this condition and intermittent claudication, migraine, amaurosis fugax and bronchial asthma. The affection, he stated, was transient and could only be due to angiospasm, possibly occurring reflexly from the teeth, but he admits that angiospasm may last for some months as evidenced by the persistence of angiospasm following a retrobulbar injection of adrenaline. This is an interesting statement in view of the contention that the vascular constriction in the present series is due to angiospasm. Another interesting statement by Loewenstein is that he is of the opinion that this type of macular oedema may be allergic in origin and that the antigen plus the antibody produce a histamine-like substance which works in an angiospastic manner. These antibodies/

antibodies may arise on exposure to certain types of infection by organisms of low virulence diffused into the blood from a distant focus such as an active tubercle. It has already been pointed out, however, in the discussion on Part I of this thesis that histamine tends to produce vaso-dilatation rather than vaso-constriction. Loewenstein, further, quotes Redslob as having found a detachment of the retina without a retinal defect but with congestion of the choroidal vessels and also Redslob and Nodman as having produced an experimental detachment of the retina in a dog by ligaturing the vortex veins, thus producing very choked choroidal vessels. He stresses the importance of the choroidal vessels in the nourishment of the retina and states that the condition of angiospastic retinopathy can be ascribed to choroidal reaction in the first place including the effect of spastic contraction of the retinal vessels in the foveal area. This is an interesting observation in view of the occurrence of retinal arterial constriction in cases of choroidal rupture in the present series.

In 1944 Gifford (53) also wrote of this condition of Central Angiospastic Retinopathy and carried out an investigation into the vaso-motor condition of the rest of the body. Six out of eight cases (all aged between 17 and 43) showed evidence of angiospasm in the peripheral parts of the body which had apparently very often some connection with excessive smoking. At the same time Gifford was investigating cases of periphlebitis retinae and he found that seven out of twelve cases showed evidence of tuberculosis/

tuberculosis and that five out of seven cases referred for tests showed evidence of peripheral angio-spasm. Two showed no evidence of angiospasm and one case of periphlebitis retinae had a septic focus removed and recovered. Two cases showed evidence of both tuberculosis and peripheral angiospasm. Gifford concludes, therefore, that the situation with regard to periphlebitis retinae is not so simple as in central angiospastic retinopathy and that several factors may be responsible notably tuberculosis. This, however, would fit in with Loewenstein's suggestion (52) that the retinal condition may be an allergic response to organisms originating from a distant focus.

In 1946 Pines (54) went so far as to describe physiological angiospasm in middle age. He asserted that careful tracing out of arteries away from the optic disc revealed very often a sudden narrowing of the arterial lumen to 1/10 of its original diameter without any alteration in the colour and light reflex of the vessel. He found variations in the oscillometer readings in these patients and concluded that they were due to the vasomotor play in the body.

An attempt therefore has been made to show that angiospasm in the retina is a real entity occurring in a large variety of conditions. It will be noted that it is not confined to the middle age and elderly groups but can occur in the younger age-groups. It is with such an age-group that the present thesis is concerned -- that from which the fighting services are drawn in time/

time of war. It has been shown, however, that angiospasm is commoner in the older age-groups in such conditions as arterio-sclerosis. This condition may be ascribed to the accumulation of various stresses and strains and toxic absorption and it is possible to picture a gun-shot wound of the orbit as providing a very much accelerated disturbance to the eye. As we have seen, such changes as parallel sheathing and even pipe-stem sheathing and cork-screw vessels at the macula may follow upon such an injury and these are lesions usually ascribed to arterio-sclerosis.

It must not, therefore, be assumed that the vascular system of a young eye is immune from pathological changes on account simply of its youth but may undergo somewhat modified arterio-sclerotic changes as the result of such catastrophes as gun-shot wounds or toxic absorption.

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UNIVERSITY OF GLASGOW.

Thesis Submitted for the Degree of M.D.

SOME VASCULAR ABNORMALITIES IN THE EYES OF YOUNG MEN OF SERVING AGE

With Illustrations.

By

A.M. Wright Thomson M.B. Ch.B.

Vol. II - Diagrams & Illustrations.

1947.

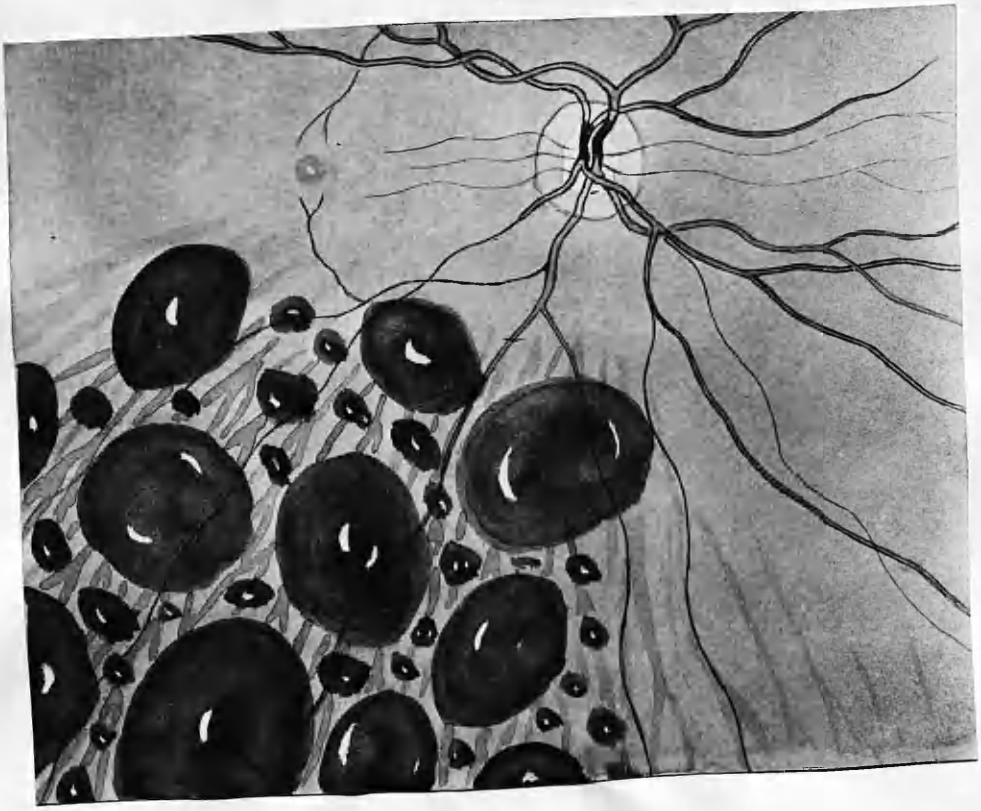

Fig. I.

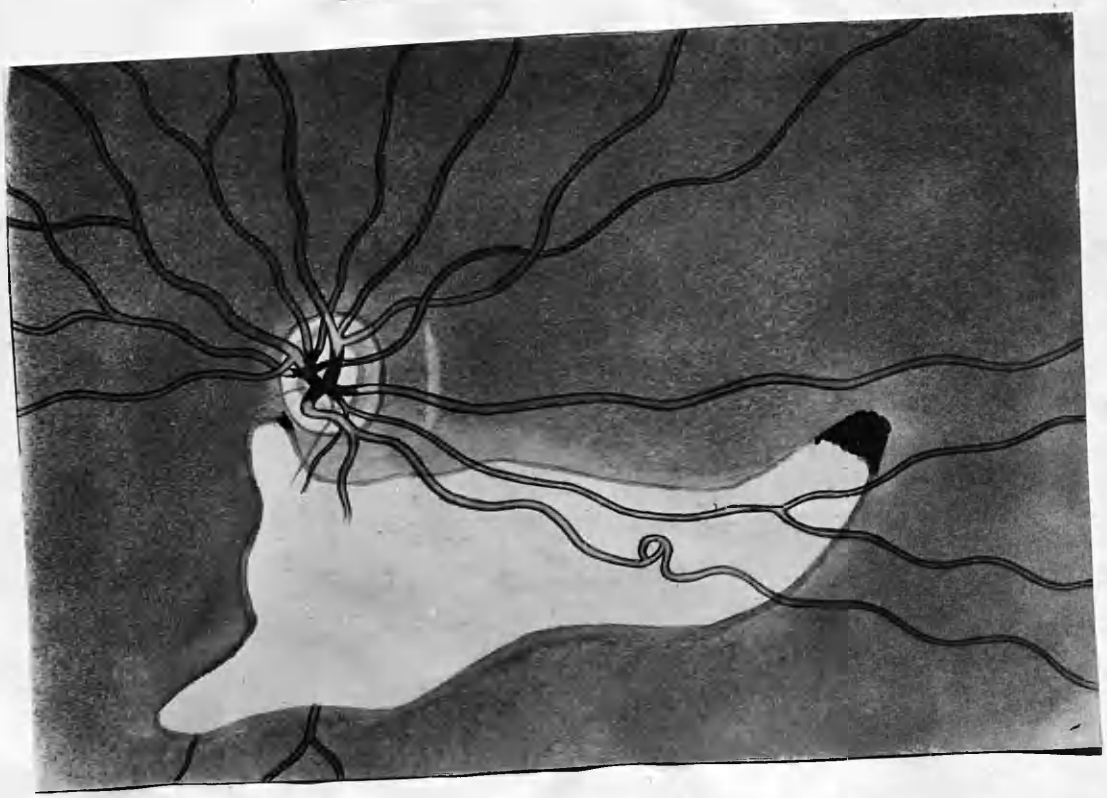

Fig. II.

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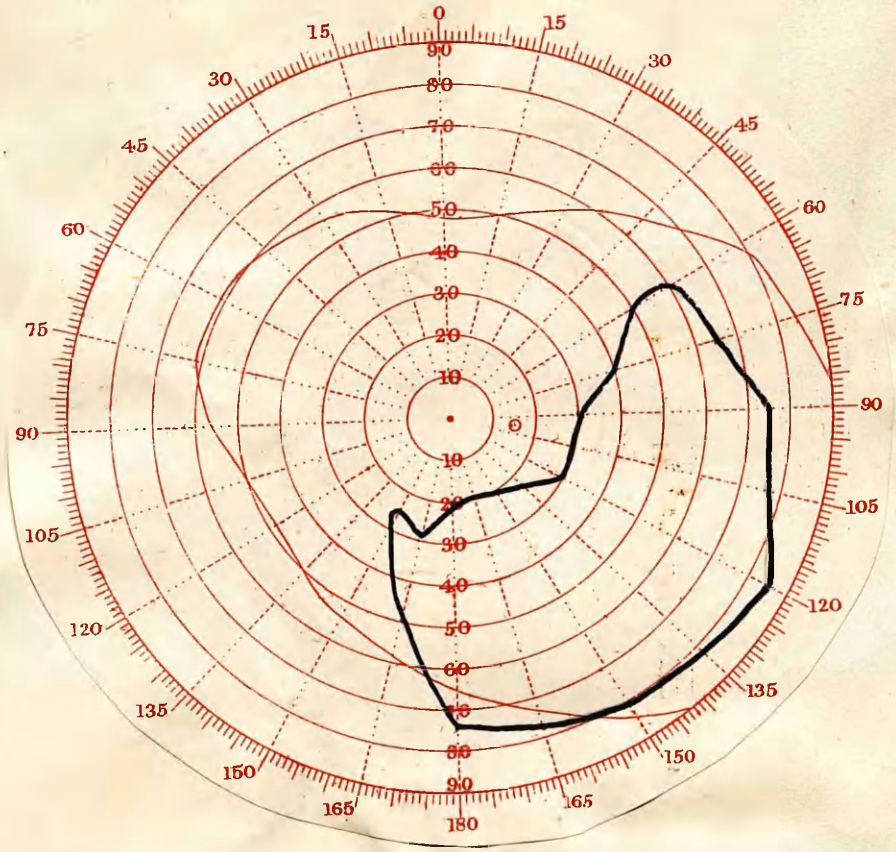

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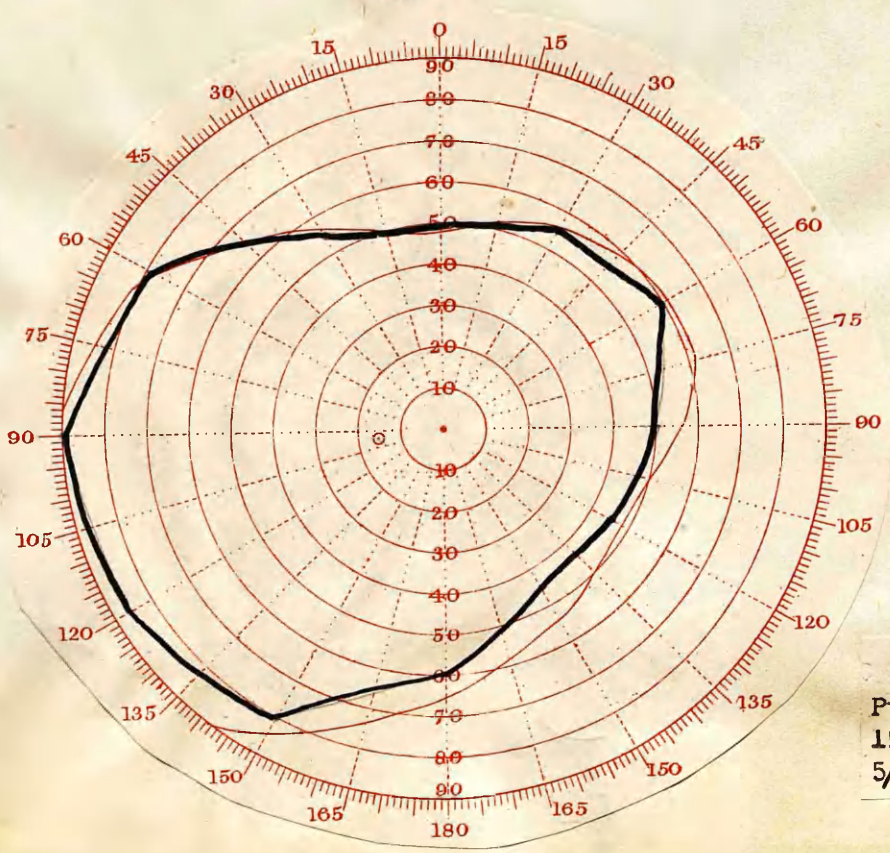

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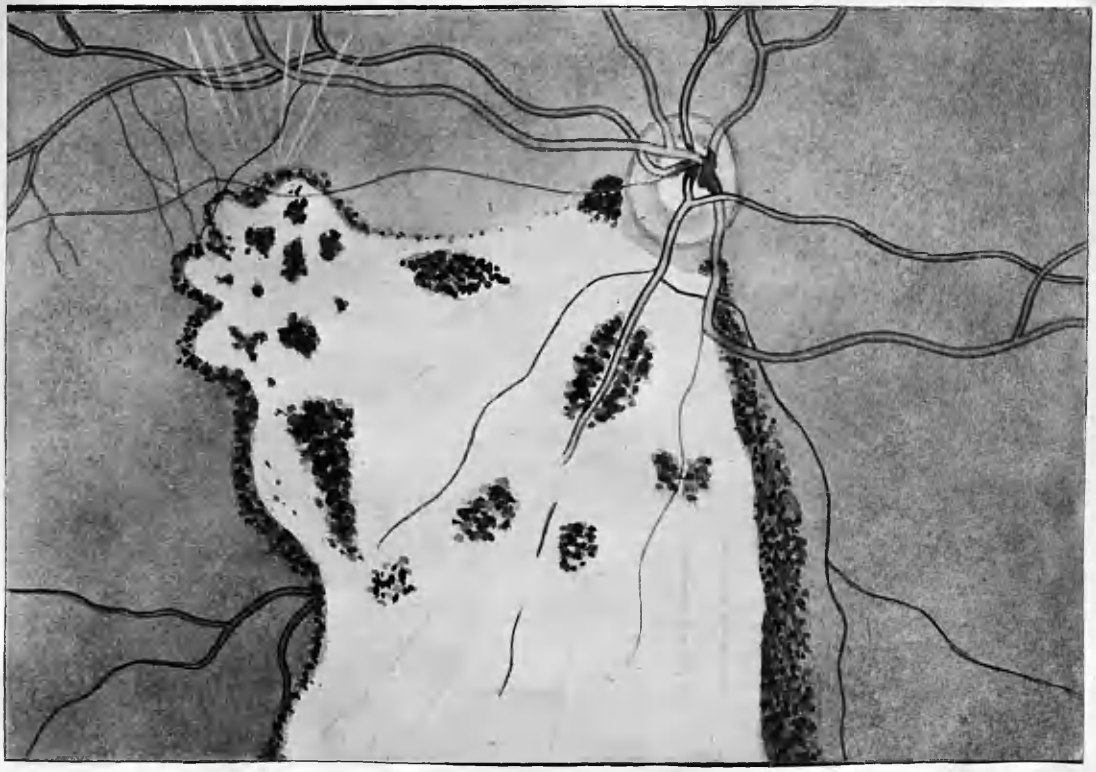


Fig. III.

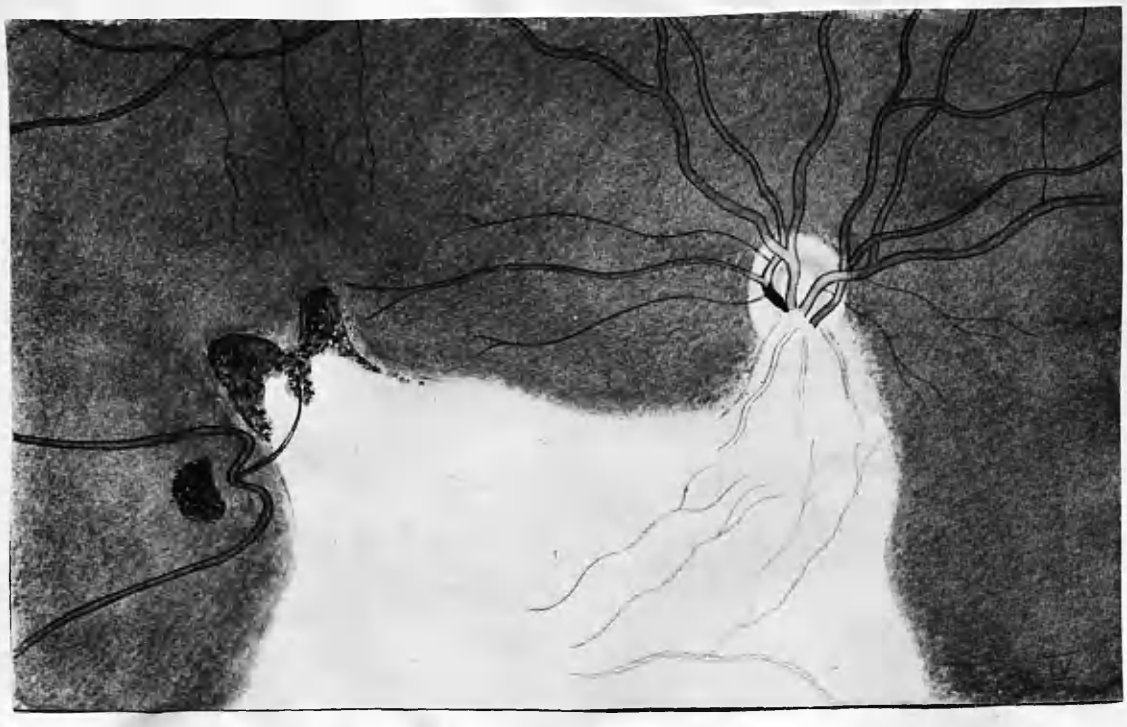
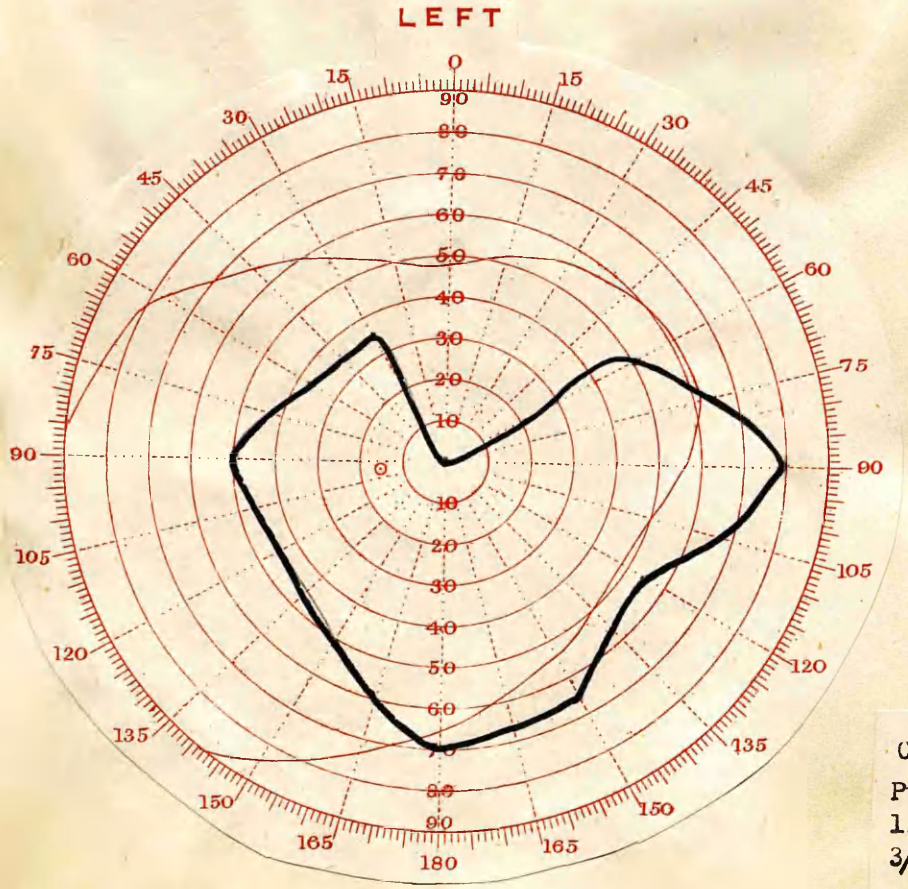


Fig. IV.

Fig. V.



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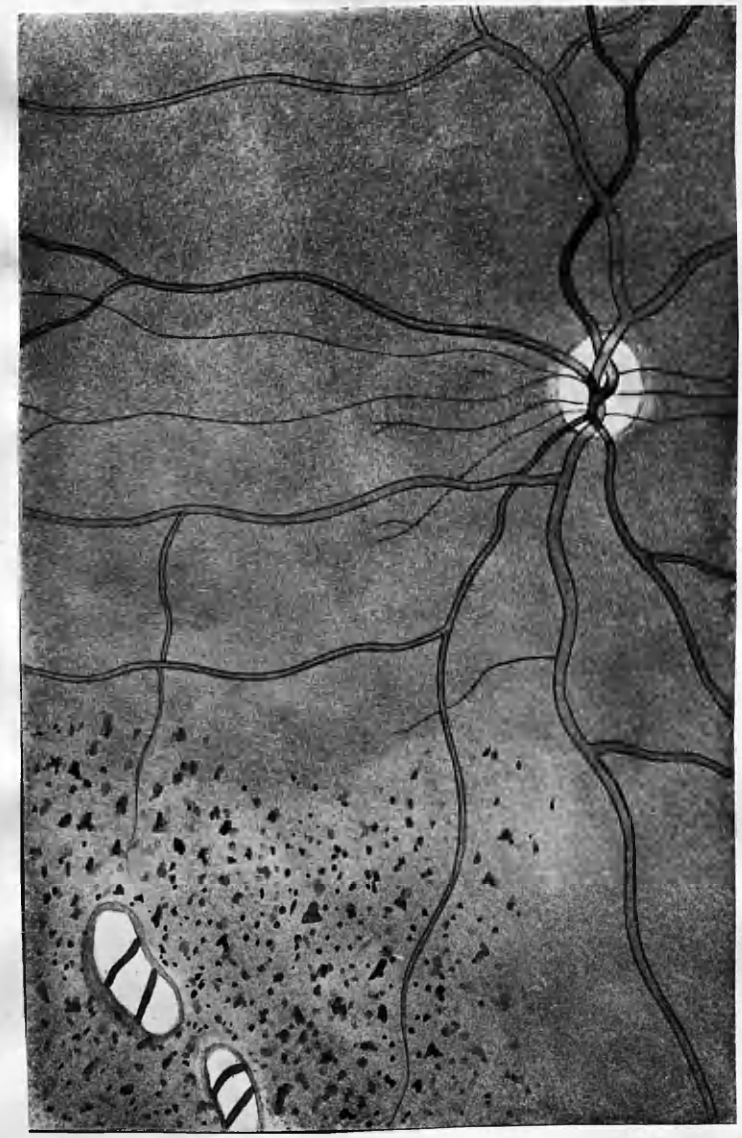

Fig. VI.

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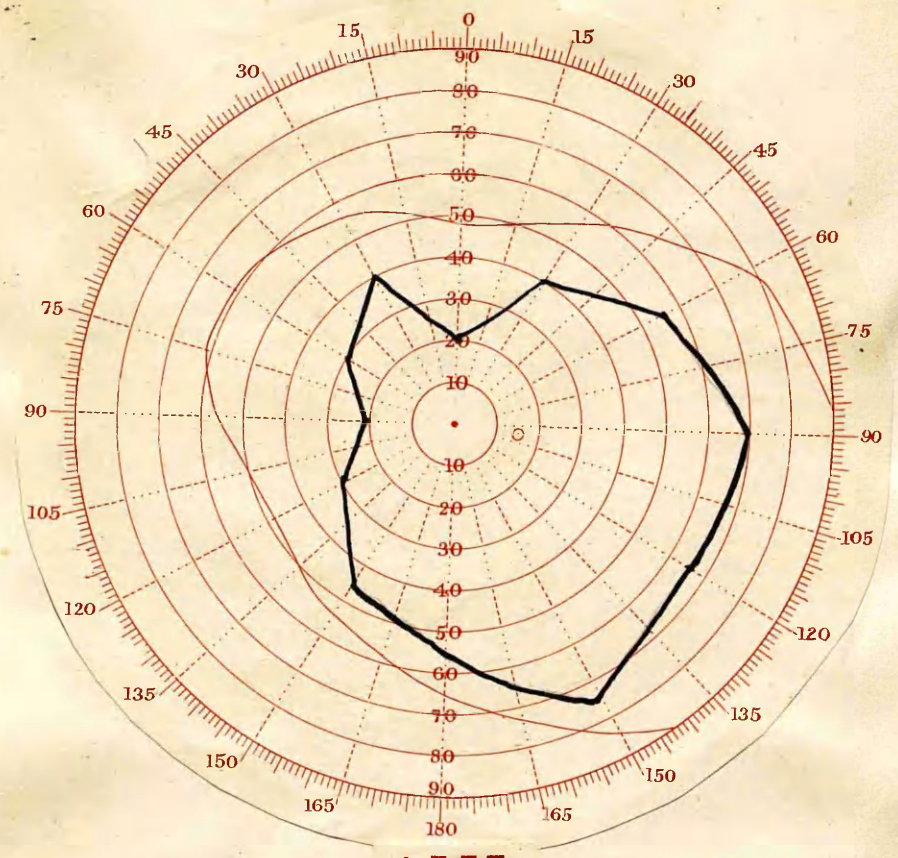

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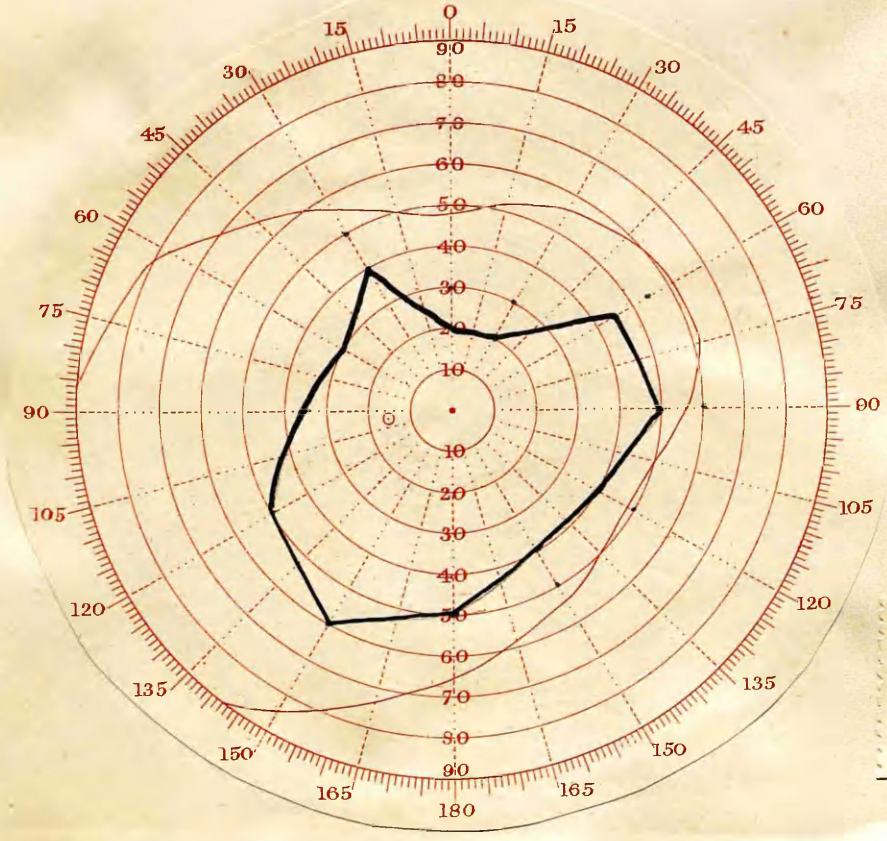

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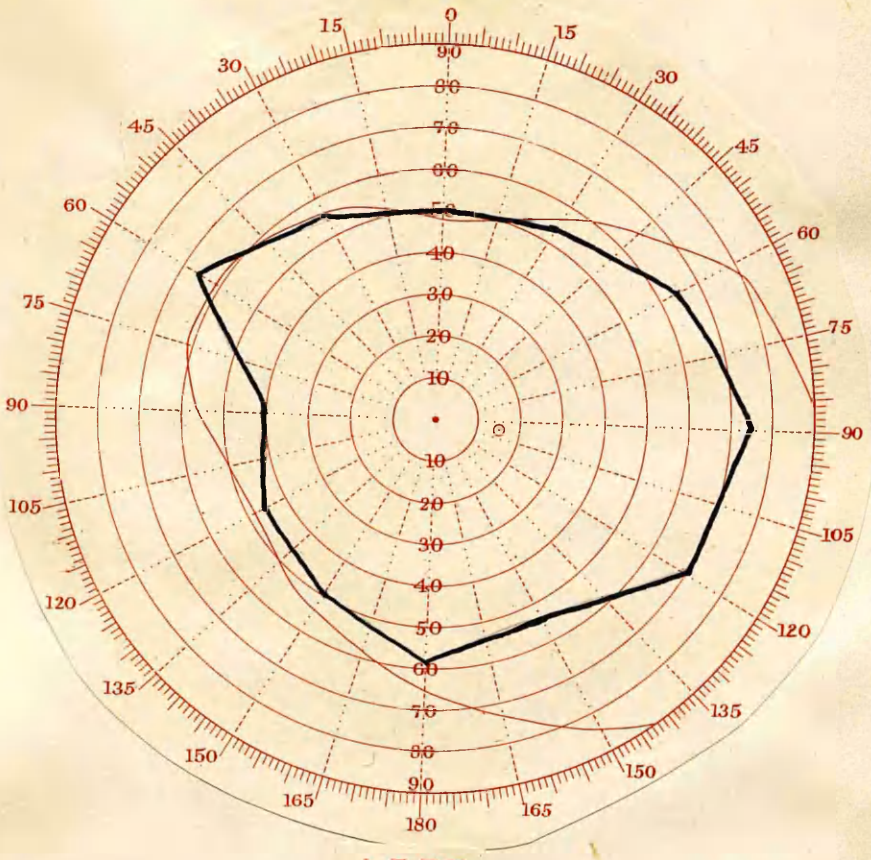
3/330 White.

RIGHT.



LEFT.

CHART IV.
Pte. Moody.
6.4.44.
3/330 White.

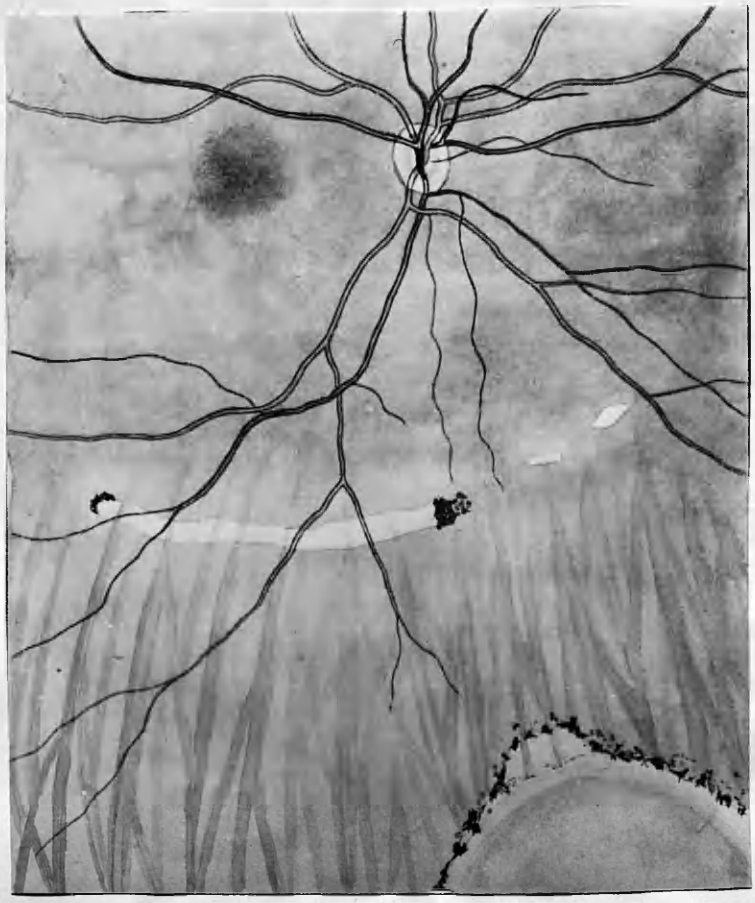
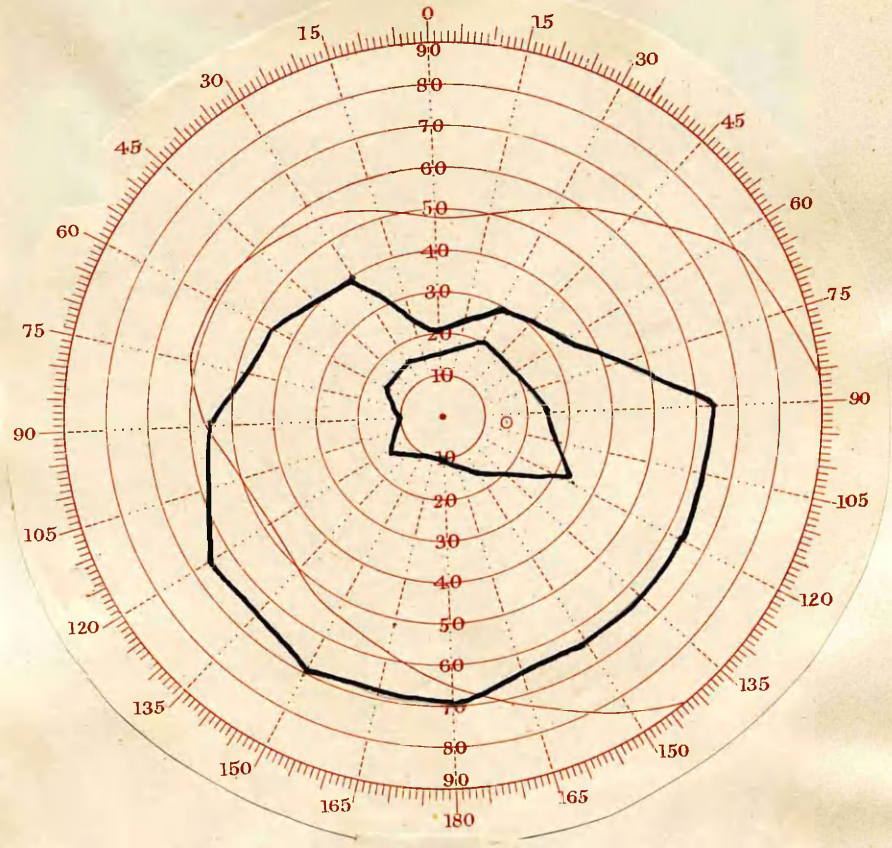


Fig. VII.

RIGHT.



LEFT.

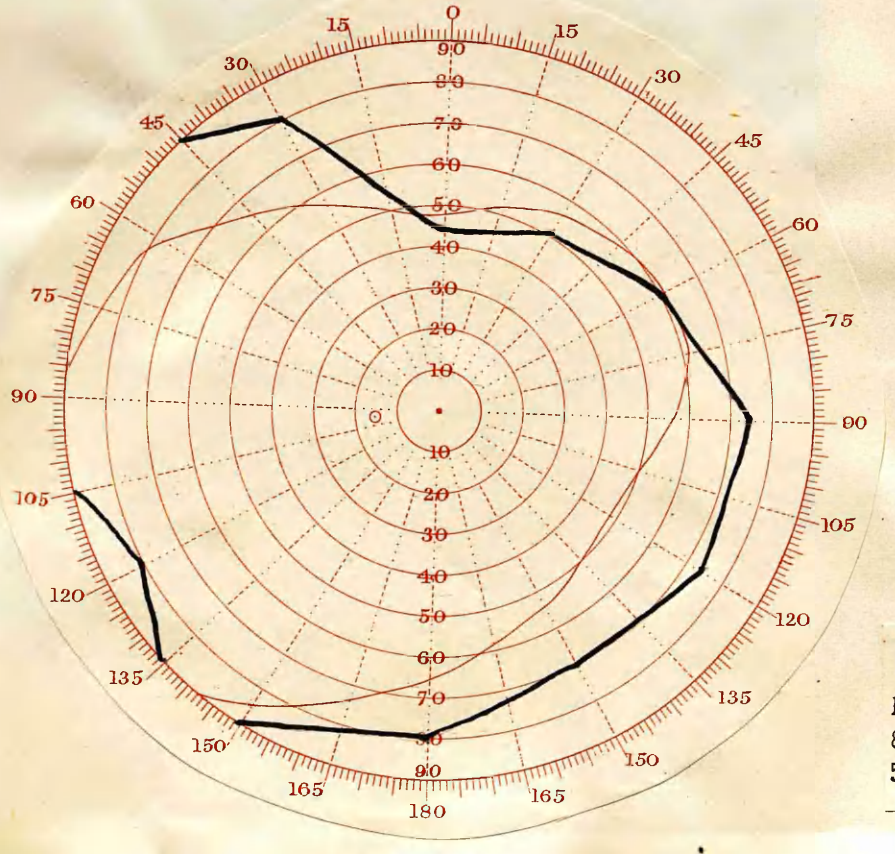


CHART V.
Pte. Richards.
8.7.44.
5/330 White.

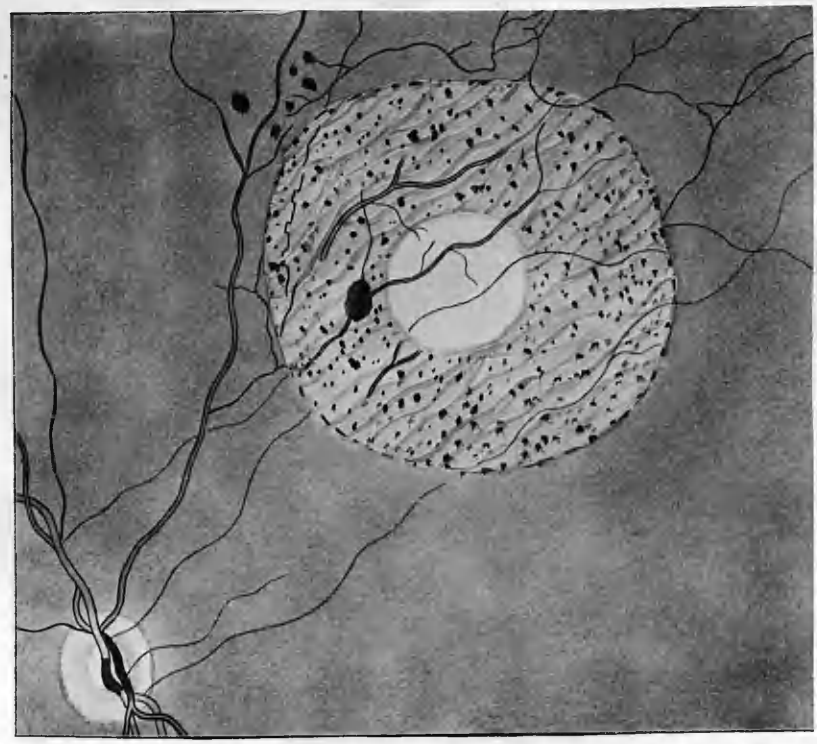


Fig. VIII.

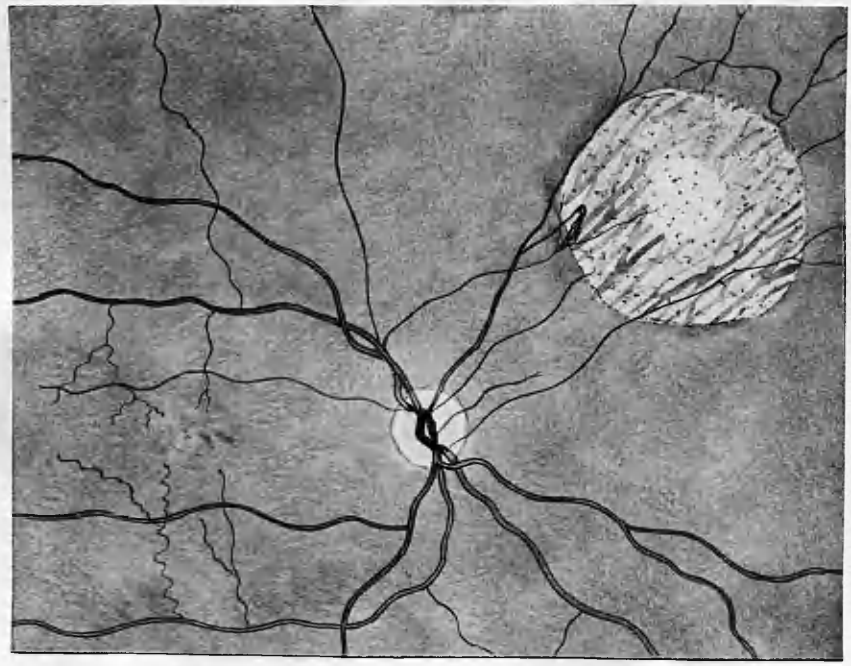
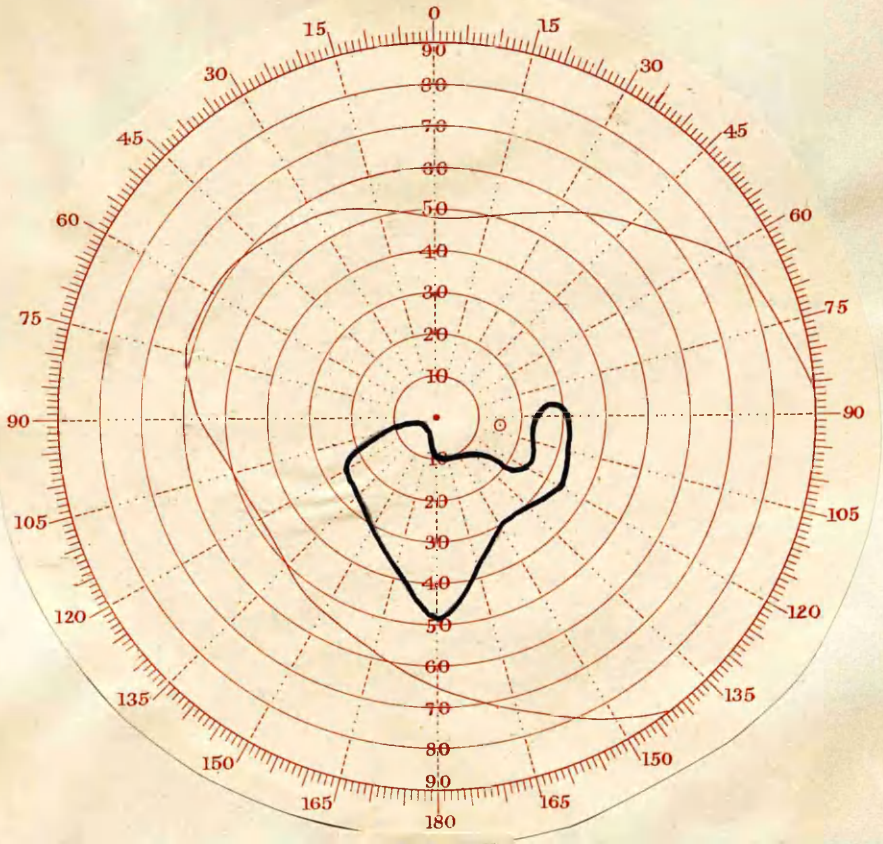


Fig. IX.

RIGHT.



LEFT.

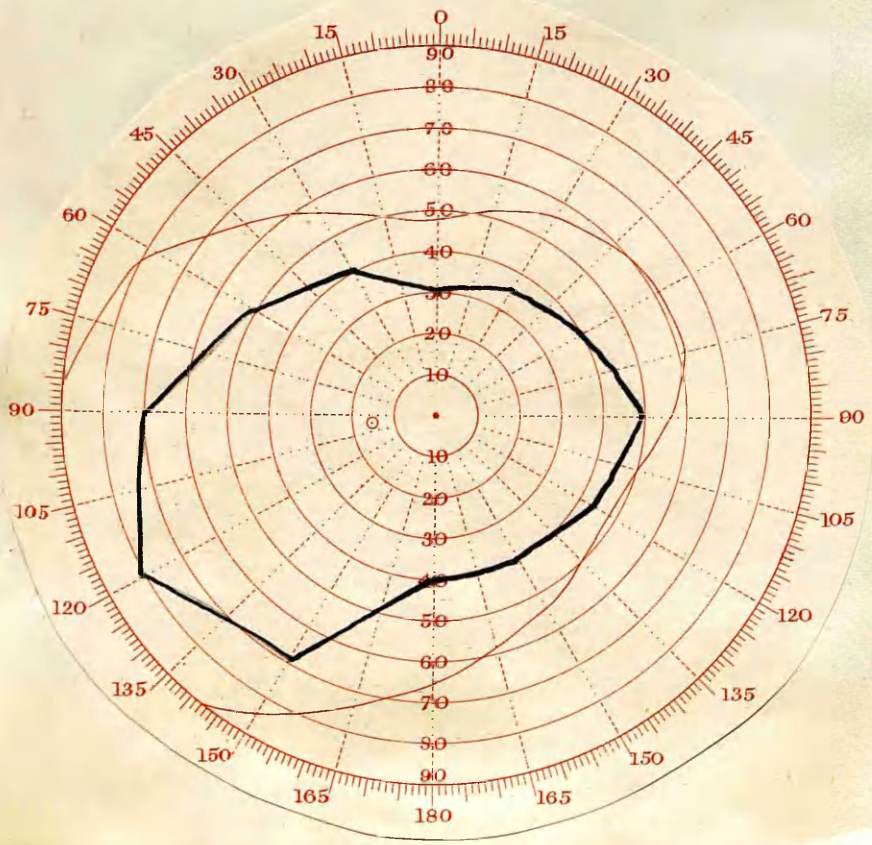


CHART VI.
Sgt. Es Sghair.
28.2.44.
3/330 White.

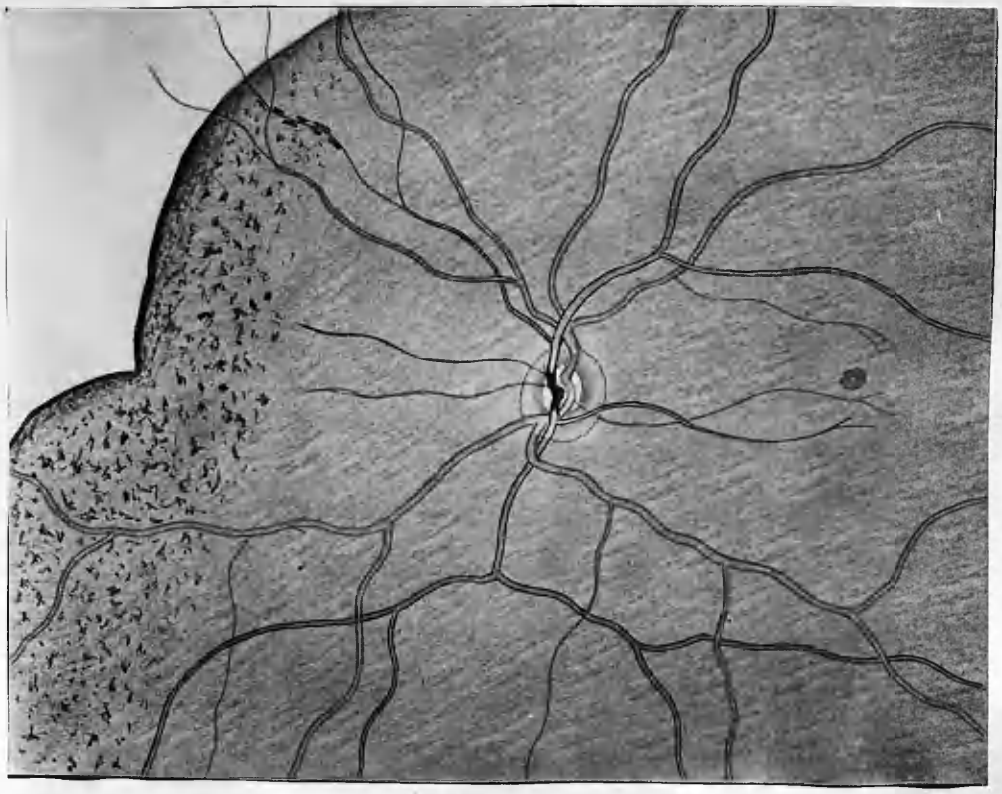


Fig. X.

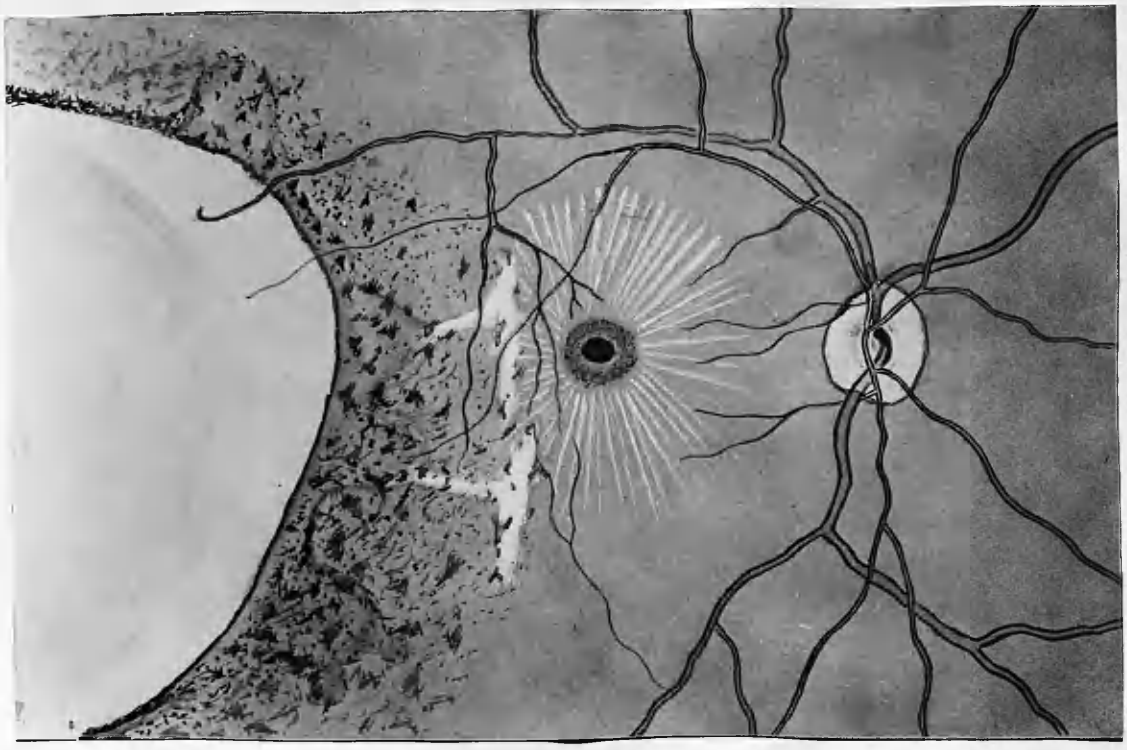
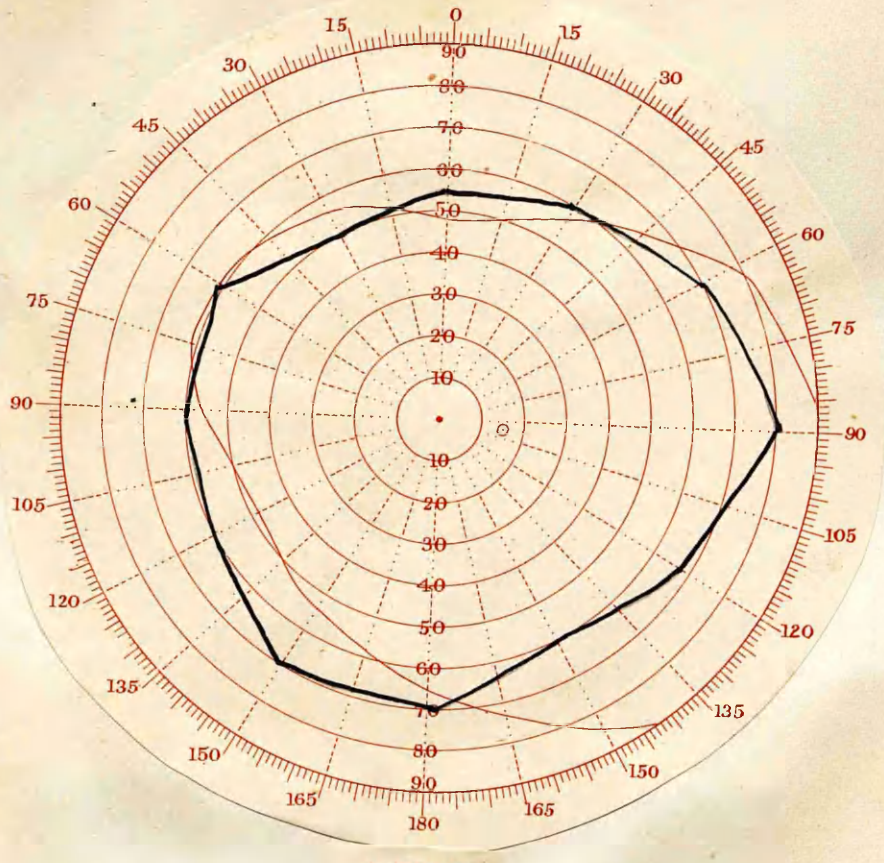


Fig. XI.

RIGHT.



LEFT.

CHART VII.
Pte. Walters.
15.7.44.
5/330 White.

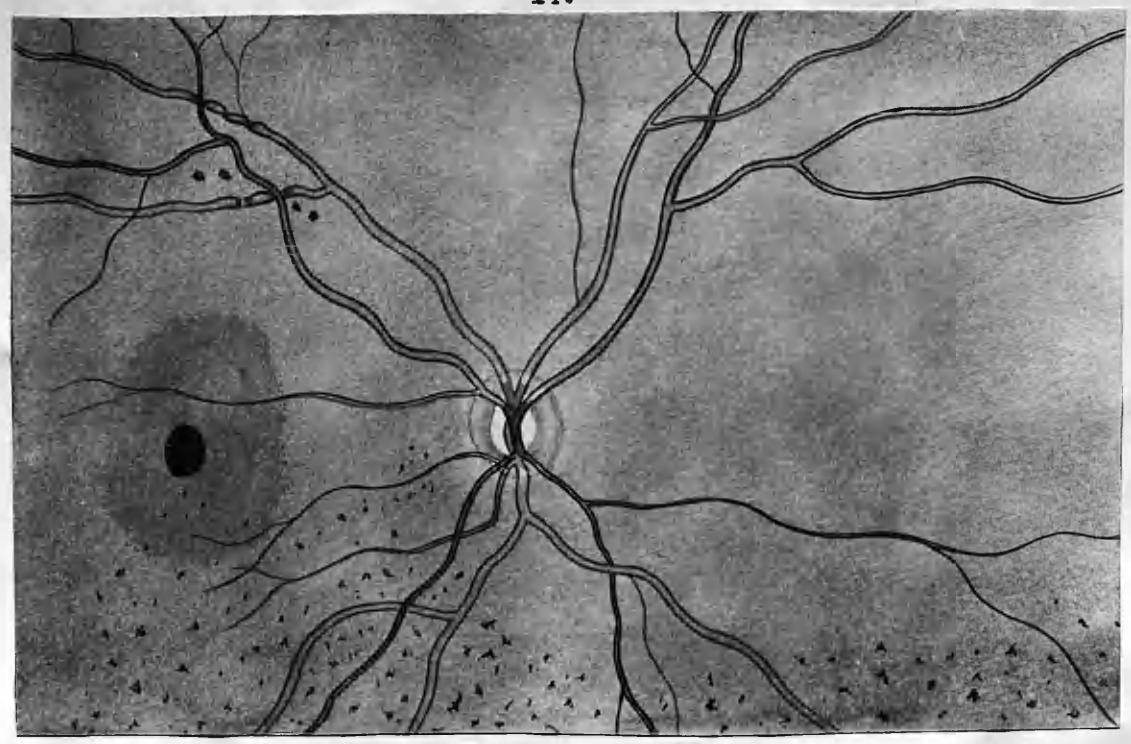


Fig. XII.

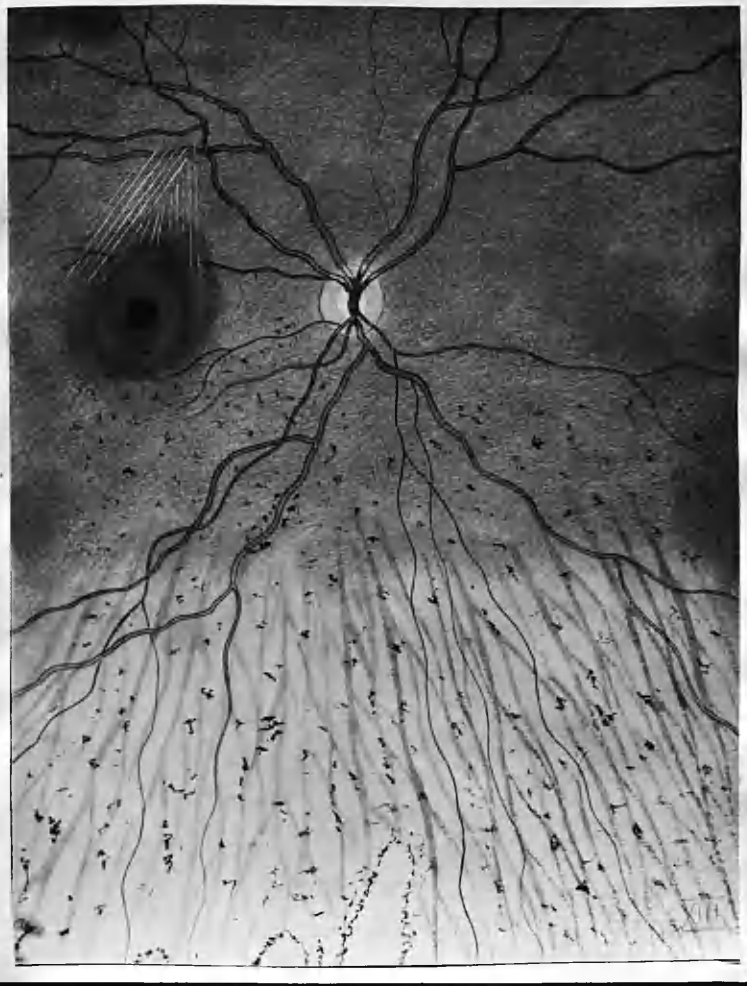
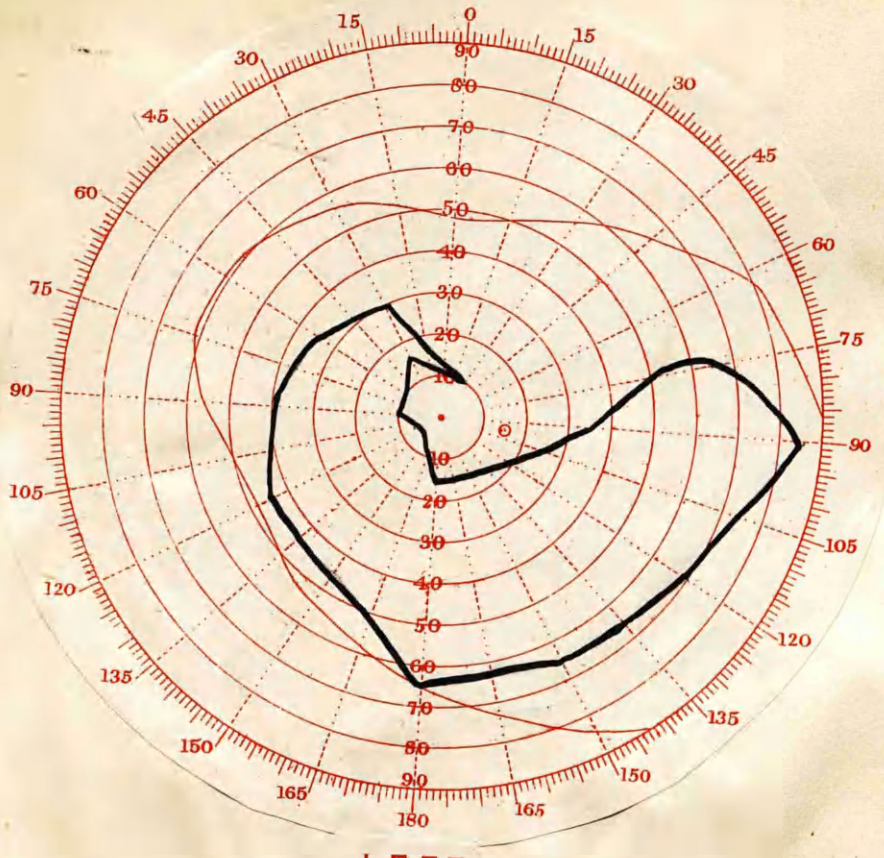


Fig. XIII.

RIGHT.



LEFT

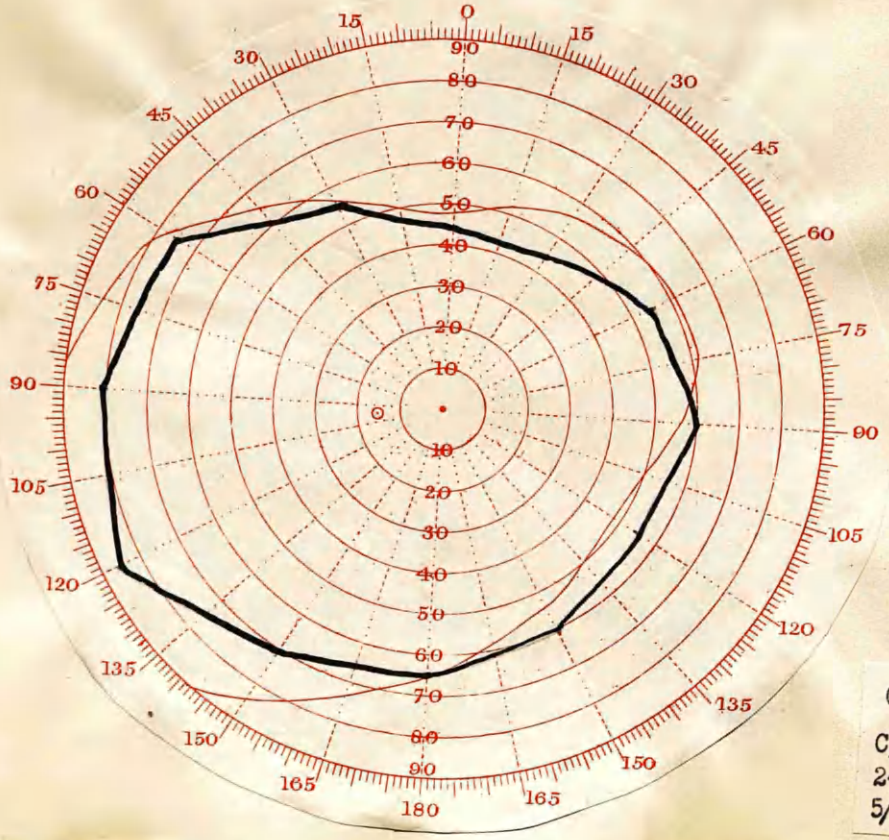


CHART VIII.

Cpl. Bainbridge.
24.10.44.
5/330. White.

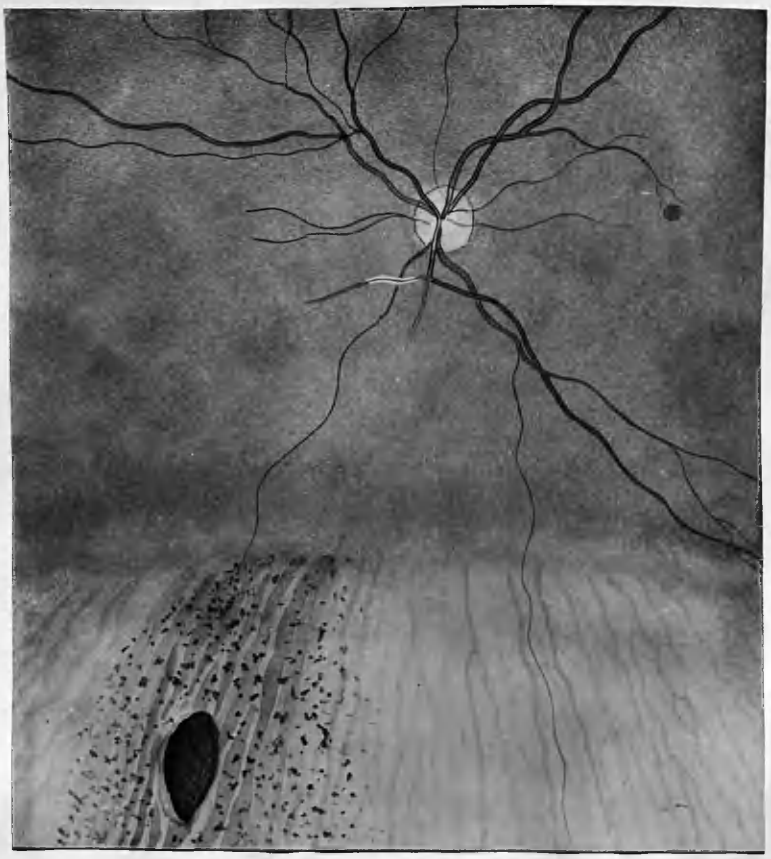


Fig. XIV.

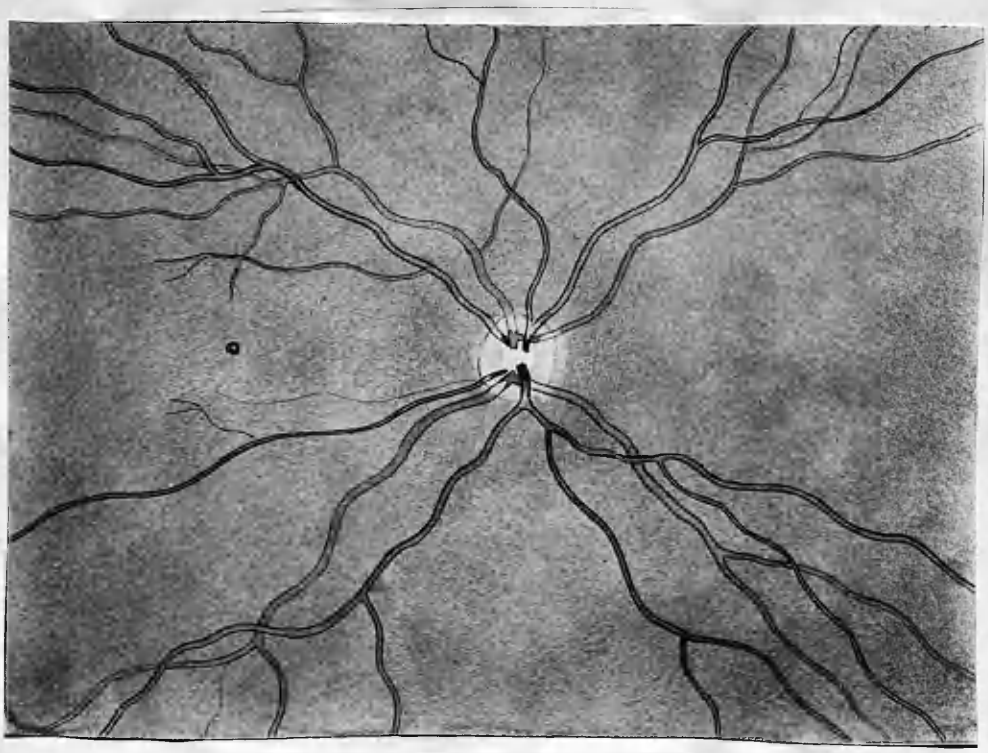
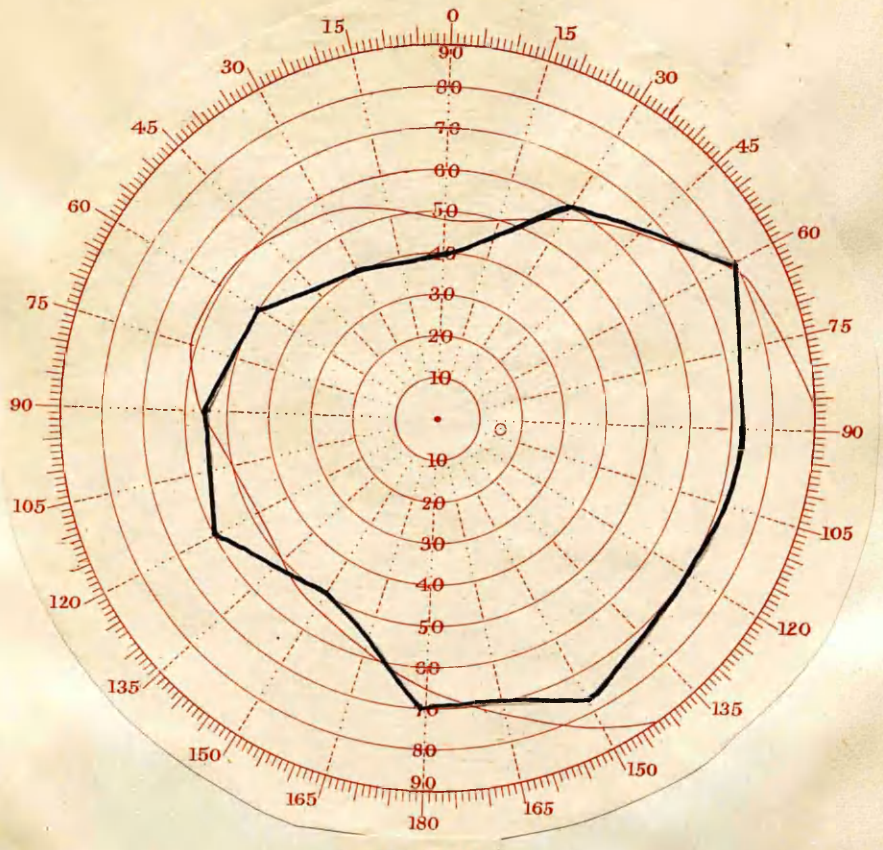


Fig. XV.

RIGHT.



LEFT.

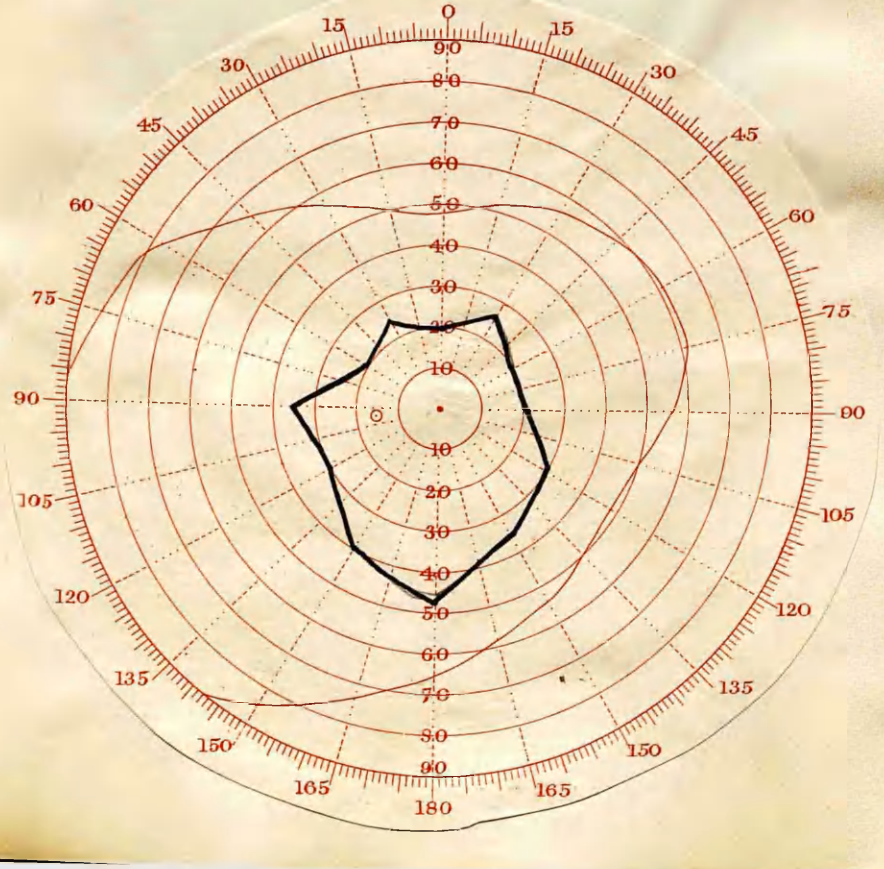


CHART IX.
Spr. Kelly.
3.11.44.
2/330 White.

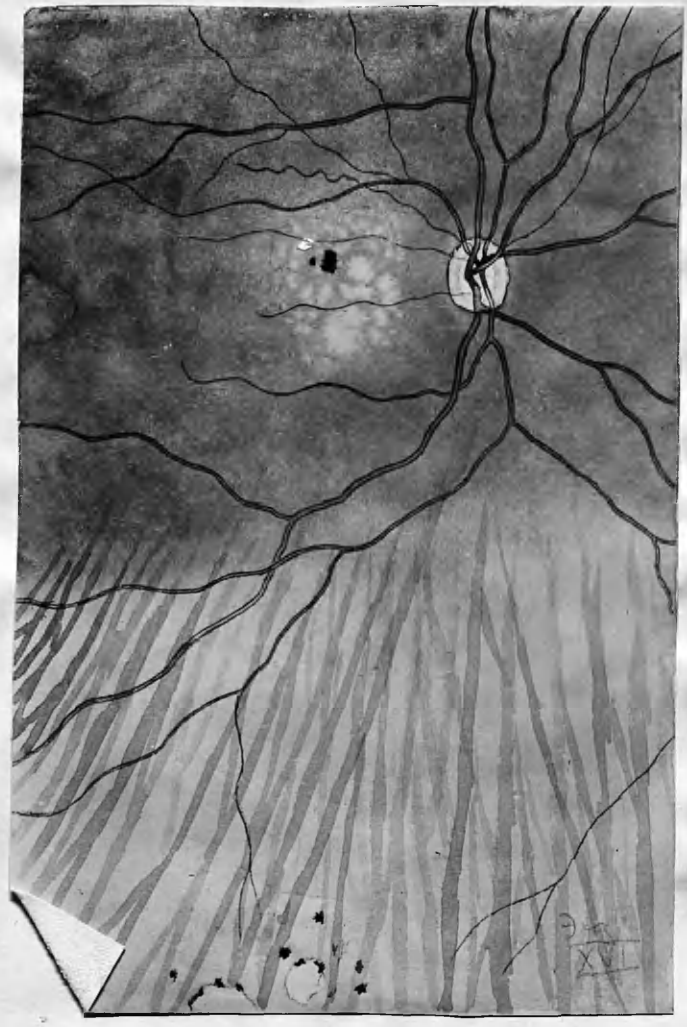


Fig. XVI.

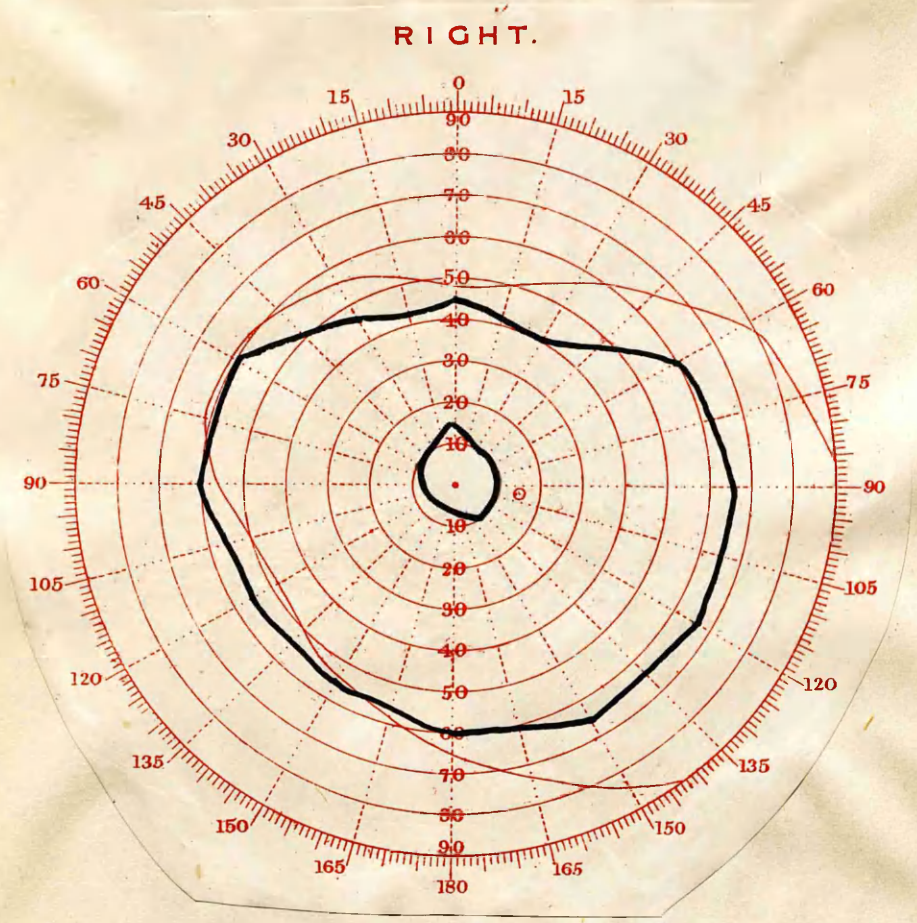


CHART X.
Spr. Taylor.
8.7.44.
3/330. White.

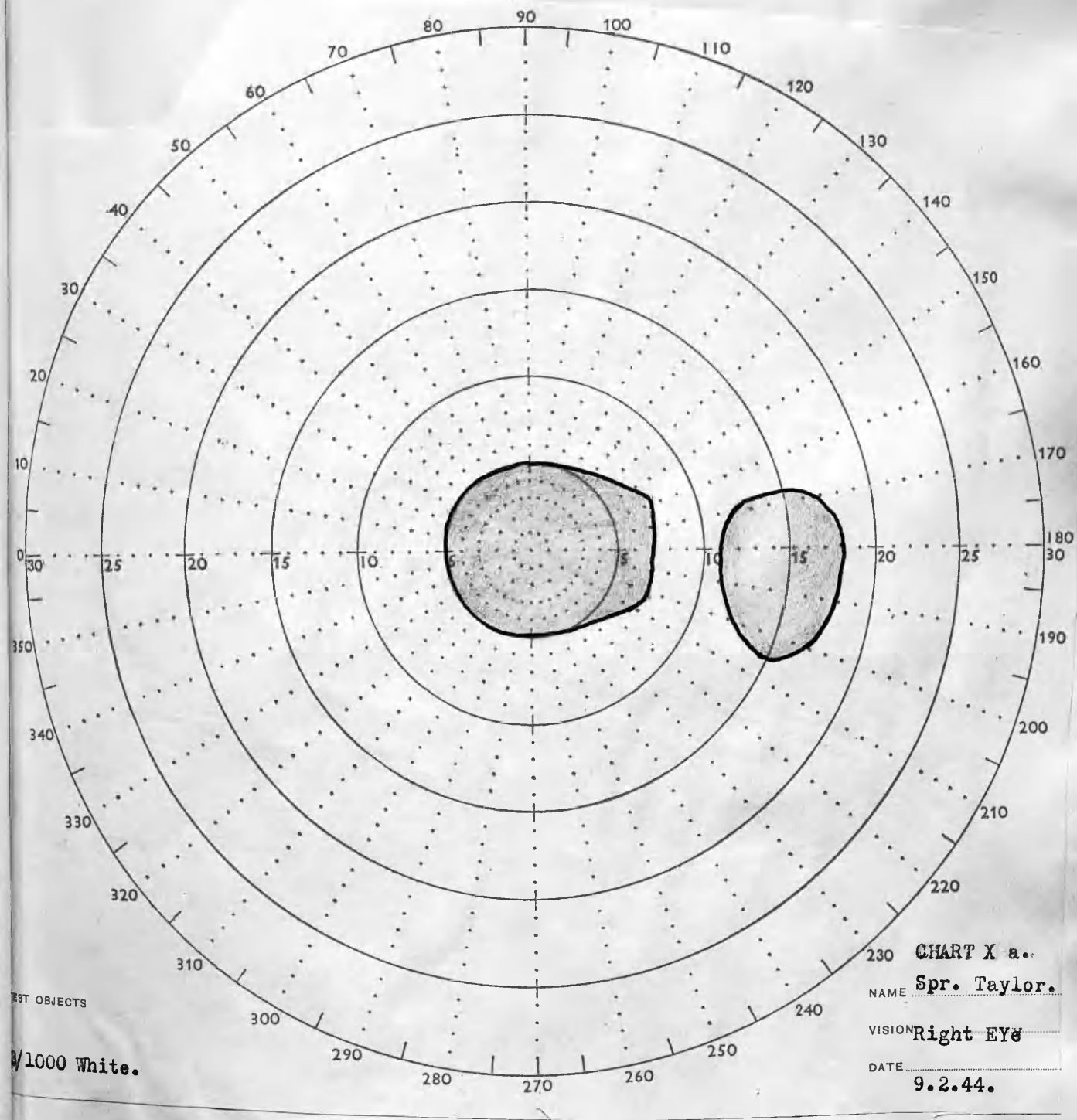


CHART X a.

NAME Spr. Taylor.

VISION Right EYe

DATE 9.2.44.

EST OBJECTS

/1000 White.

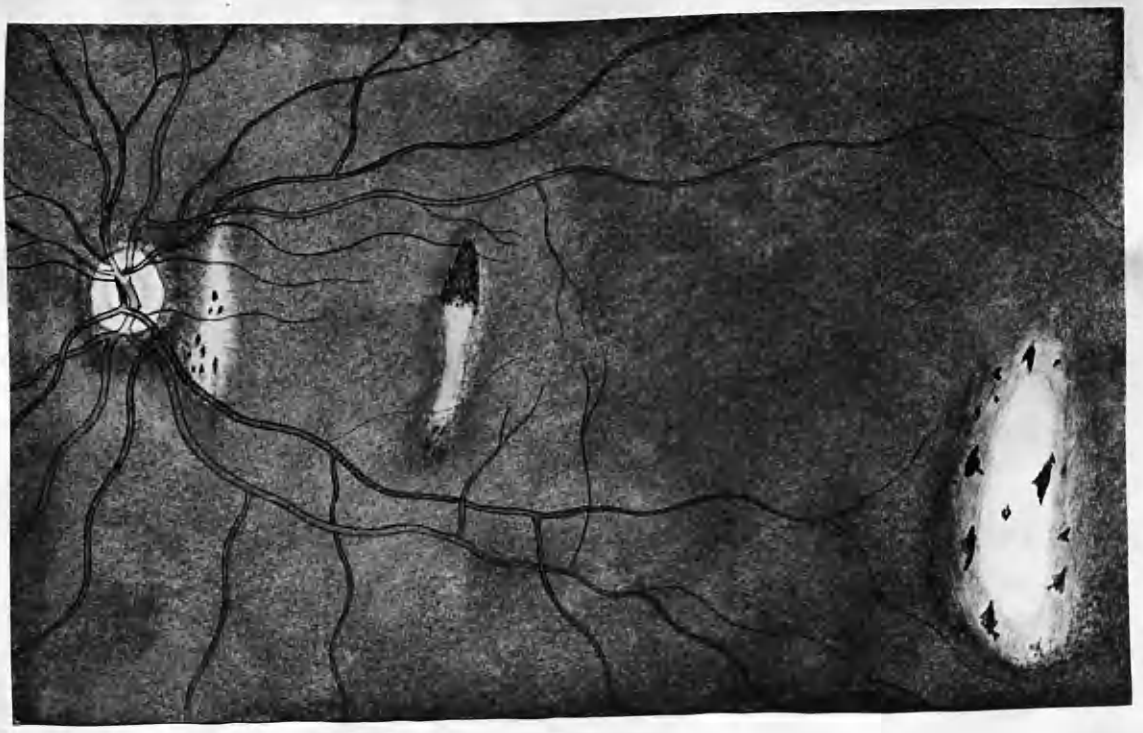


Fig. XVII.

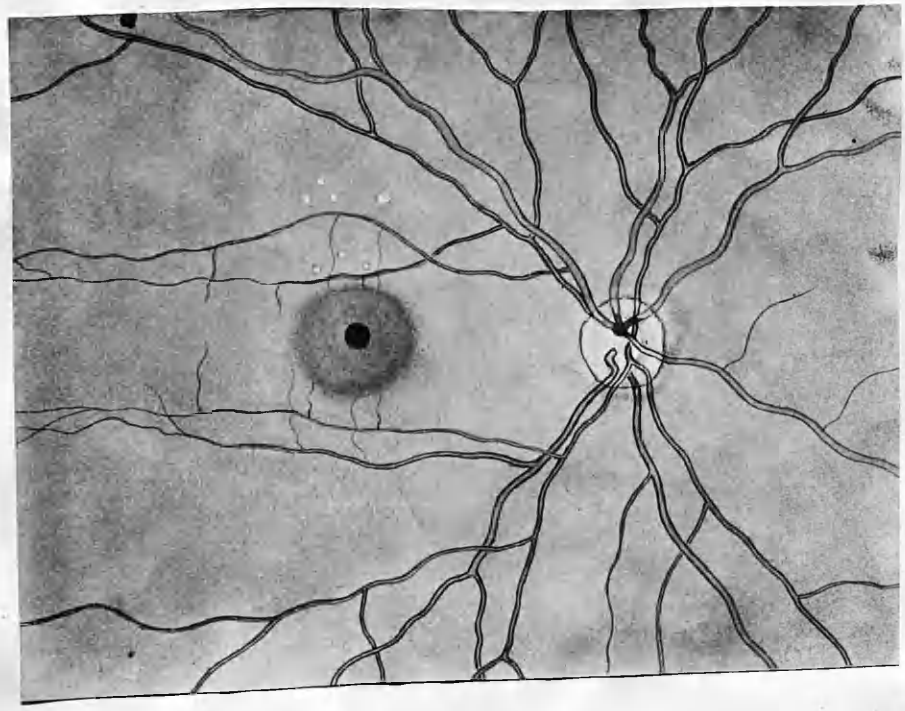
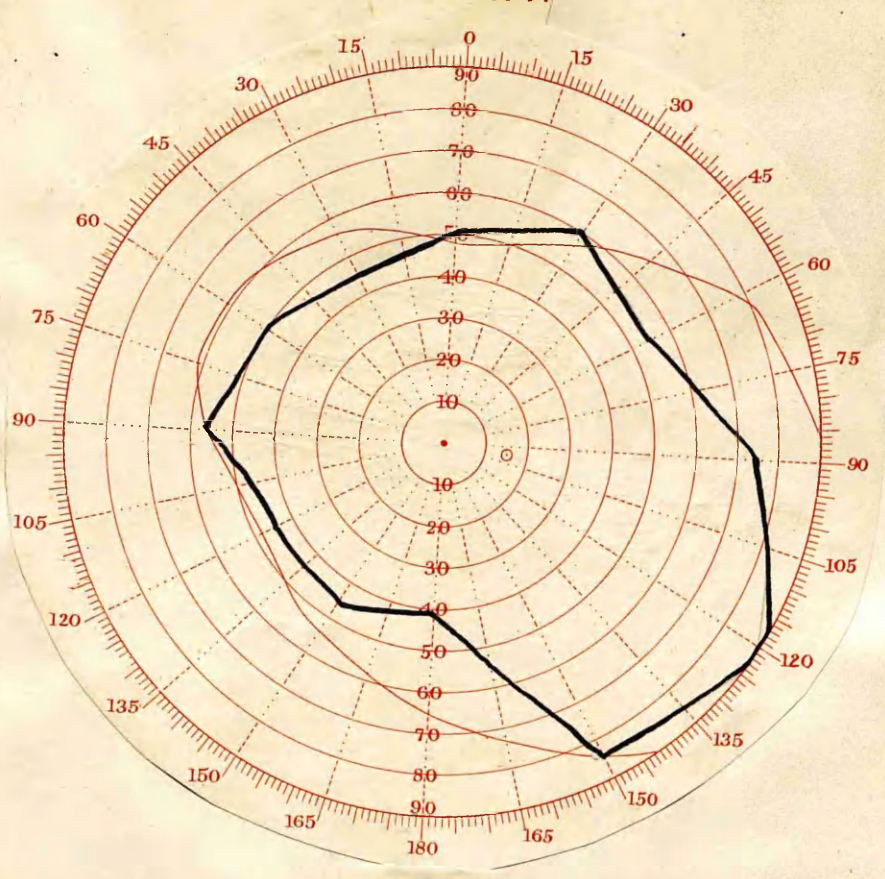


Fig. XVIII.

RIGHT.



LEFT.

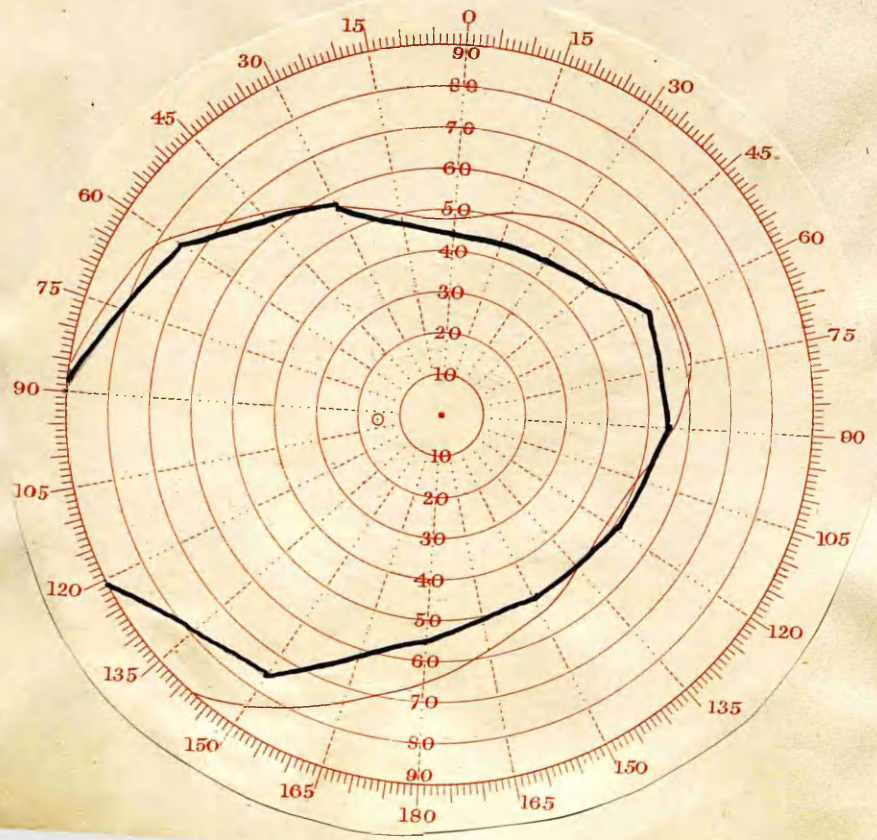


CHART XI.
Sgt. Johnson.
21.8.44.
3/330 White.

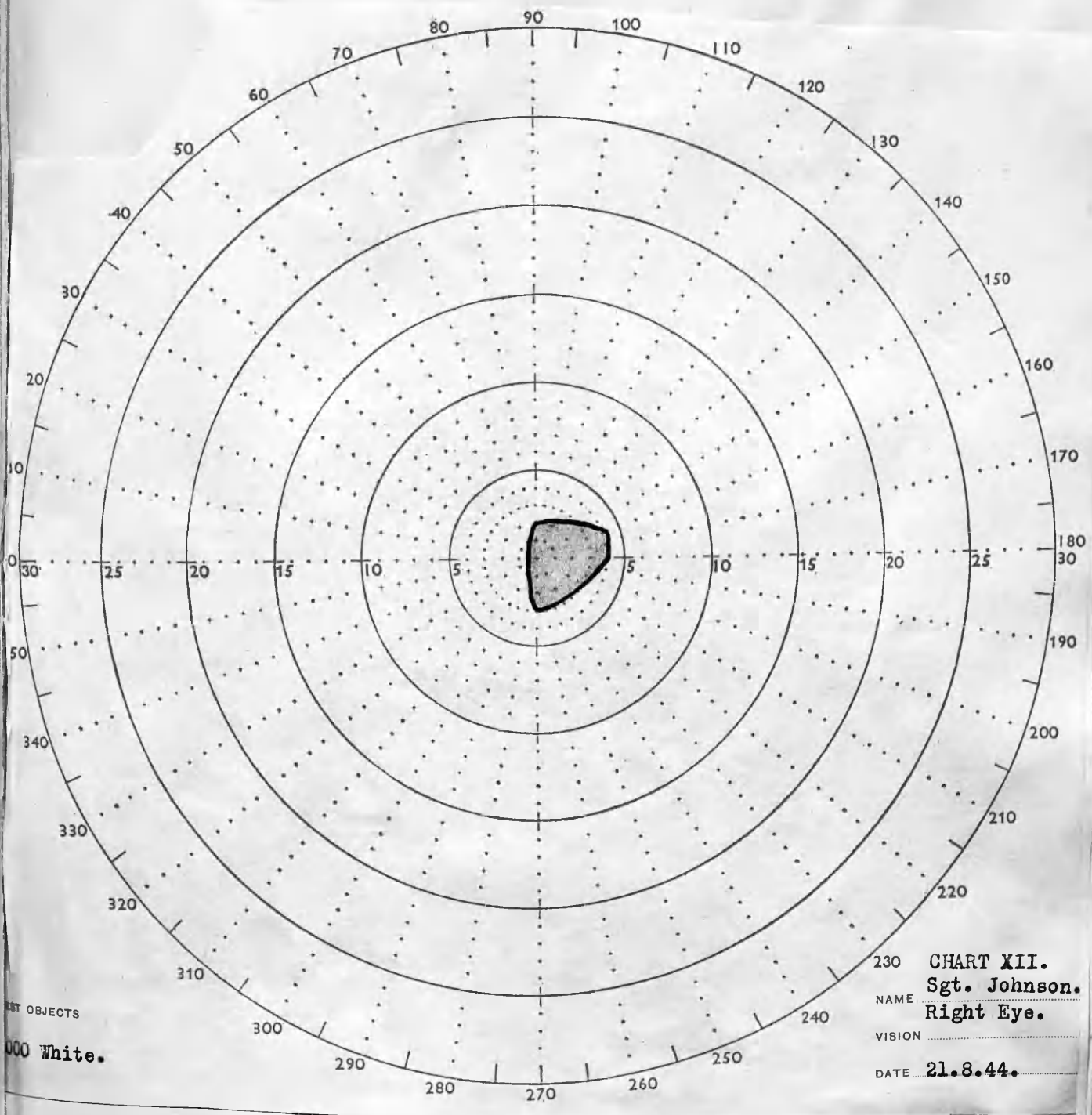


CHART XII.

NAME Sgt. Johnson.
Right Eye.

VISION .....

DATE 21.8.44.

...ST OBJECTS

...000 White.

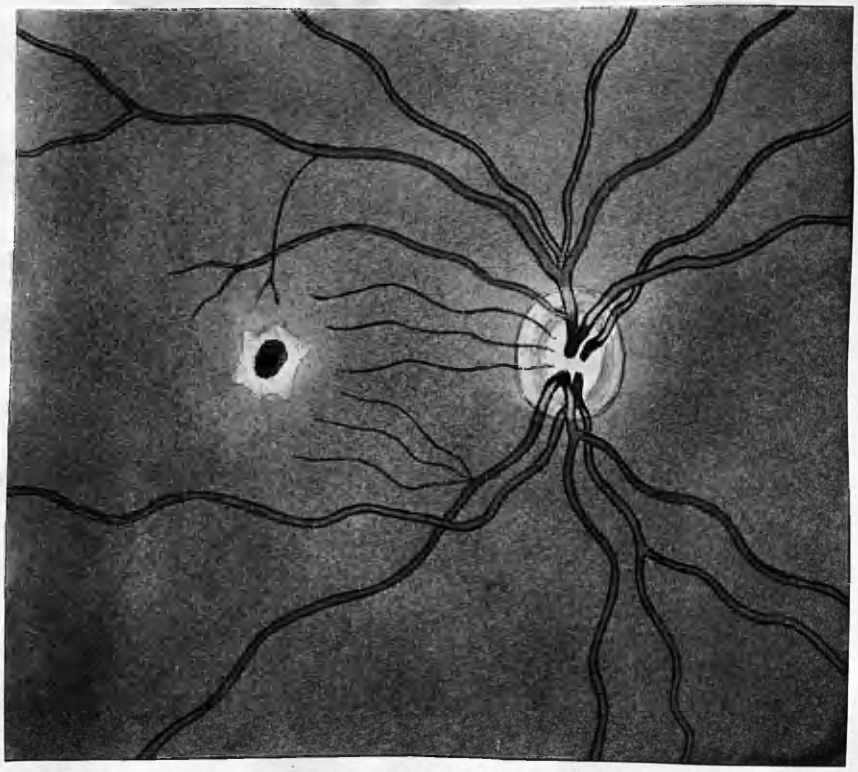


Fig. XIX.

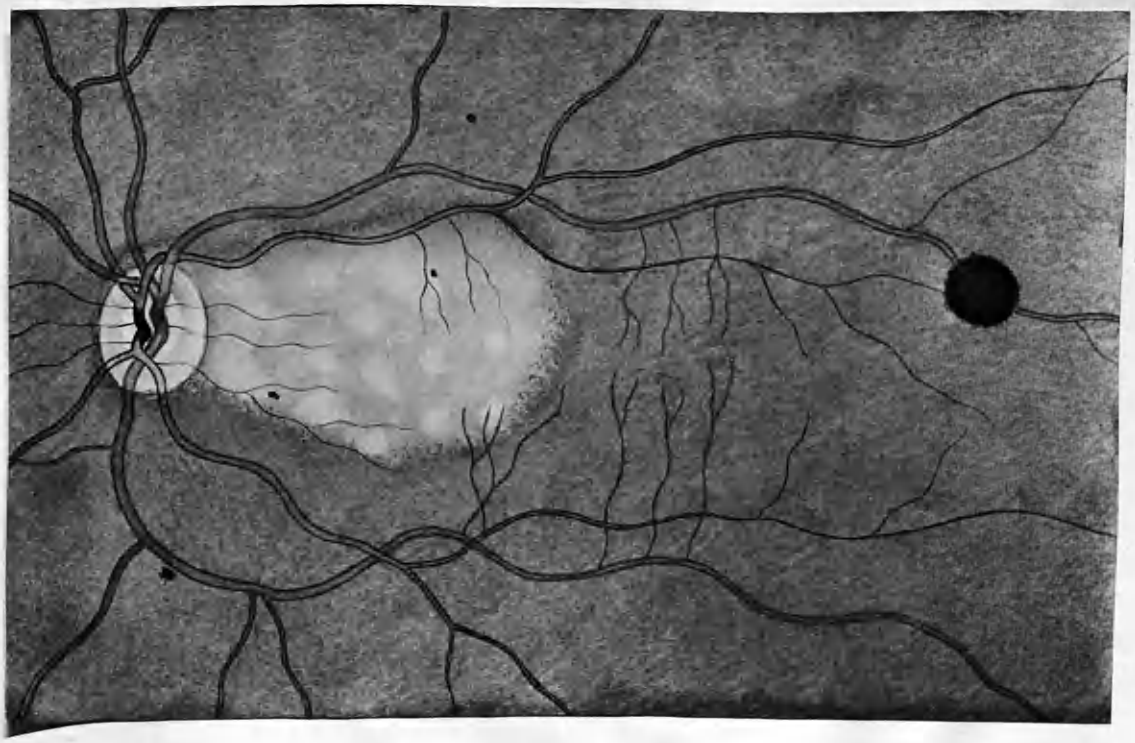


Fig. XX.

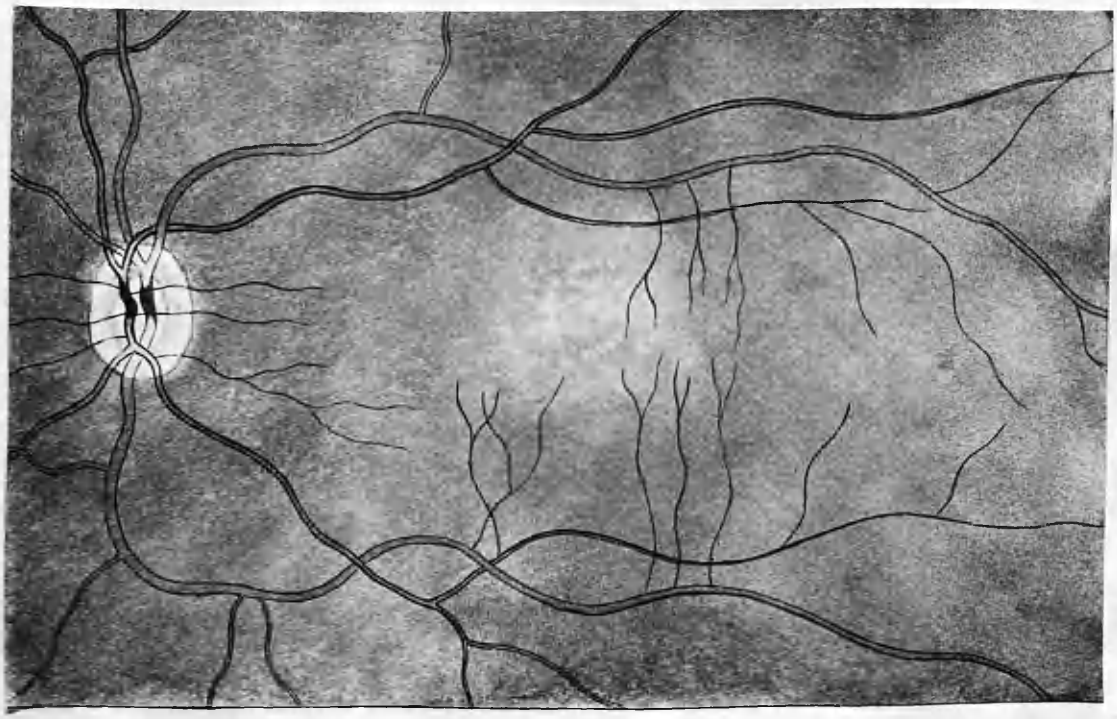
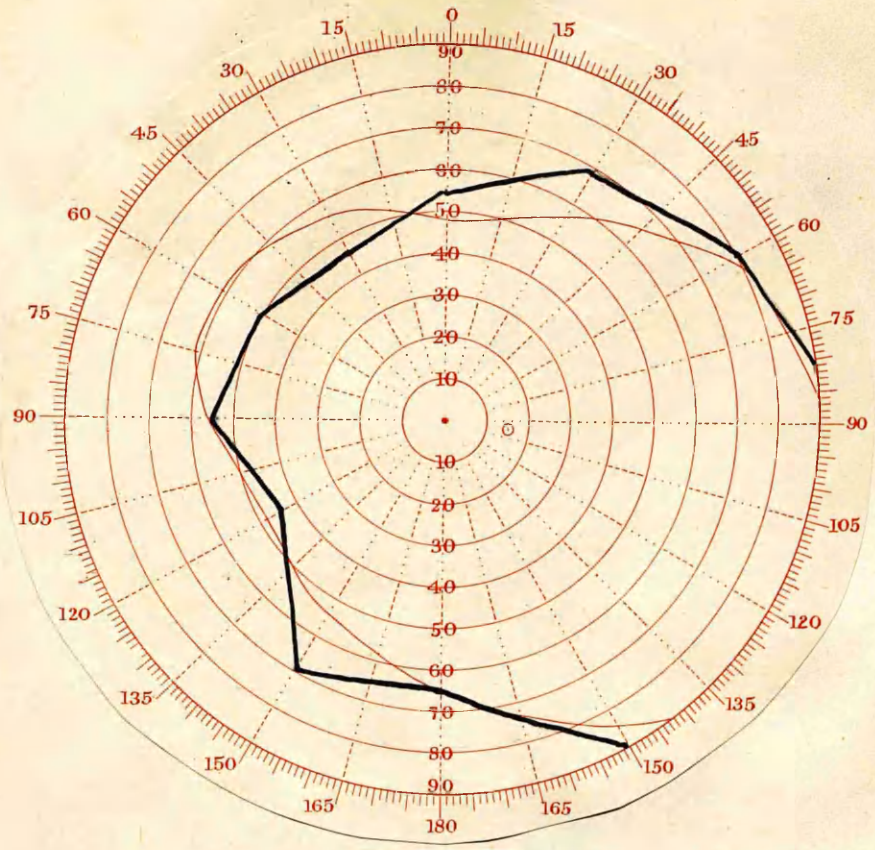


Fig. XXI.

RIGHT.



LEFT.

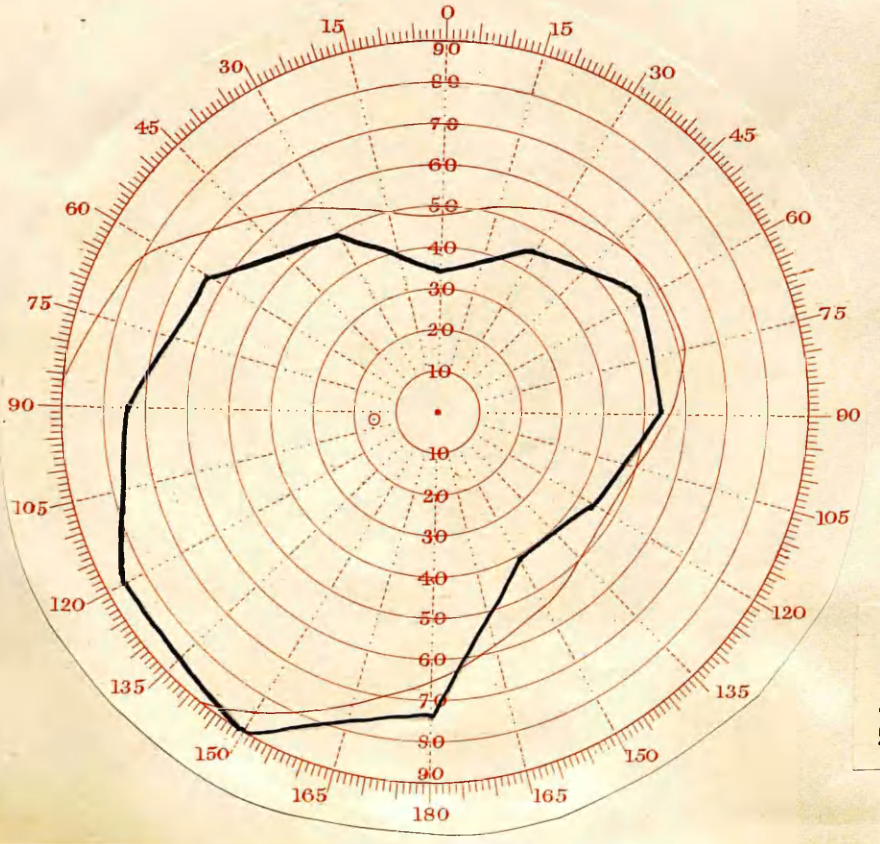


CHART XIII.
Sgt. Ainley.
31.1.45.
5/330 White.

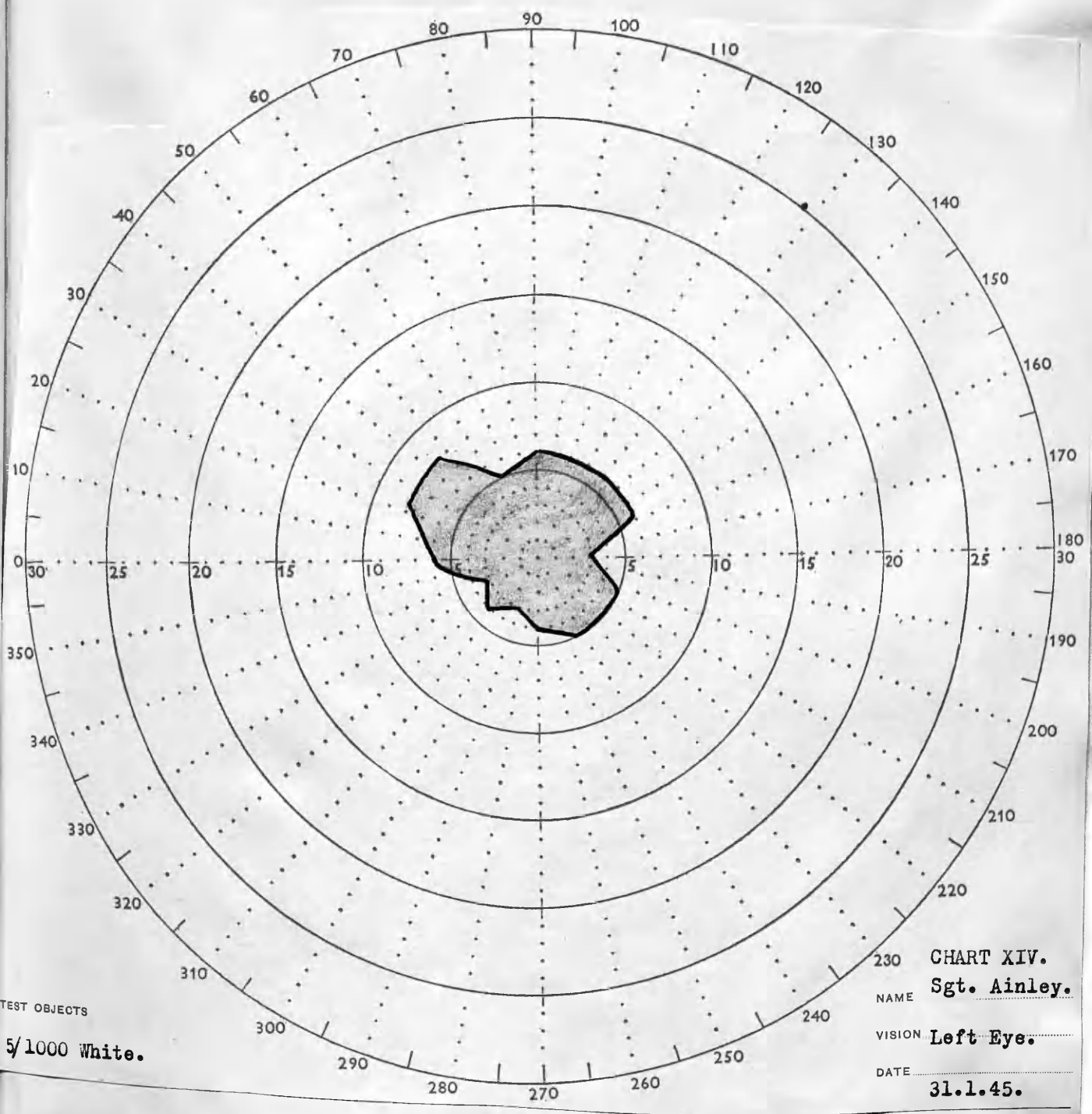


CHART XIV.

NAME Sgt. Ainley.

VISION Left Eye.

DATE 31.1.45.

TEST OBJECTS

5/1000 White.

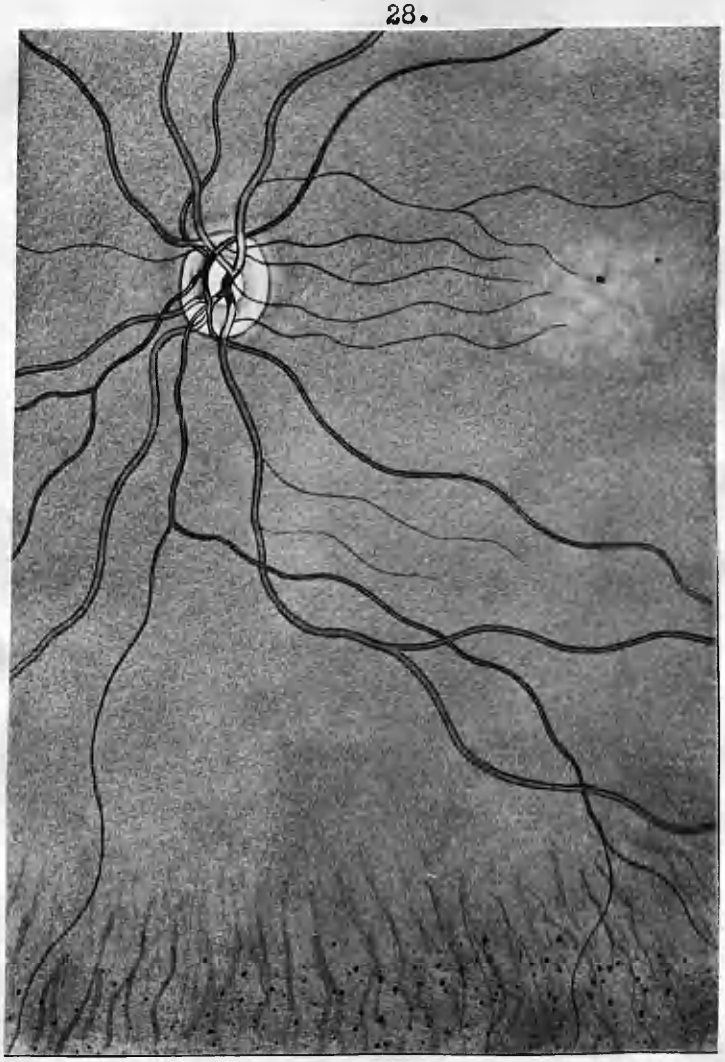


Fig. XXII.

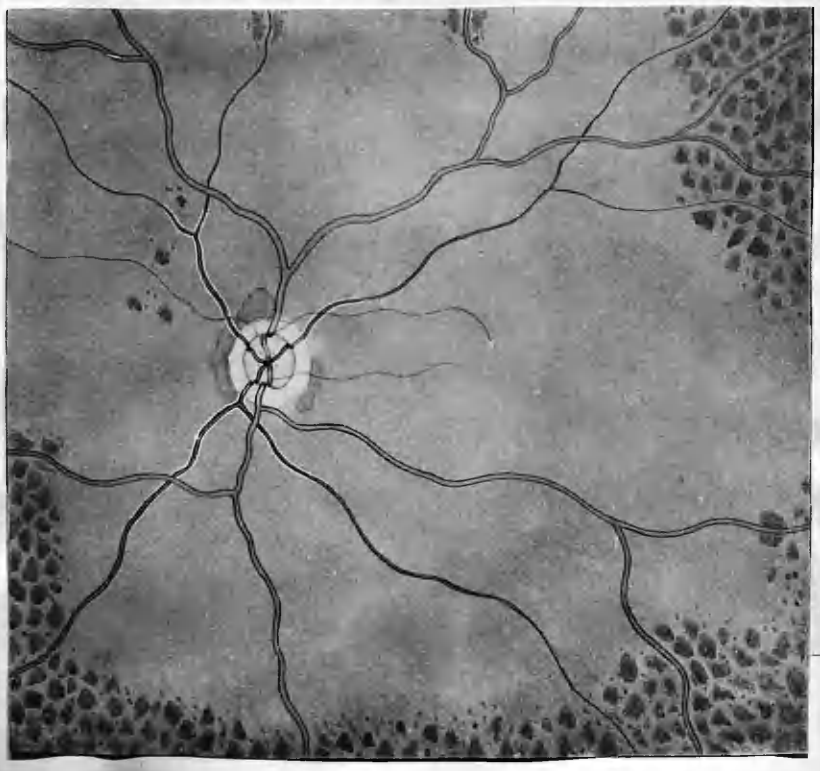
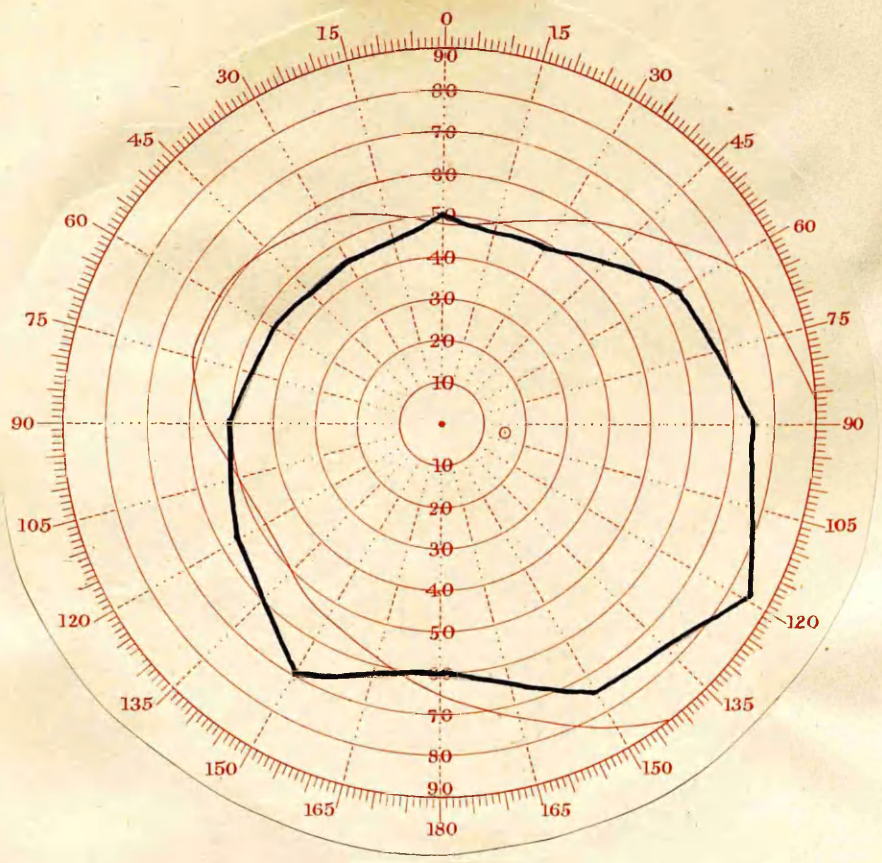


Fig. XXIII.

RIGHT.



LEFT.

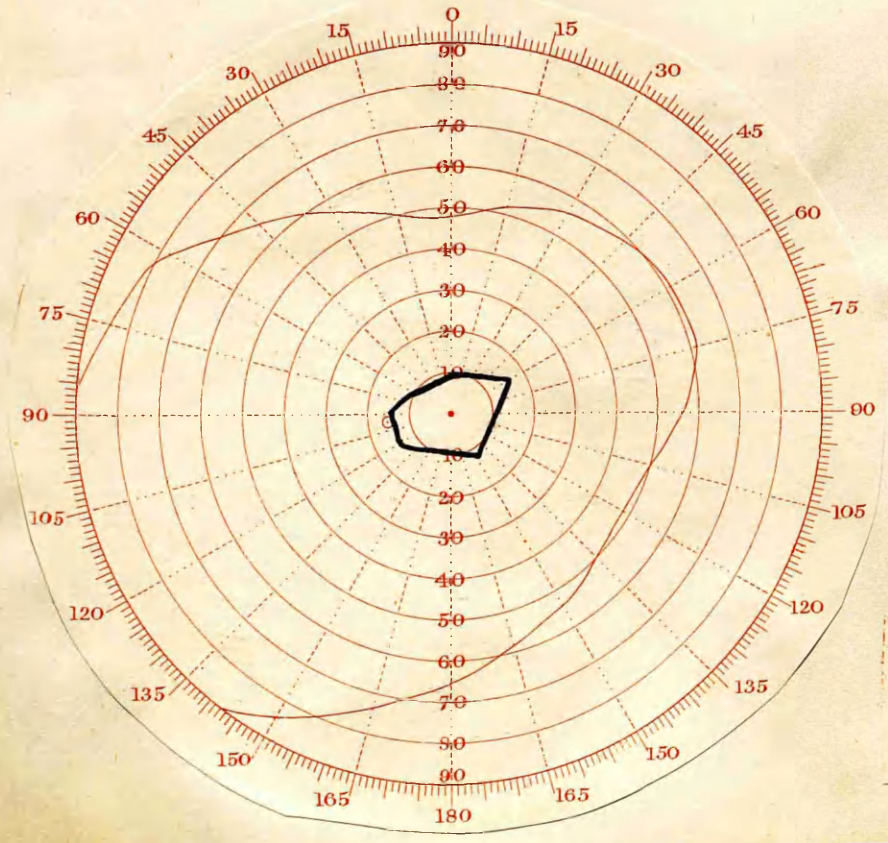
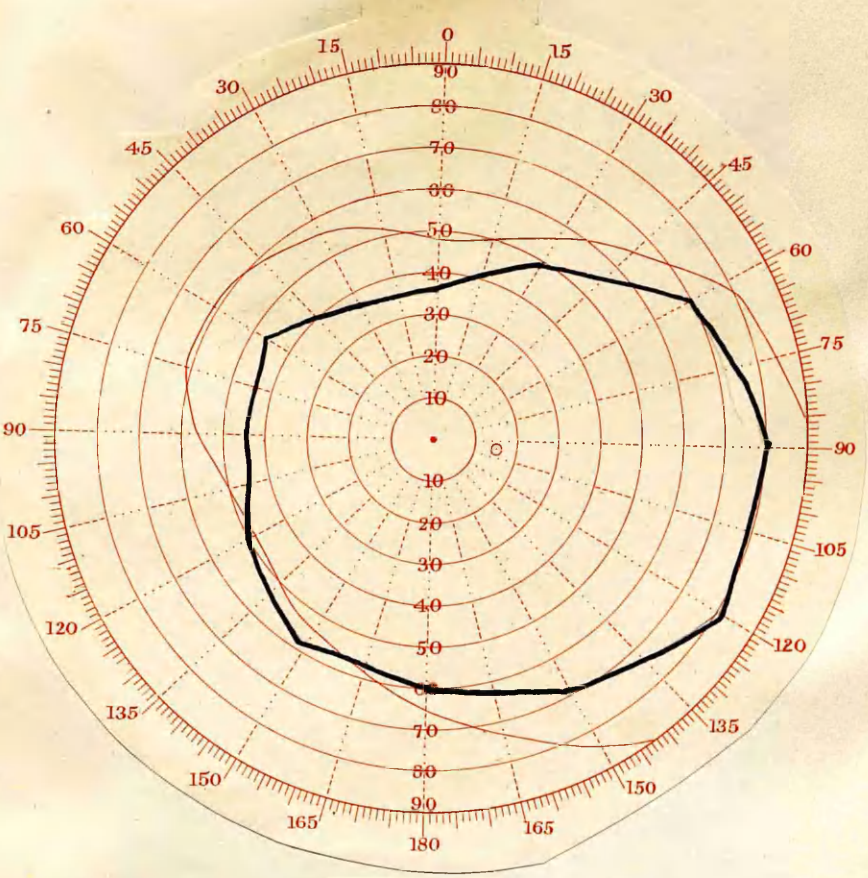


CHART XV.
Gdsm. Humphries.
29.10.43.
3/330 White.

RIGHT.



LEFT.

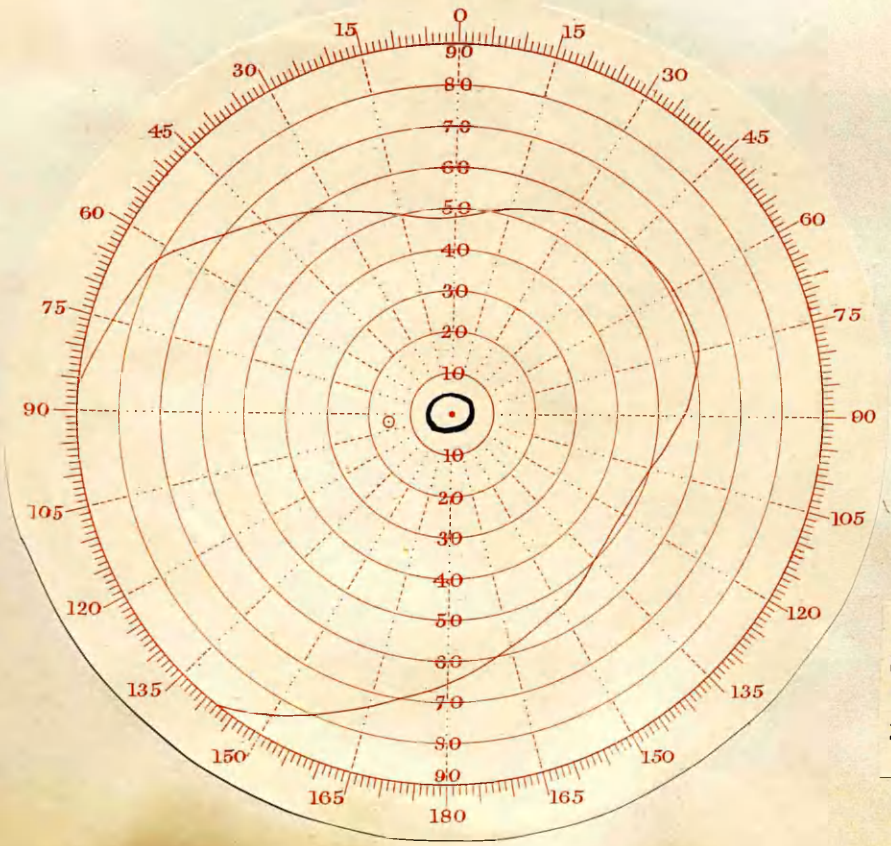


CHART XVI.
Gdsm. Humphries.
24.12.43.
3/330 White.

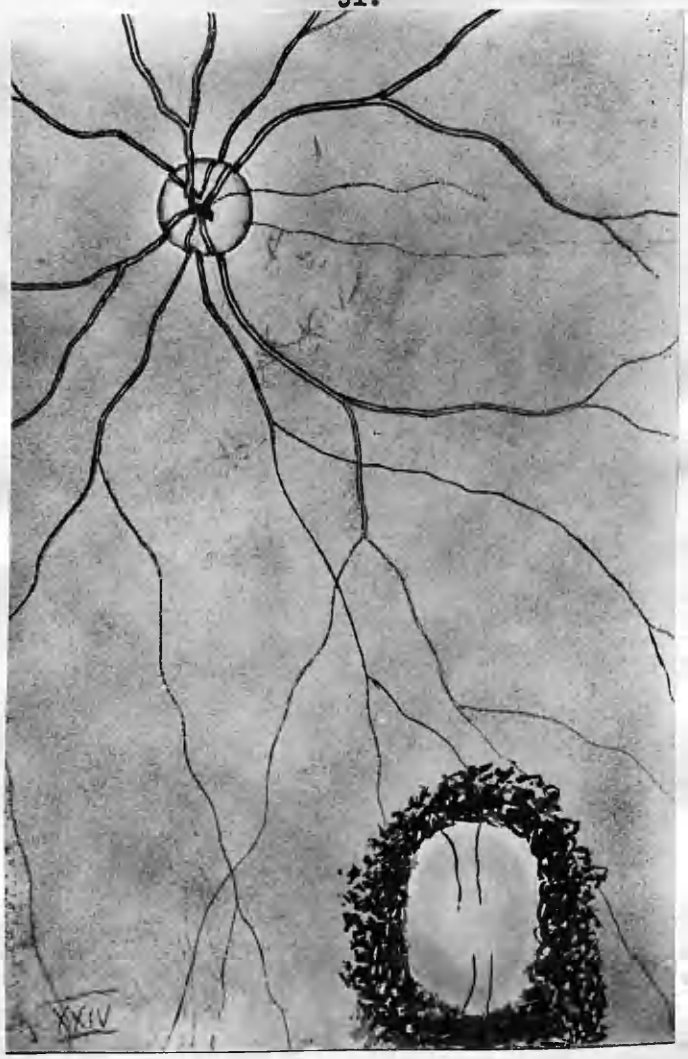


Fig. XXIV.

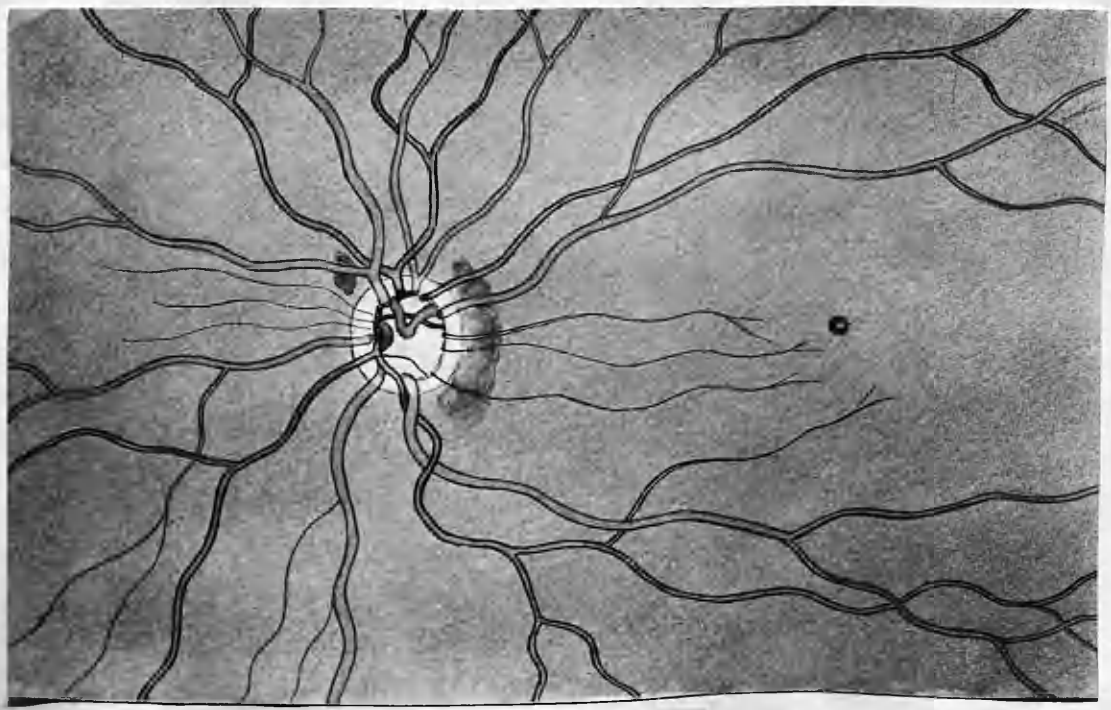


Fig. XXV.

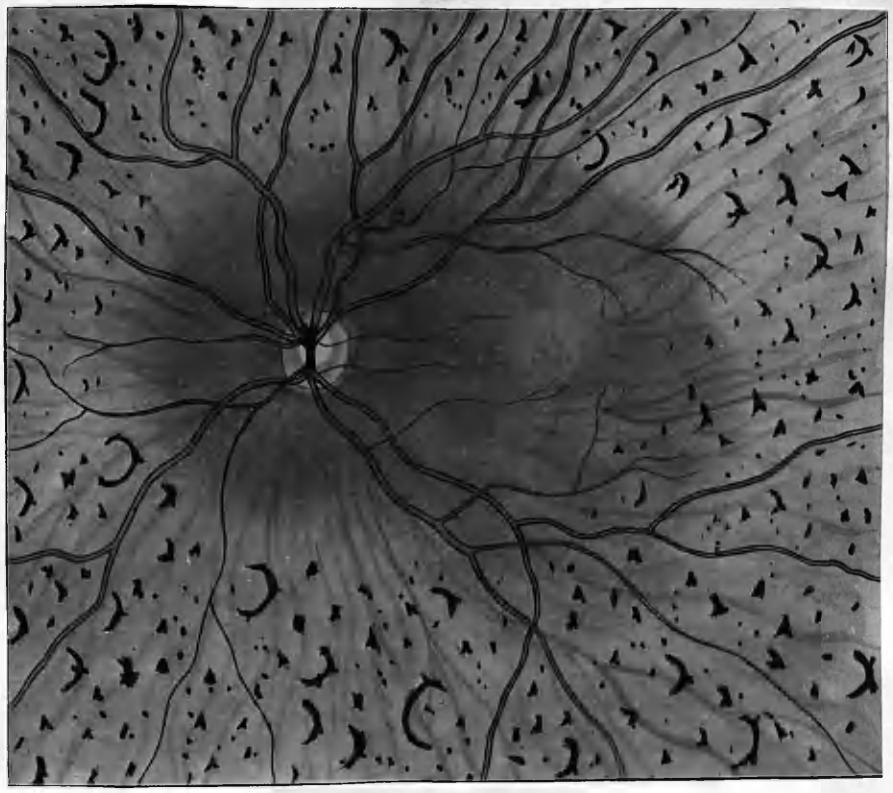


Fig. XXVI.

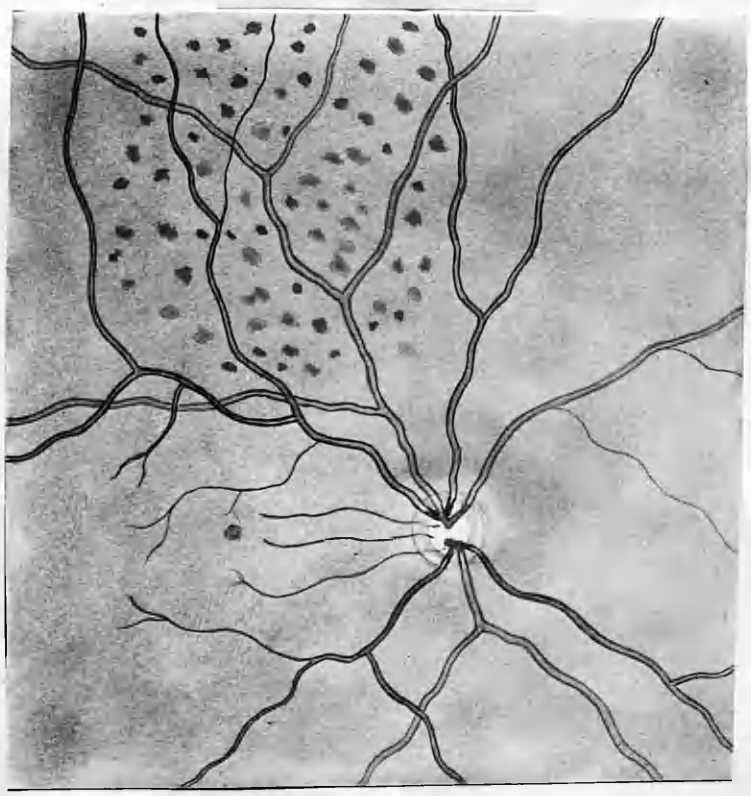


Fig. XXVII.

RIGHT.

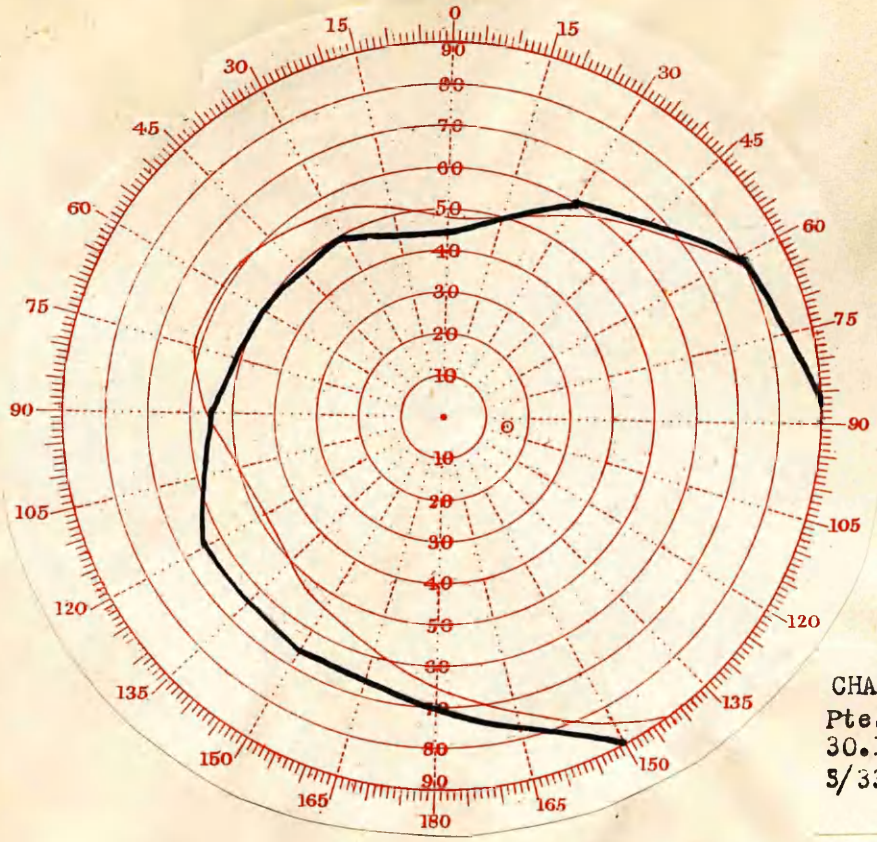


CHART XVII.
Pte. Marginson.
30.10.44. (14.30)
5/330. White.

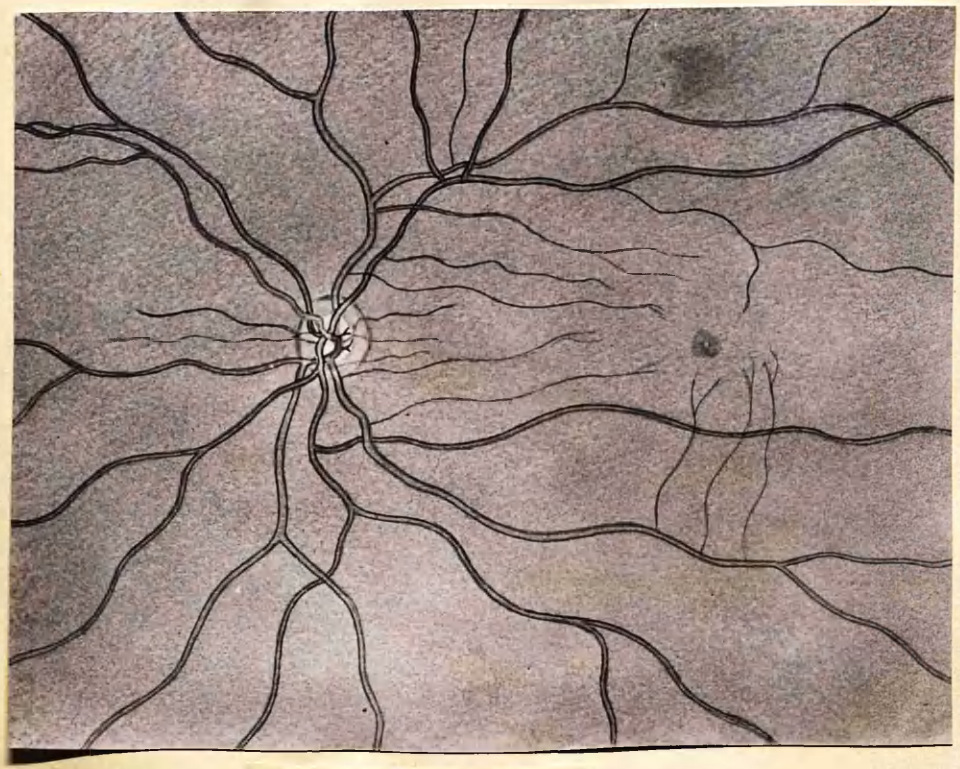
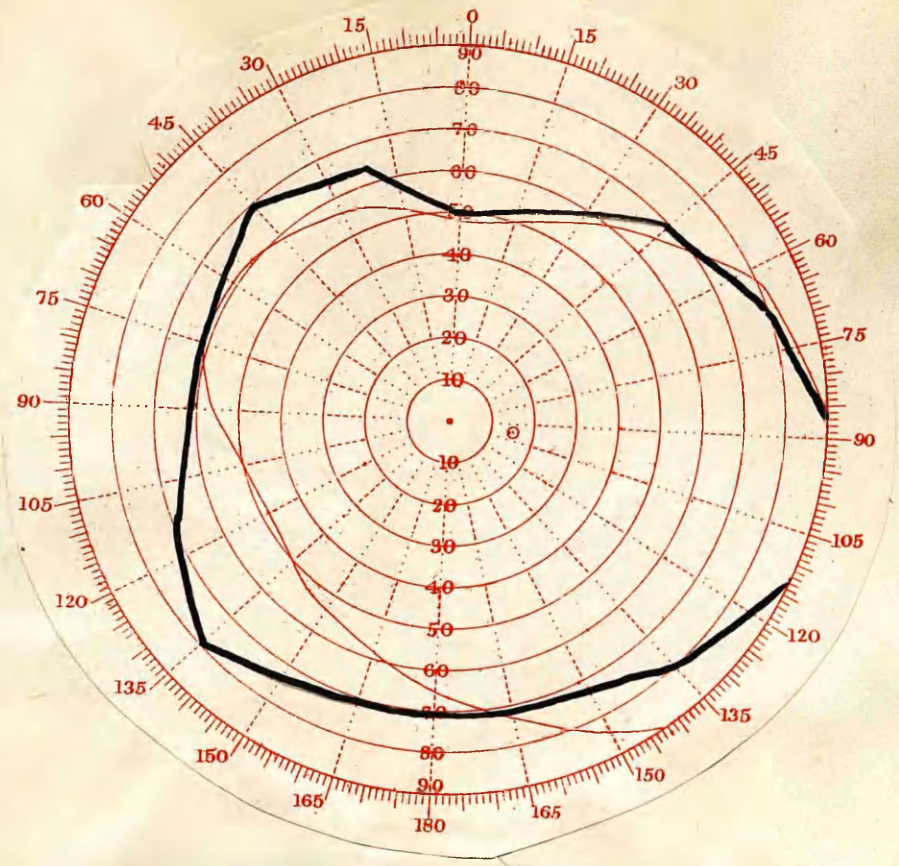


Fig. XXVIII.

RIGHT.



LEFT

CHART XVIII.
Pte. Spooner
23.2.45.
5/1000 White.
(Screen)

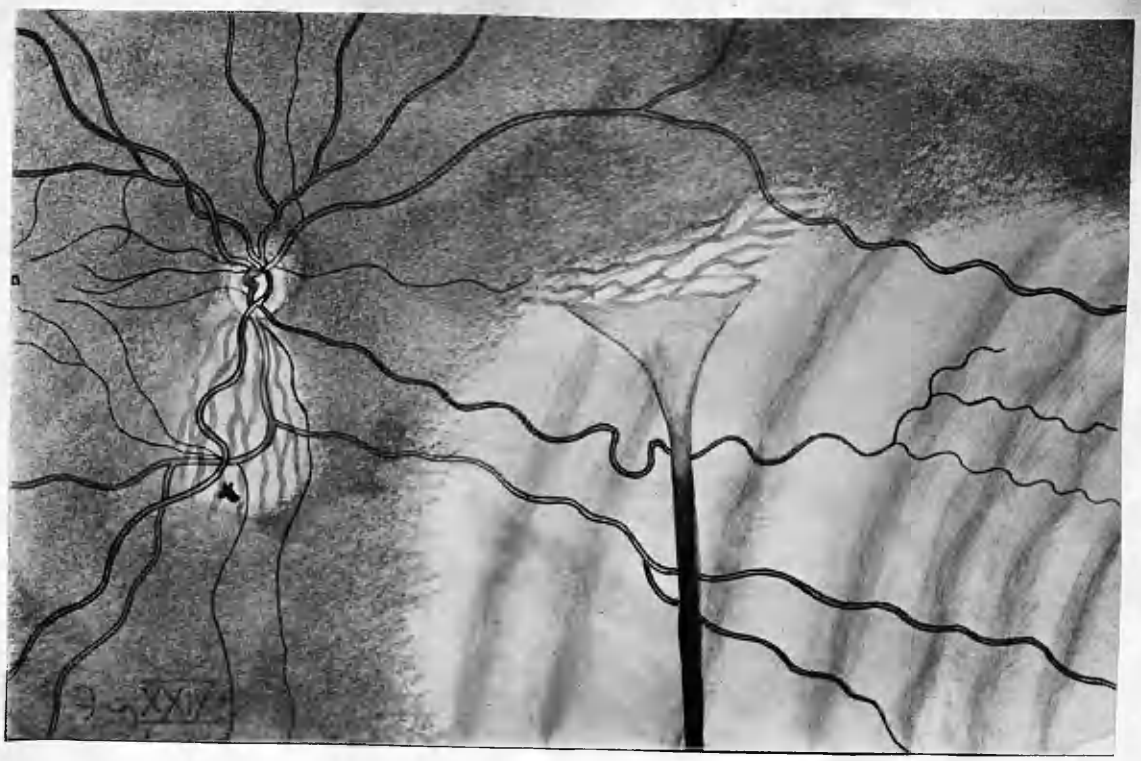


Fig. XXIX.

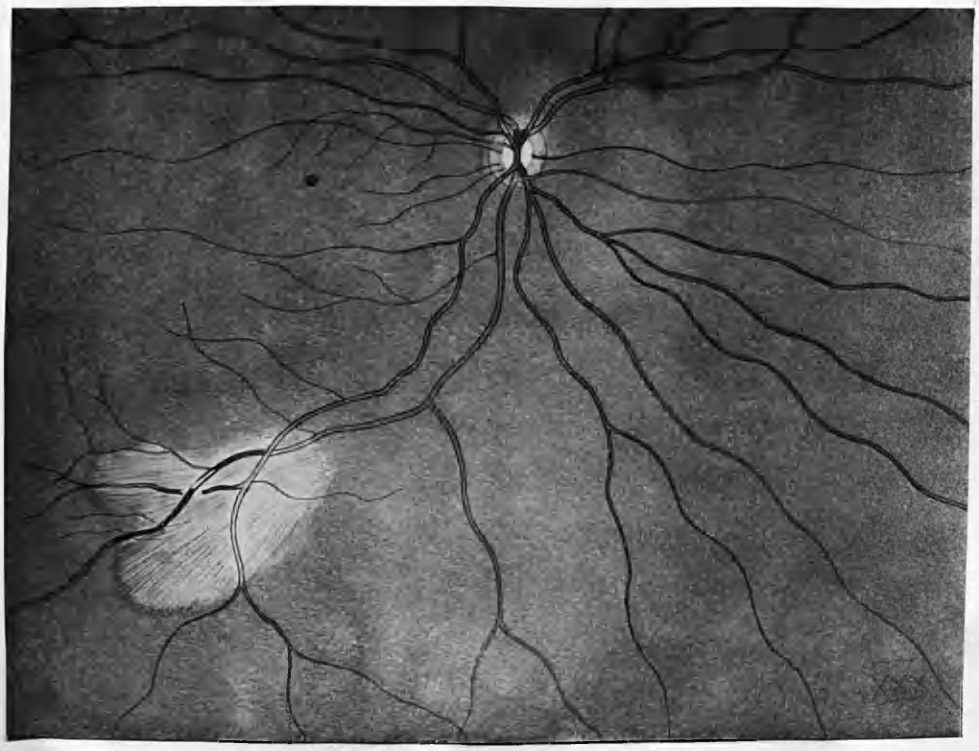


Fig. XXX.

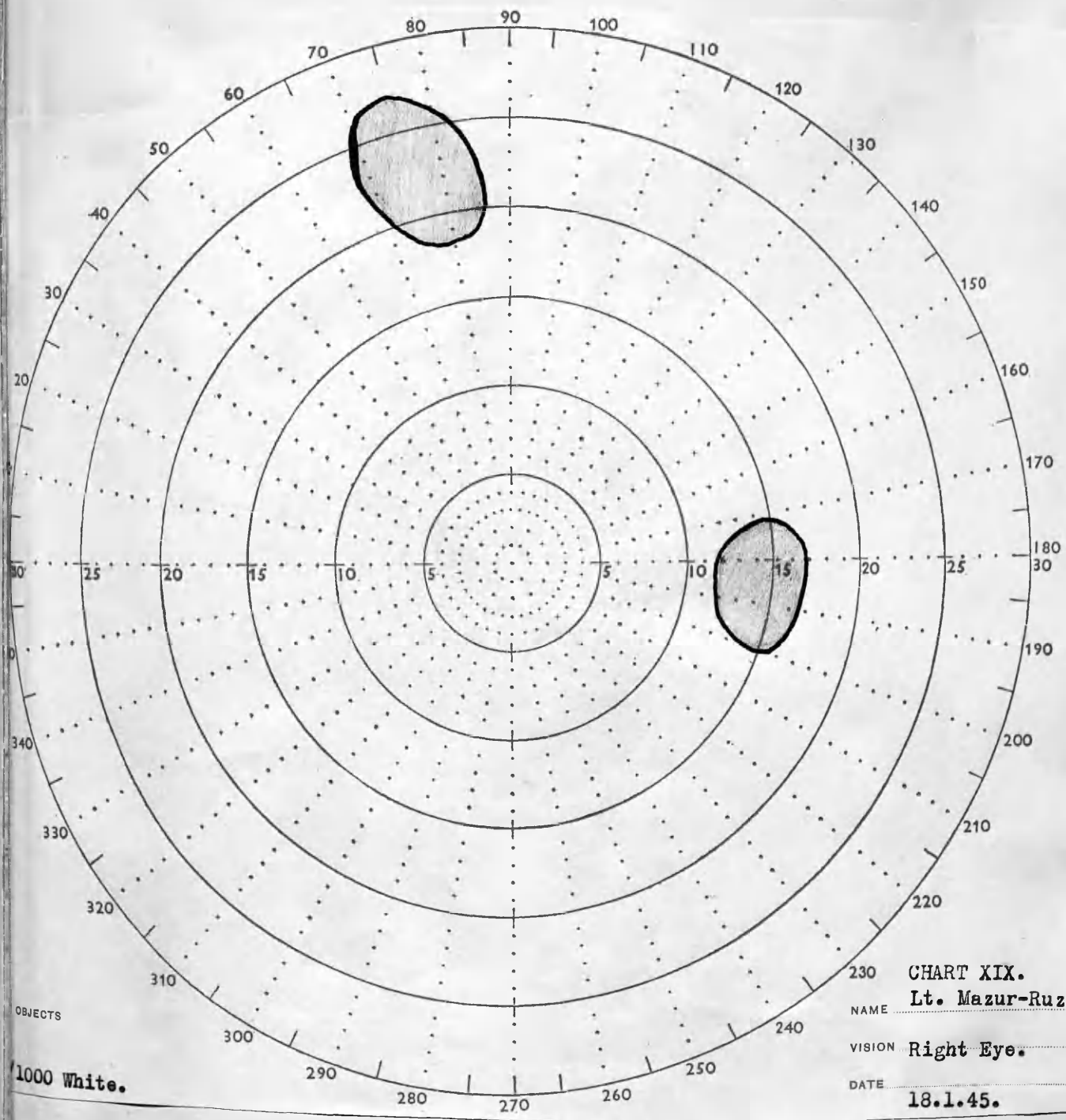


CHART XIX.

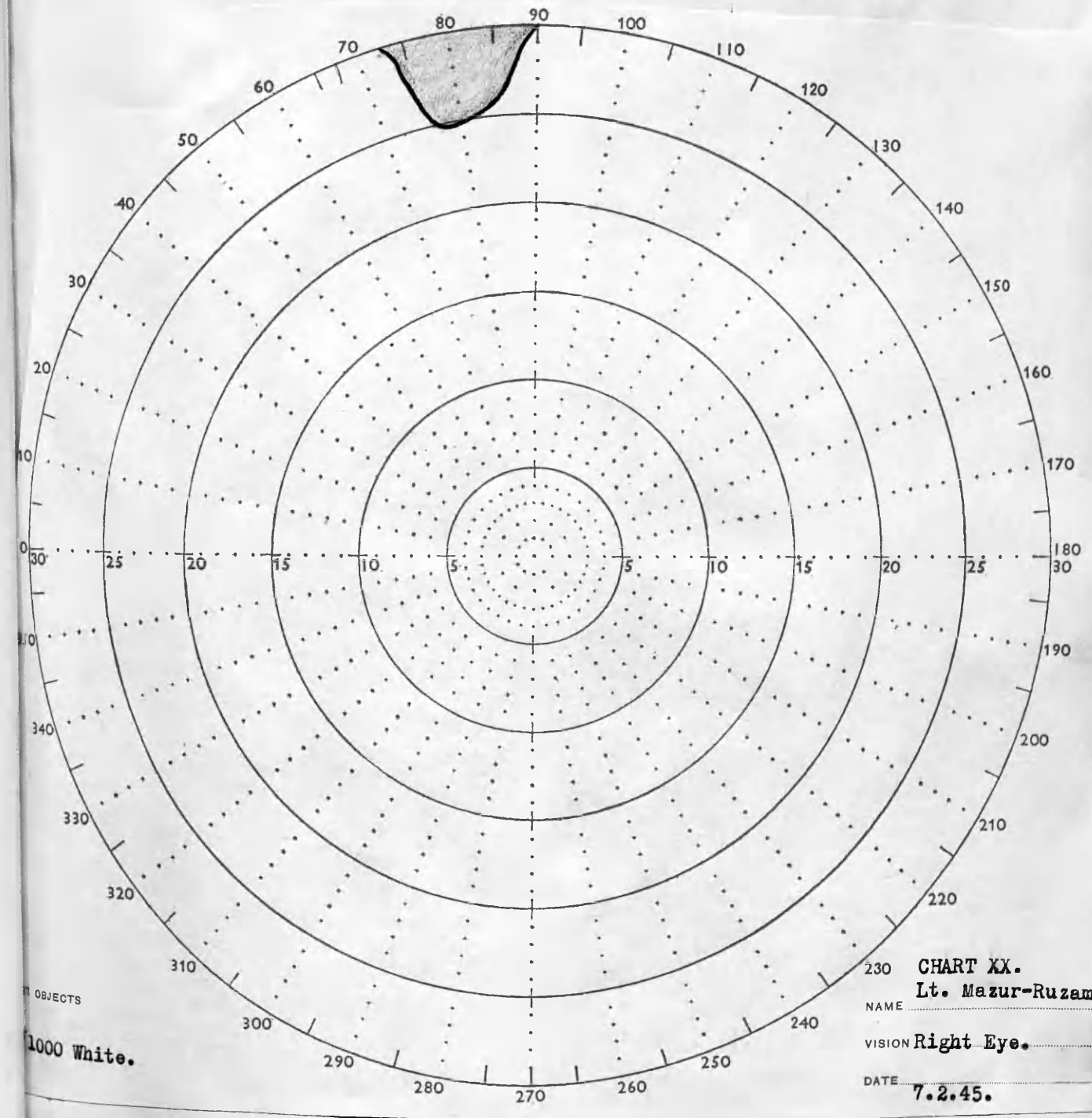
NAME Lt. Mazur-Ruzam

VISION Right Eye.

DATE 18.1.45.

OBJECTS

/1000 White.



OBJECTS

1000 White.

CHART XX.

NAME Lt. Mazur-Ruzam

VISION Right Eye.

DATE 7.2.45.

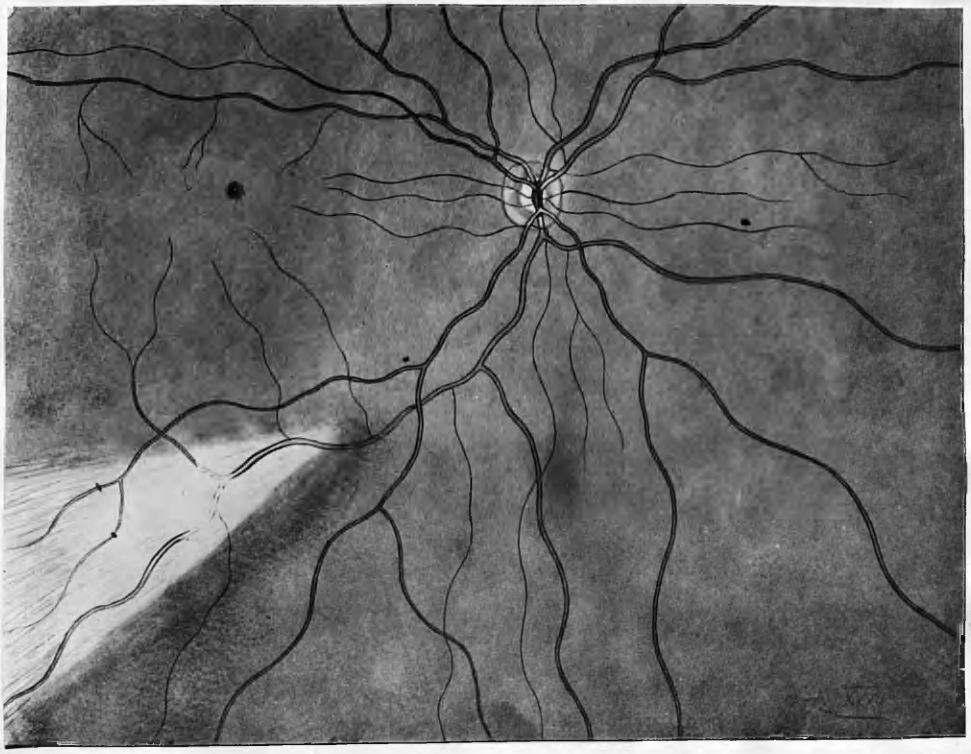
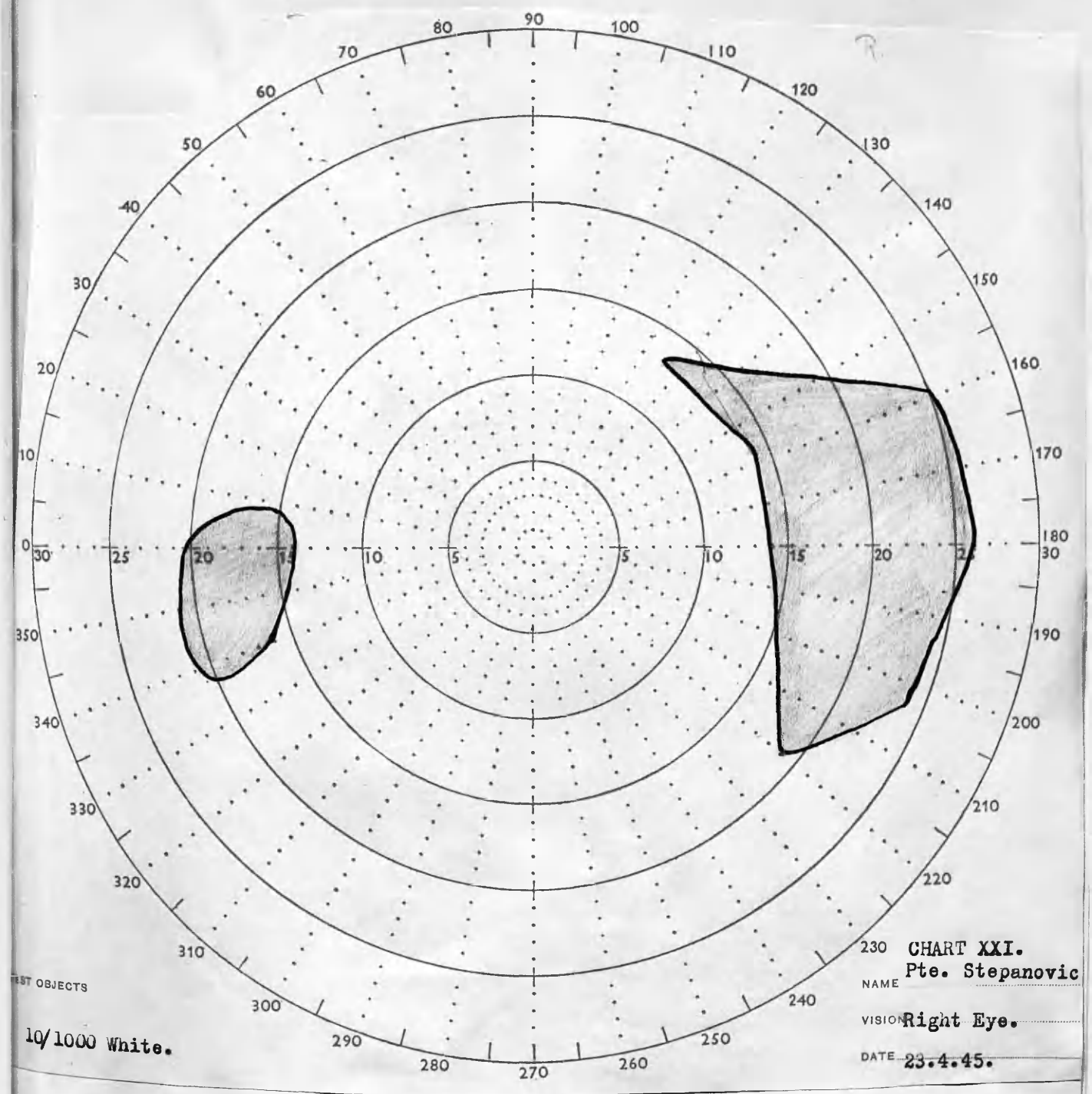


Fig. XXXI.



TEST OBJECTS

10/1000 White.

CHART XXI.

NAME Pte. Stepanovic

VISION Right Eye.

DATE 23.4.45.

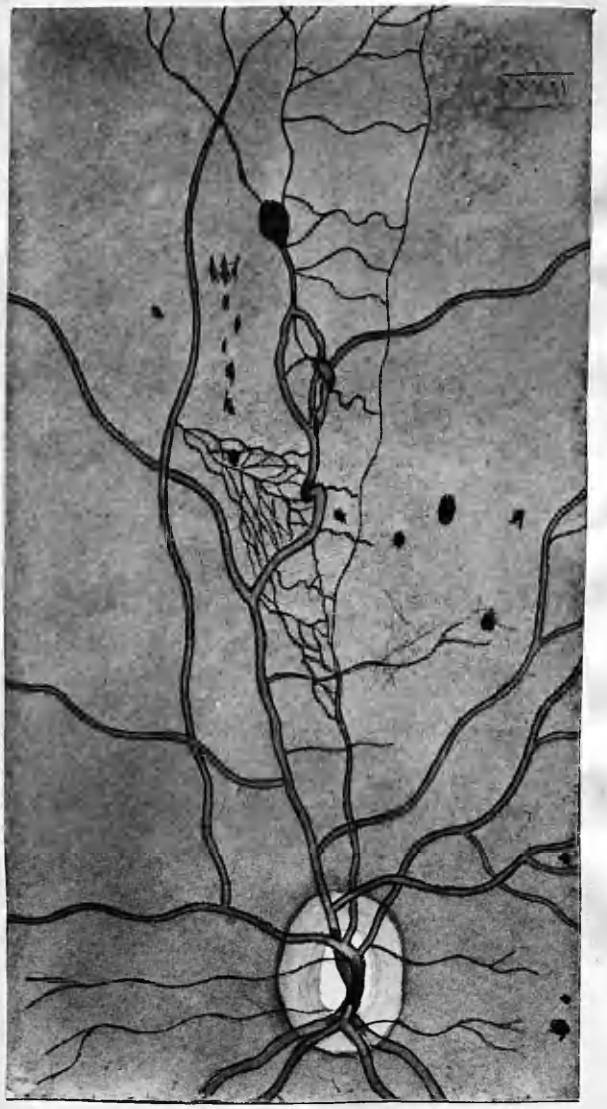


Fig. XXXII.

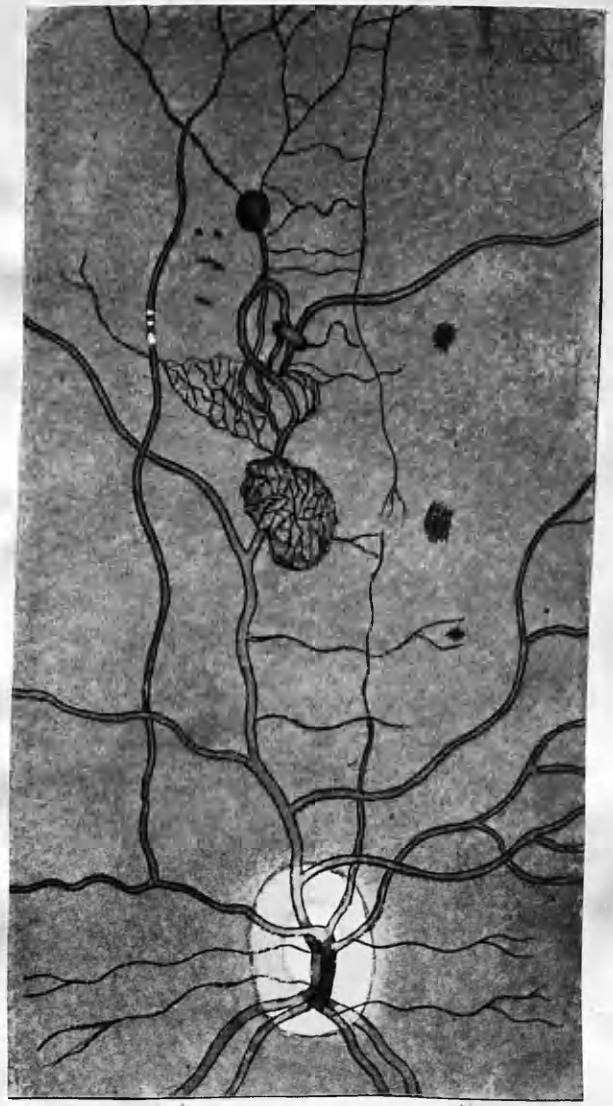
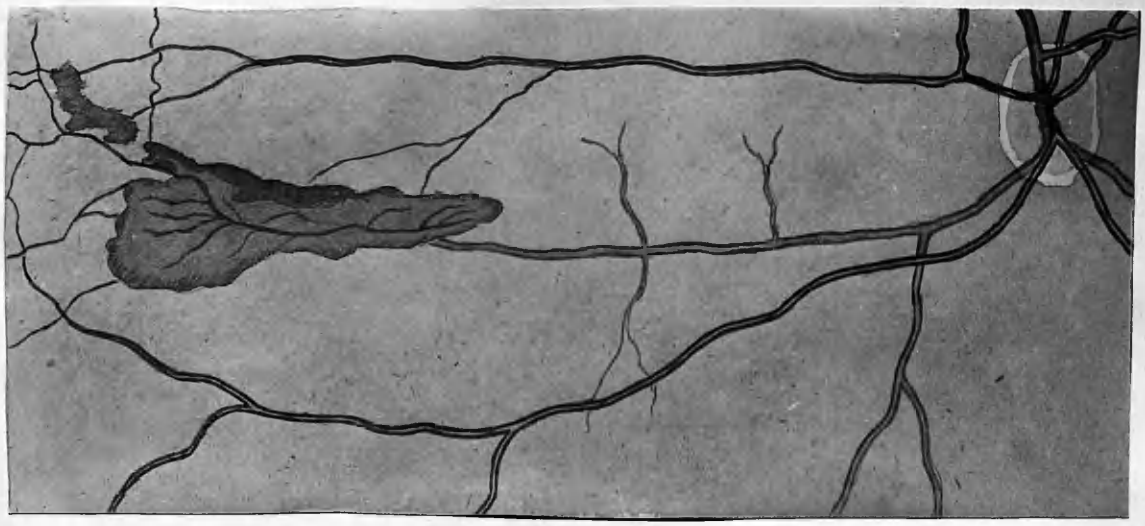


Fig. XXXIII.

Fig. XXXIV.

Fig. XXXV.



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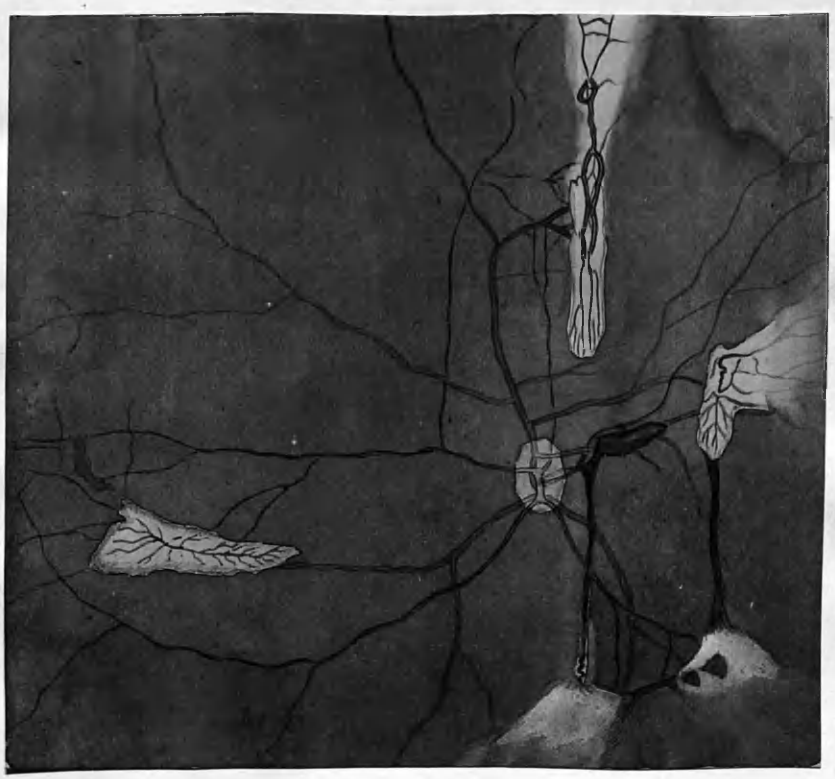

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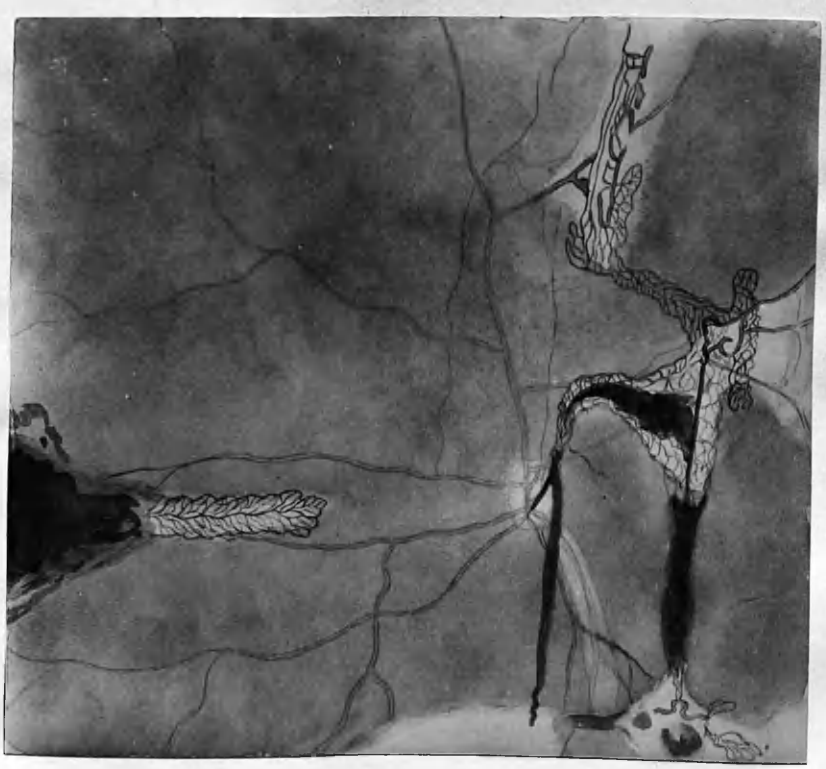

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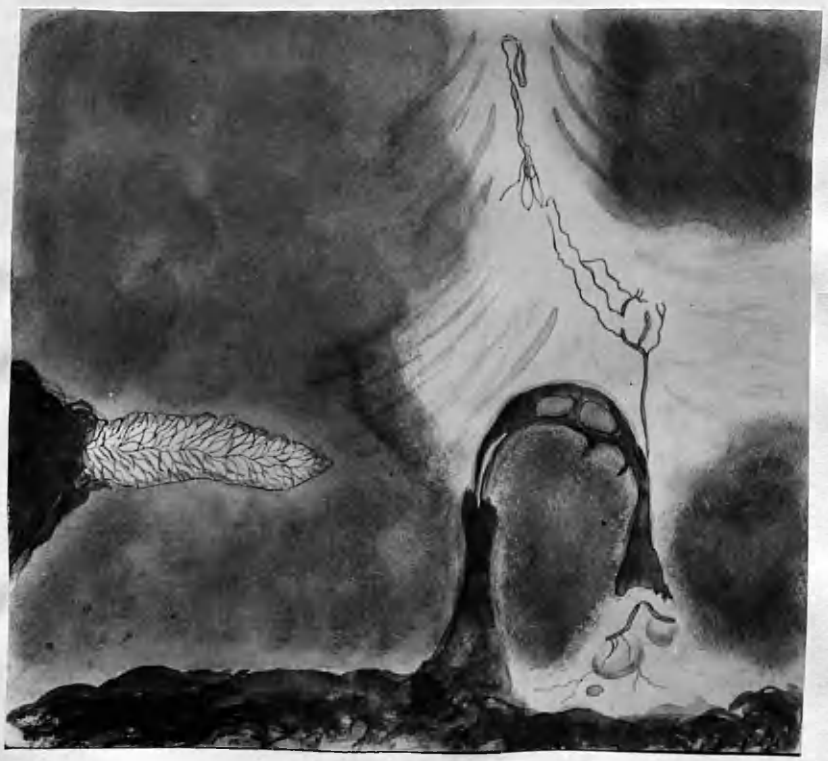

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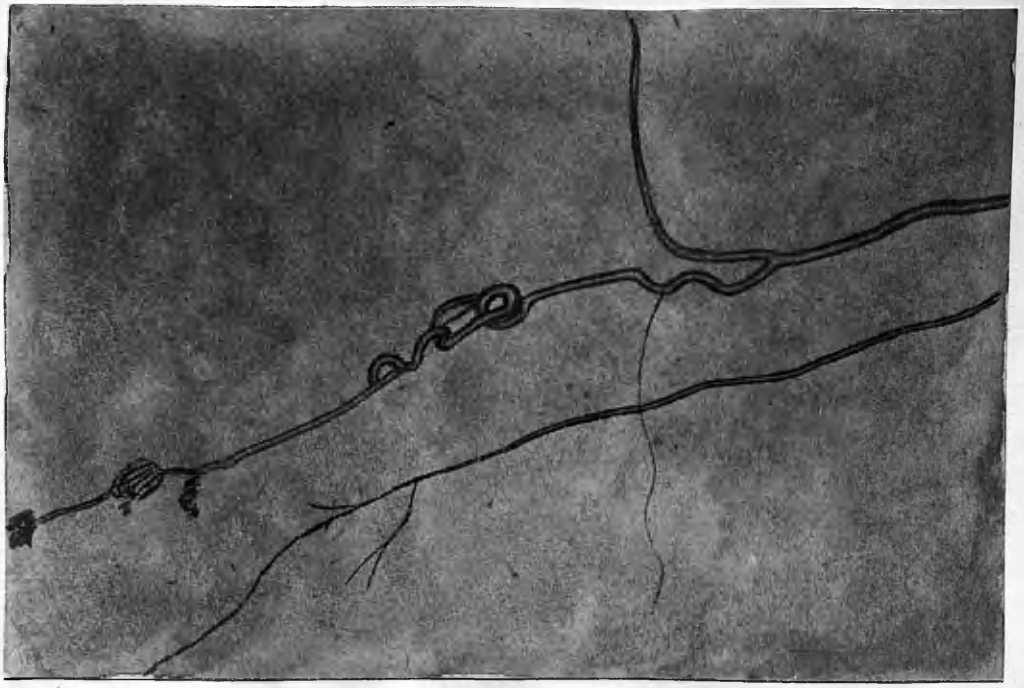
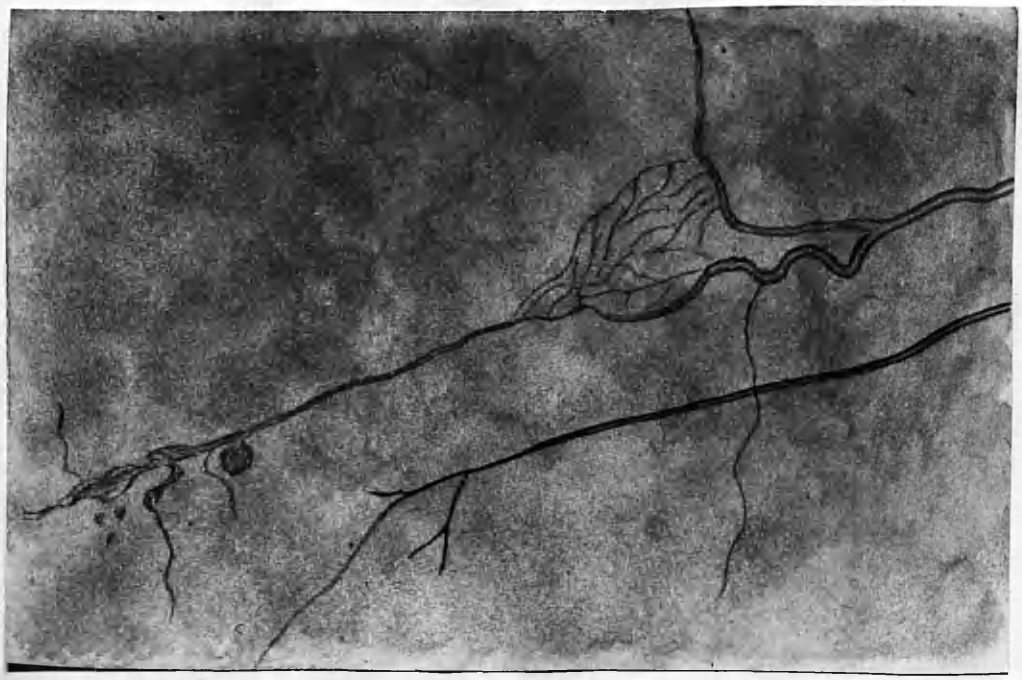


Fig. XL.



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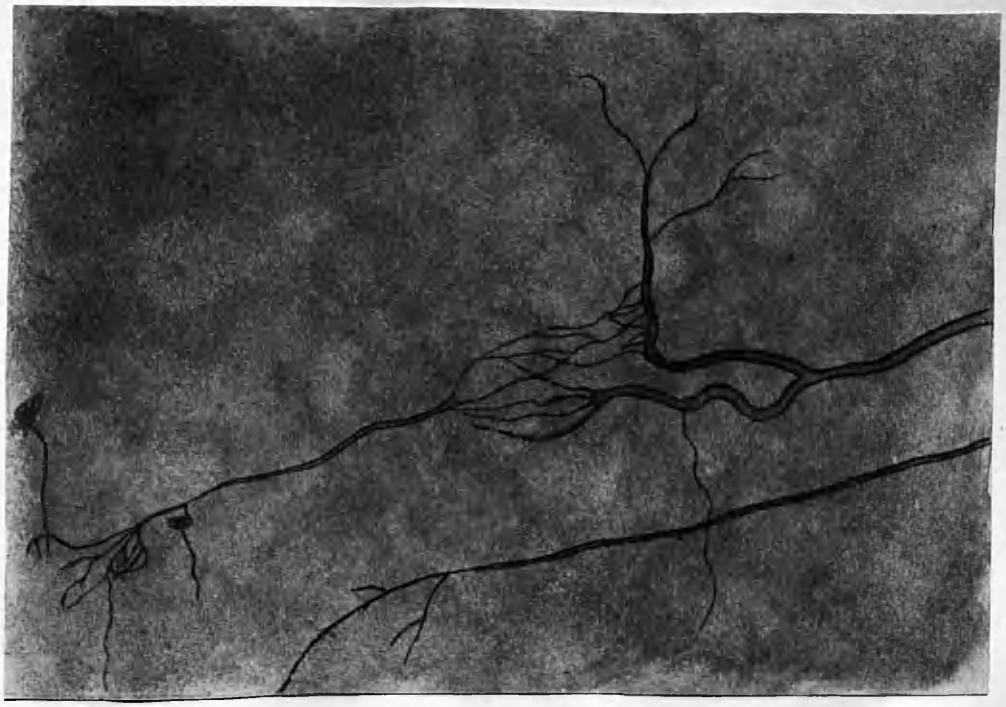

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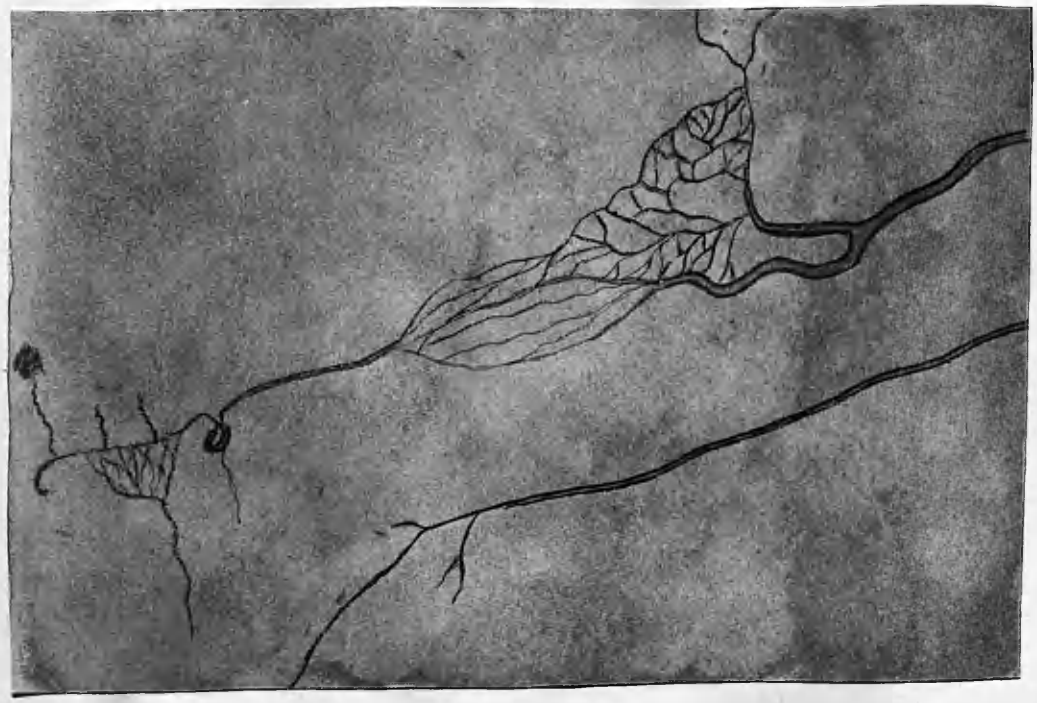

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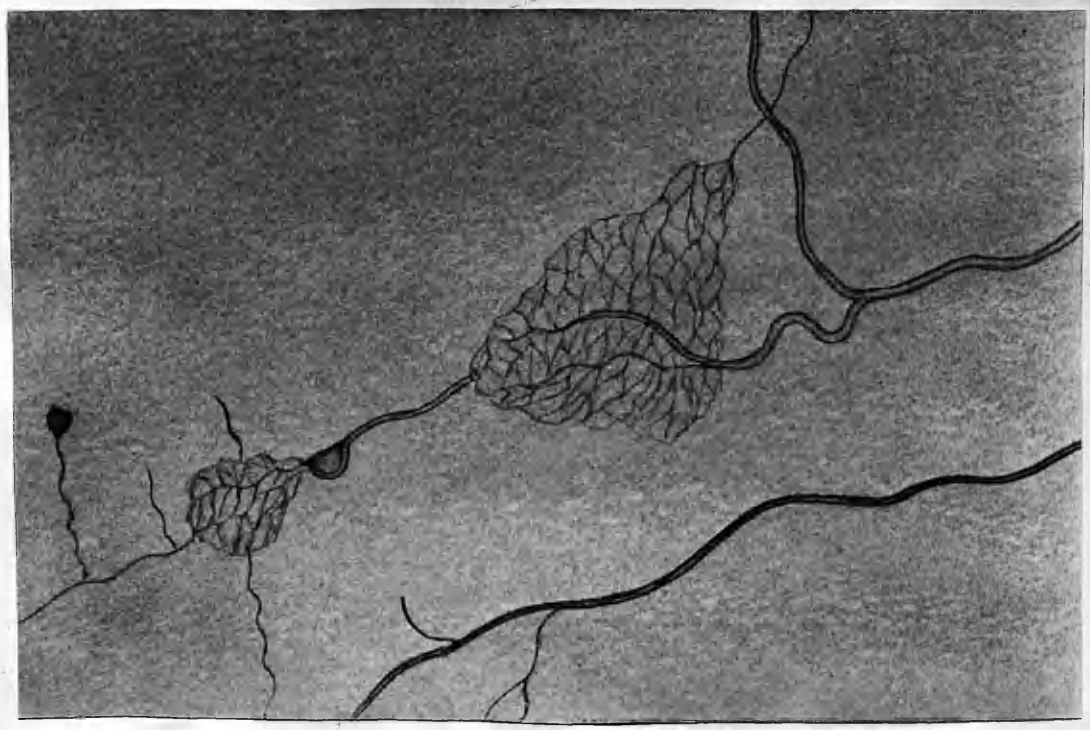
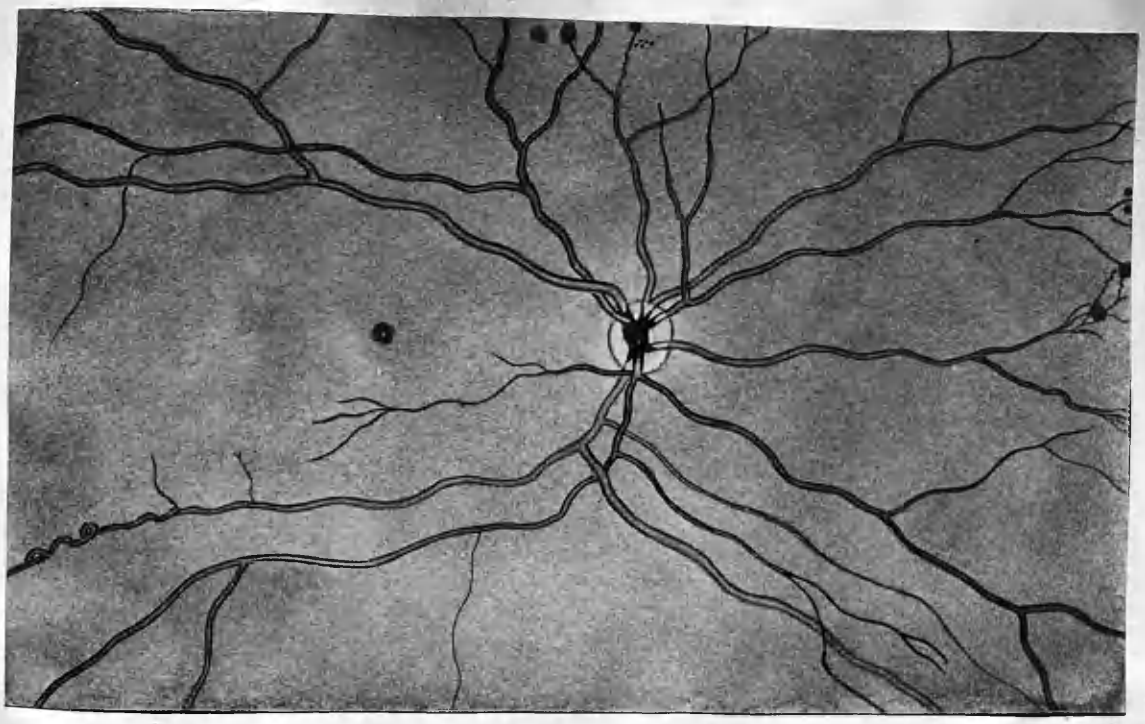


Fig. XLIV.

Fig. XLV.



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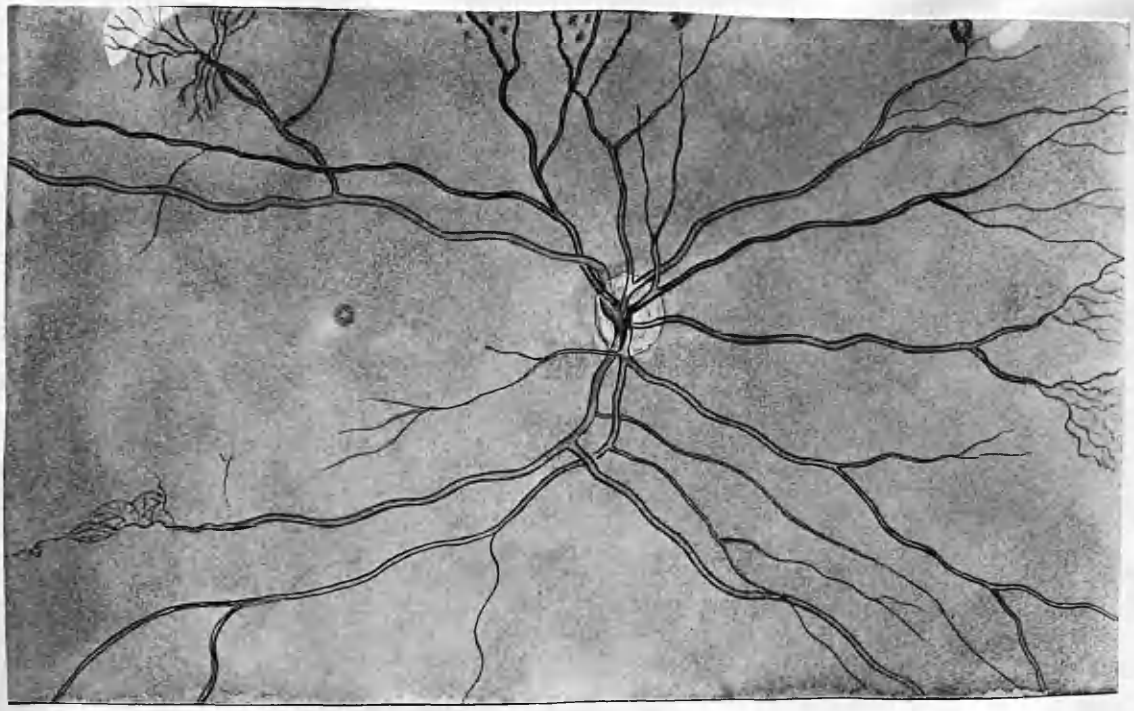

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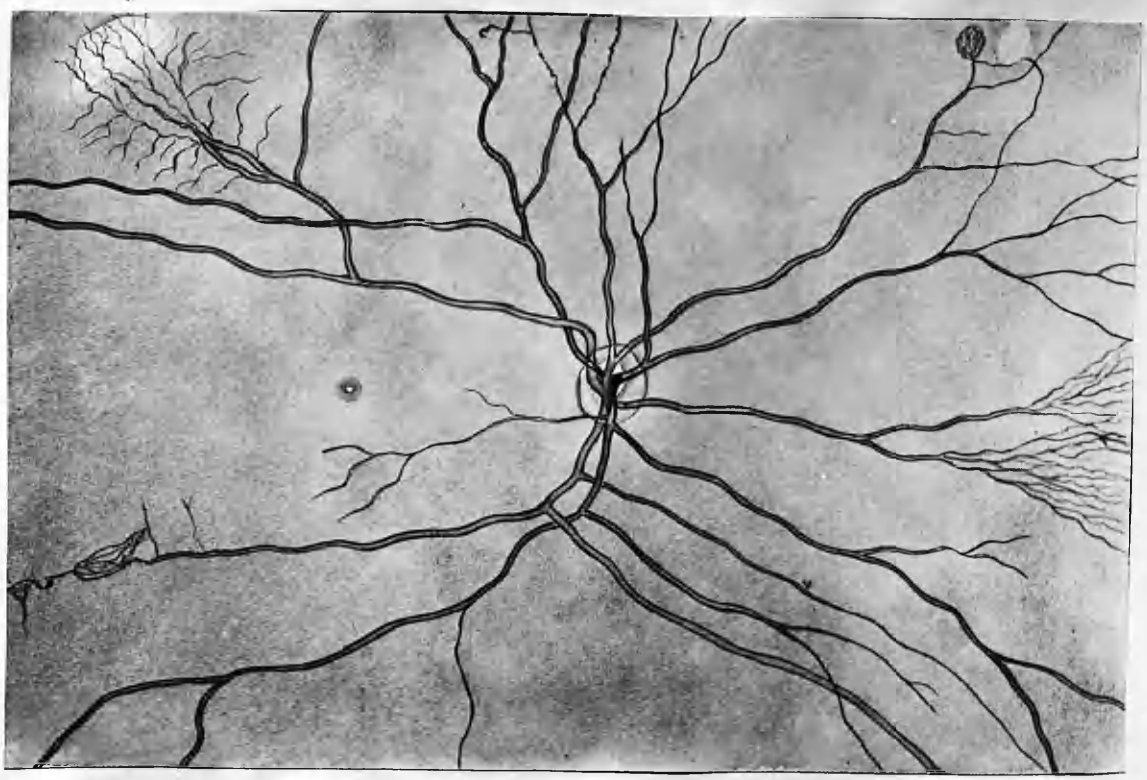

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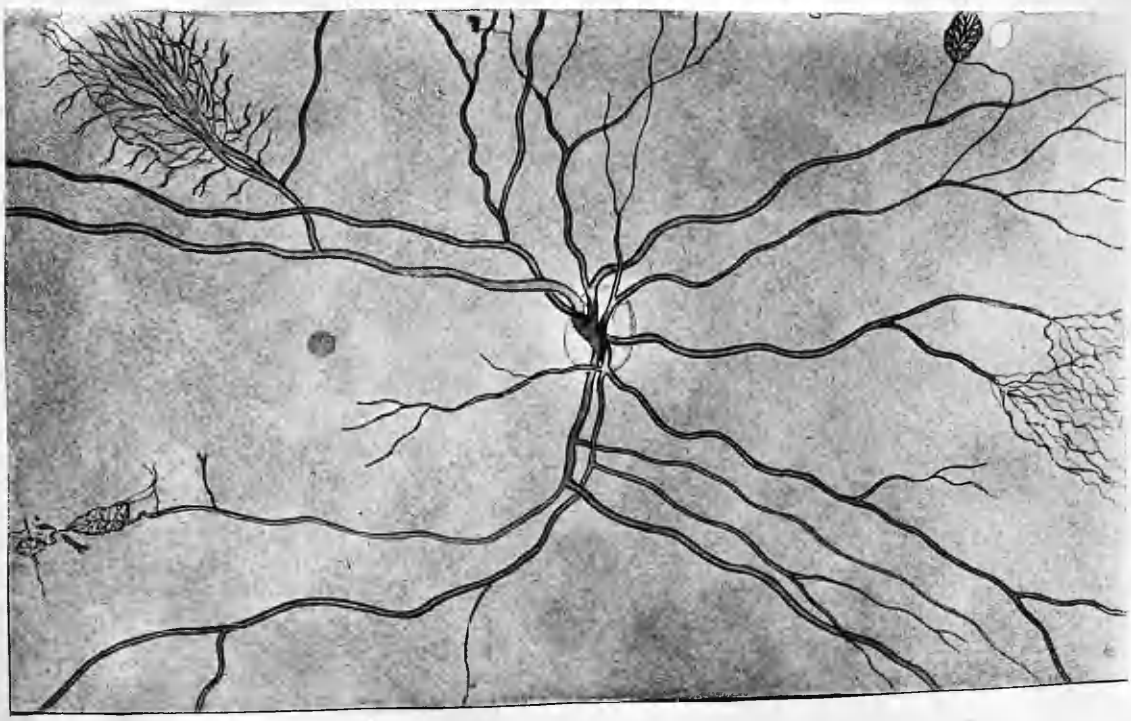

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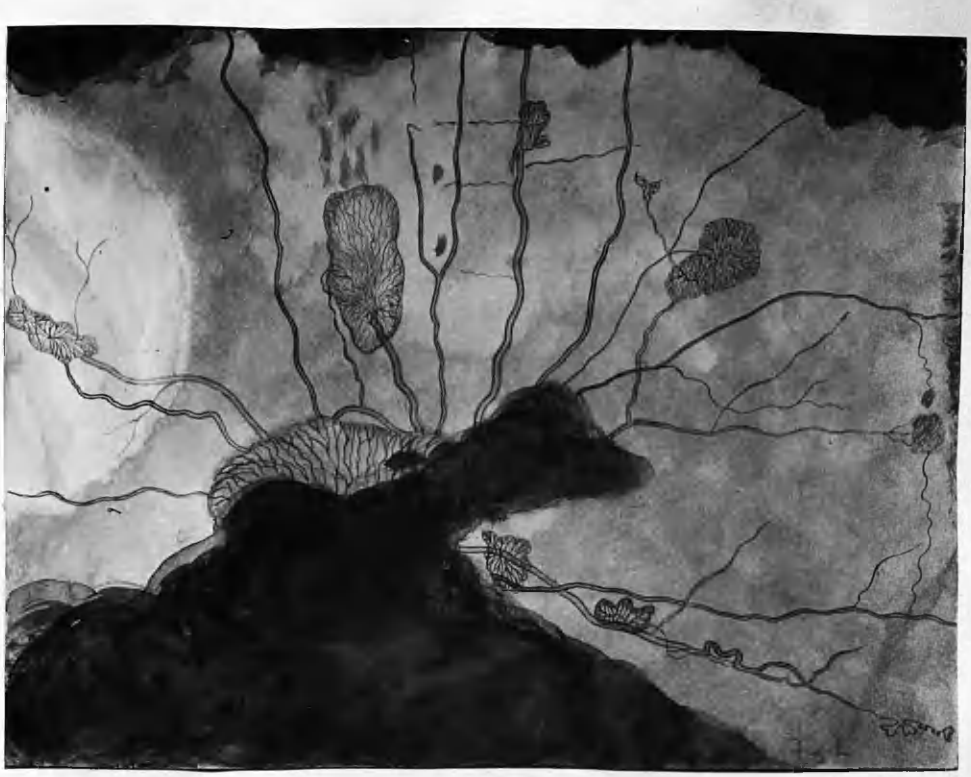

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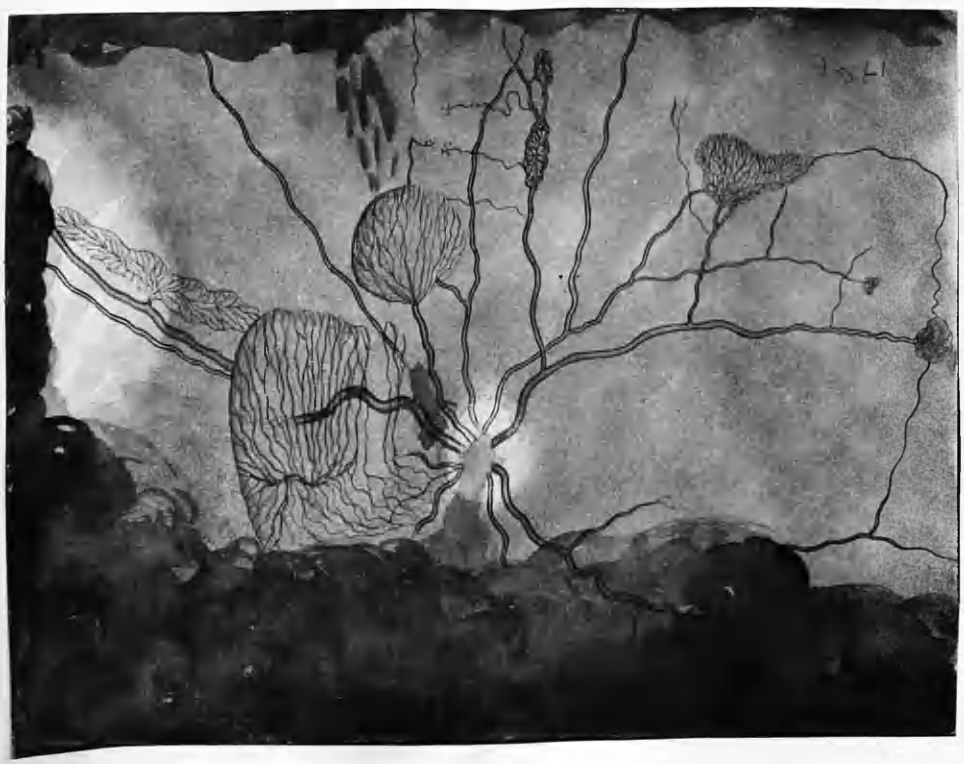

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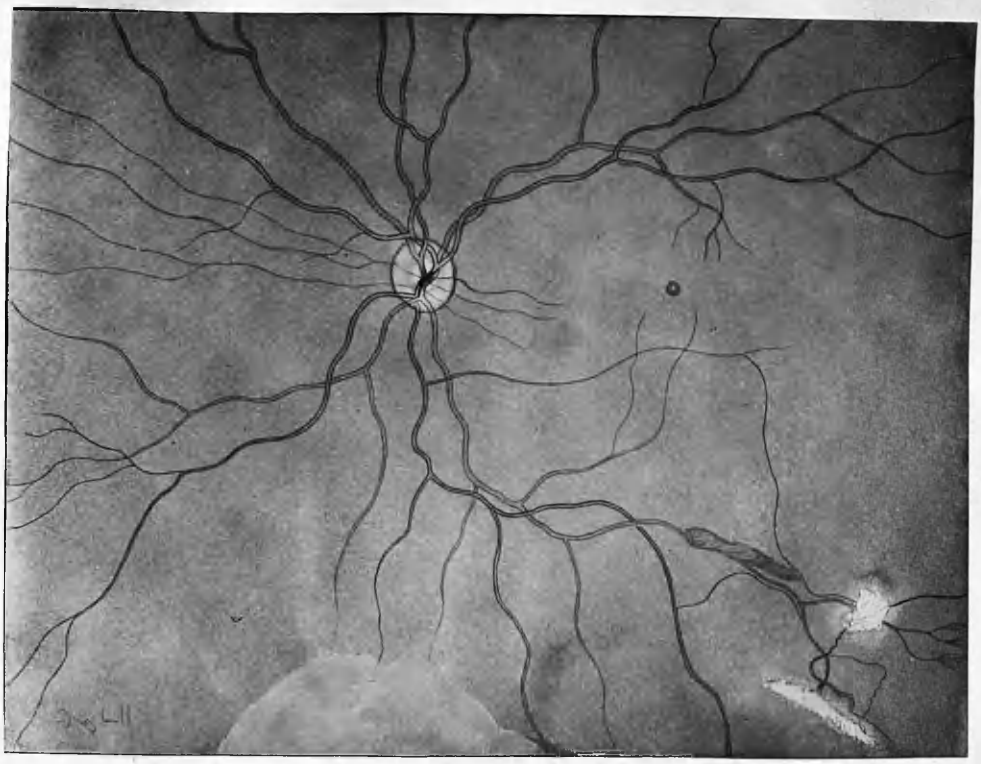

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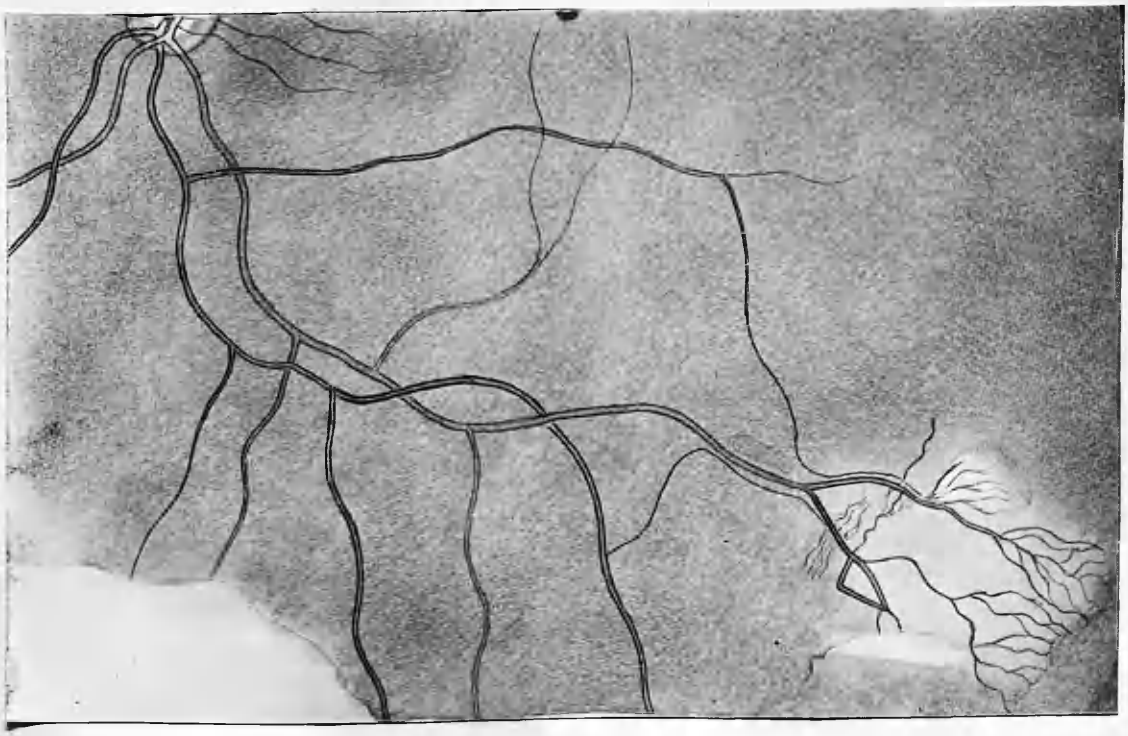

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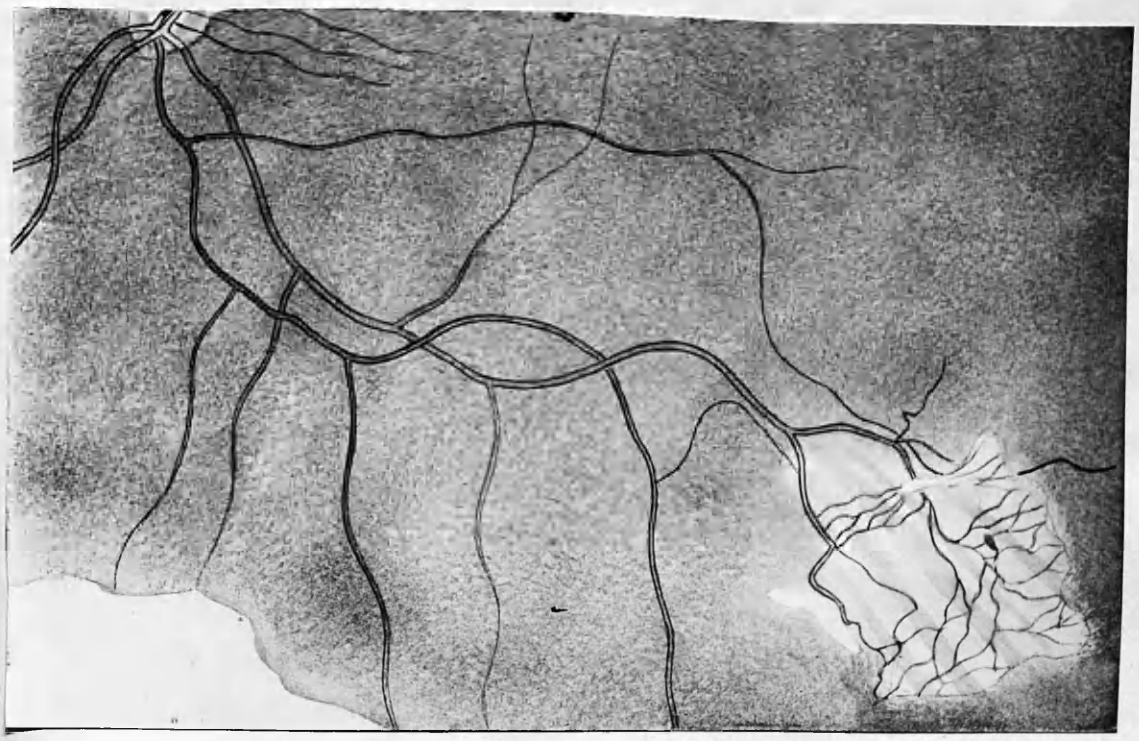

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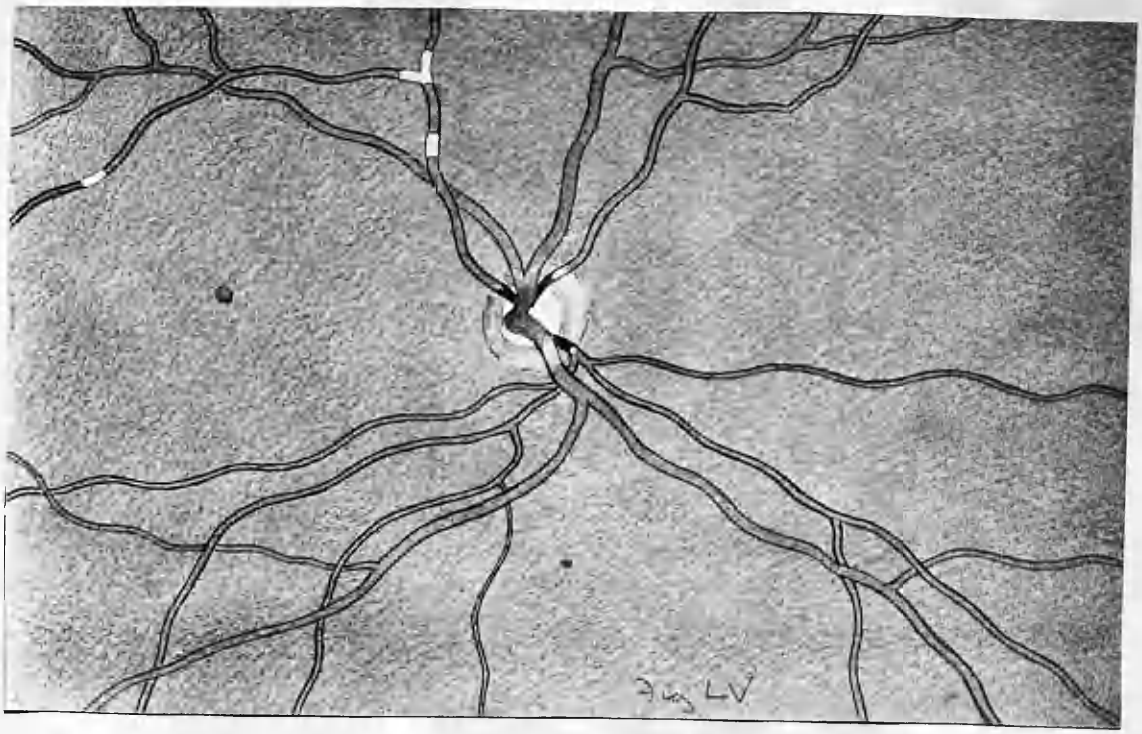

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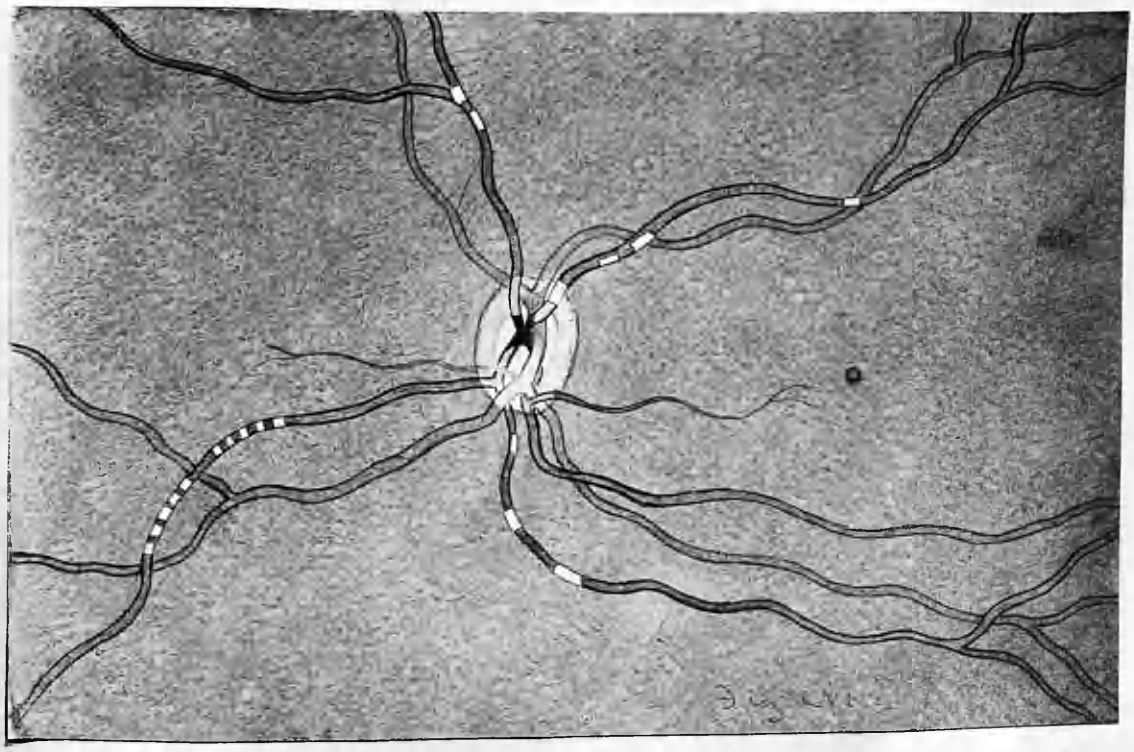

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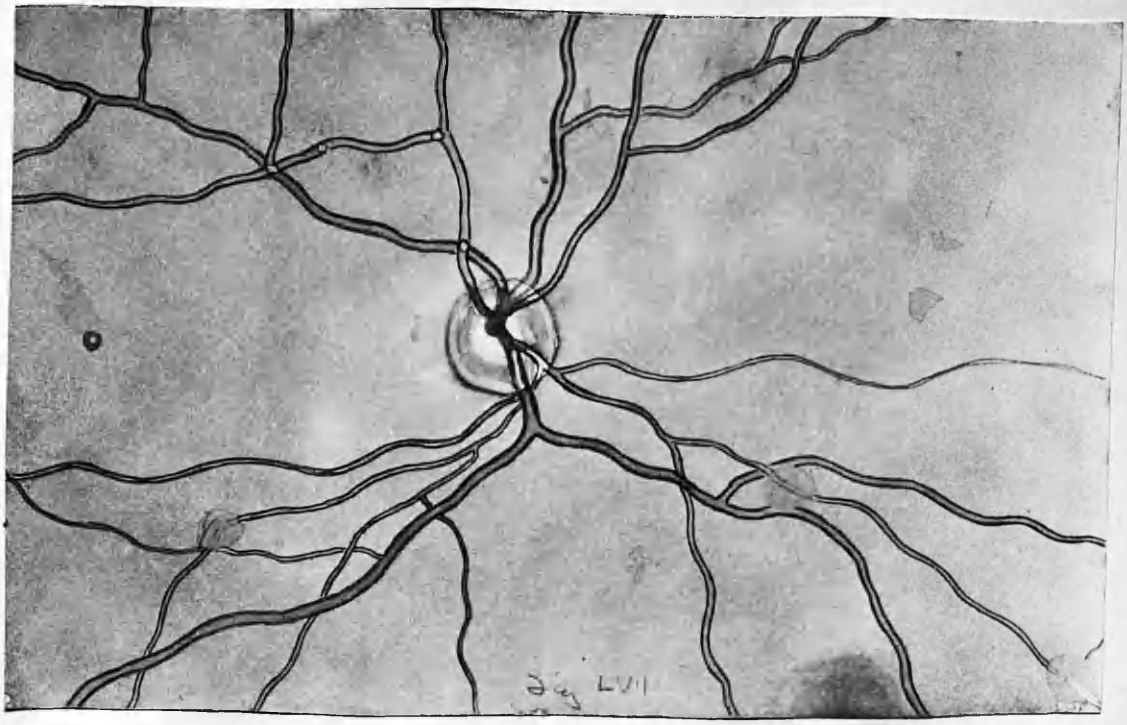

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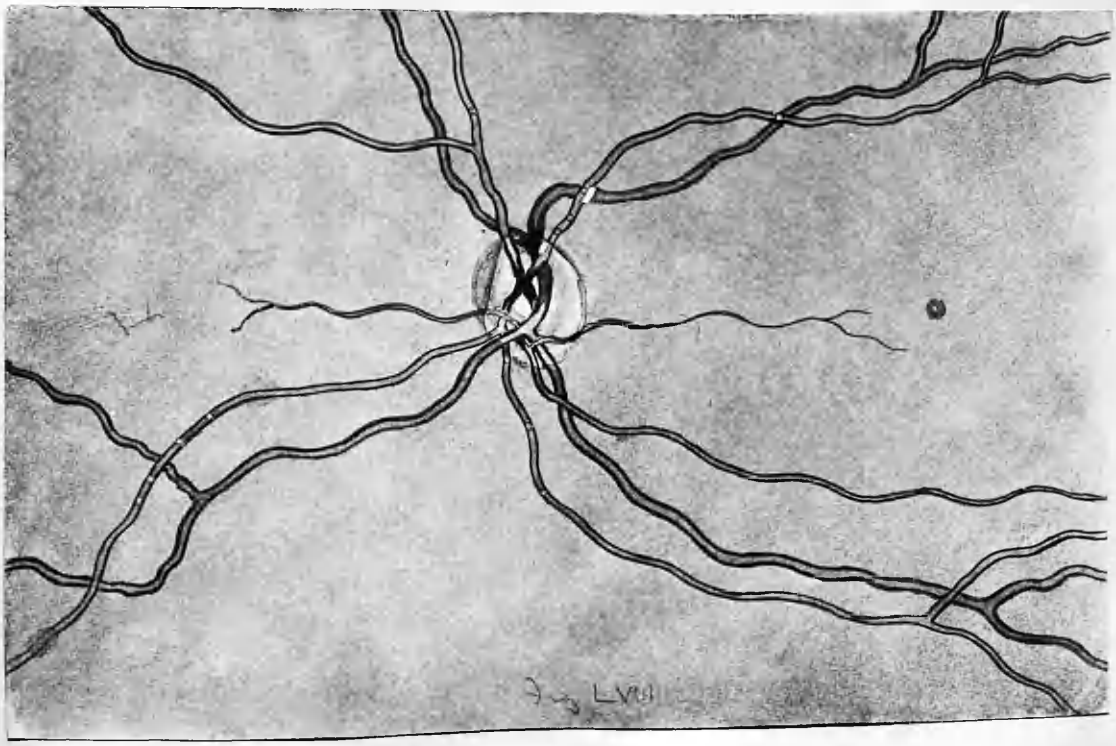

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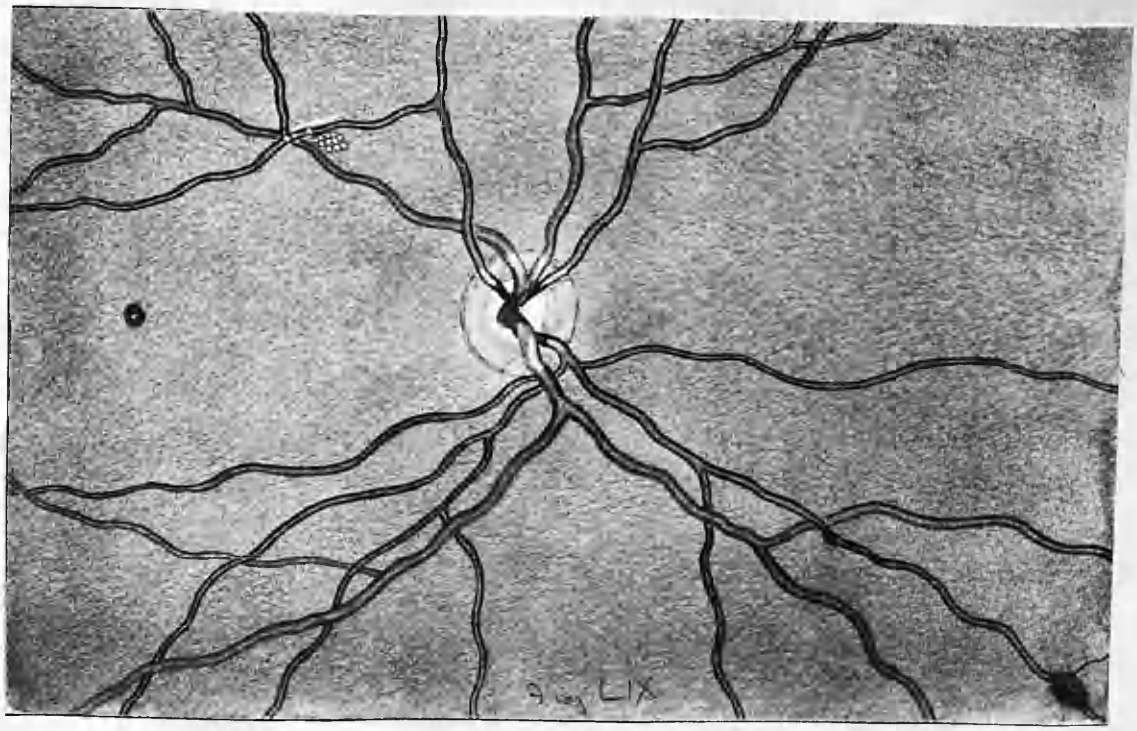

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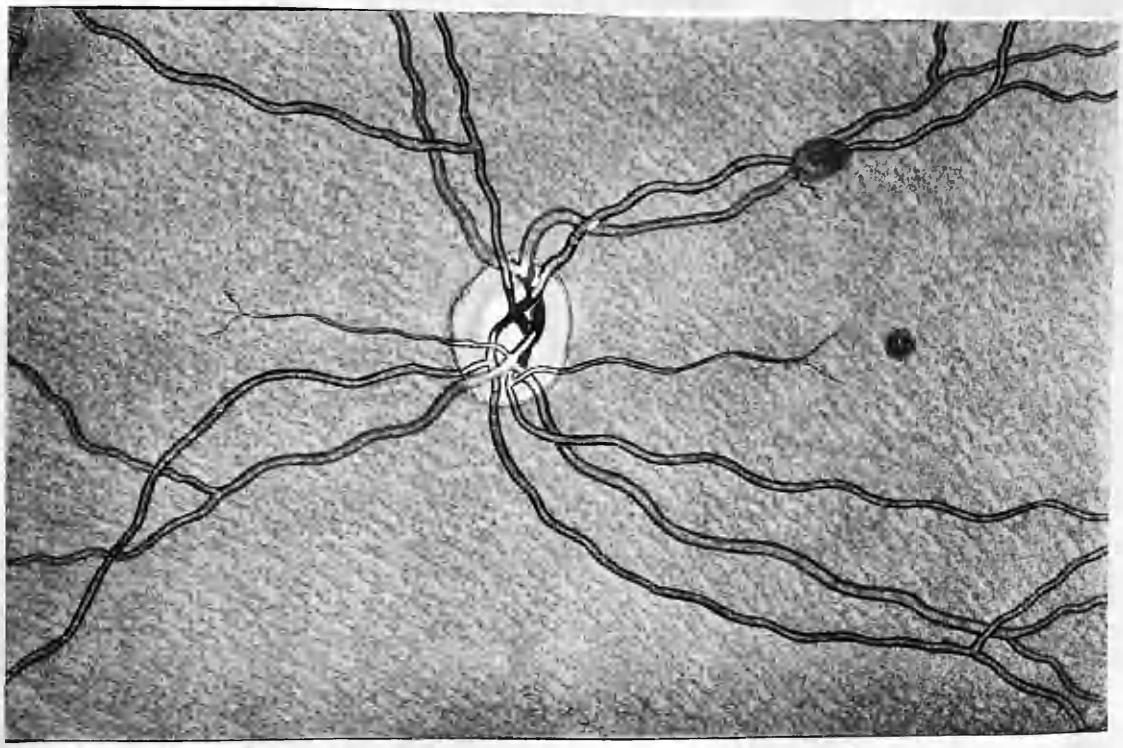

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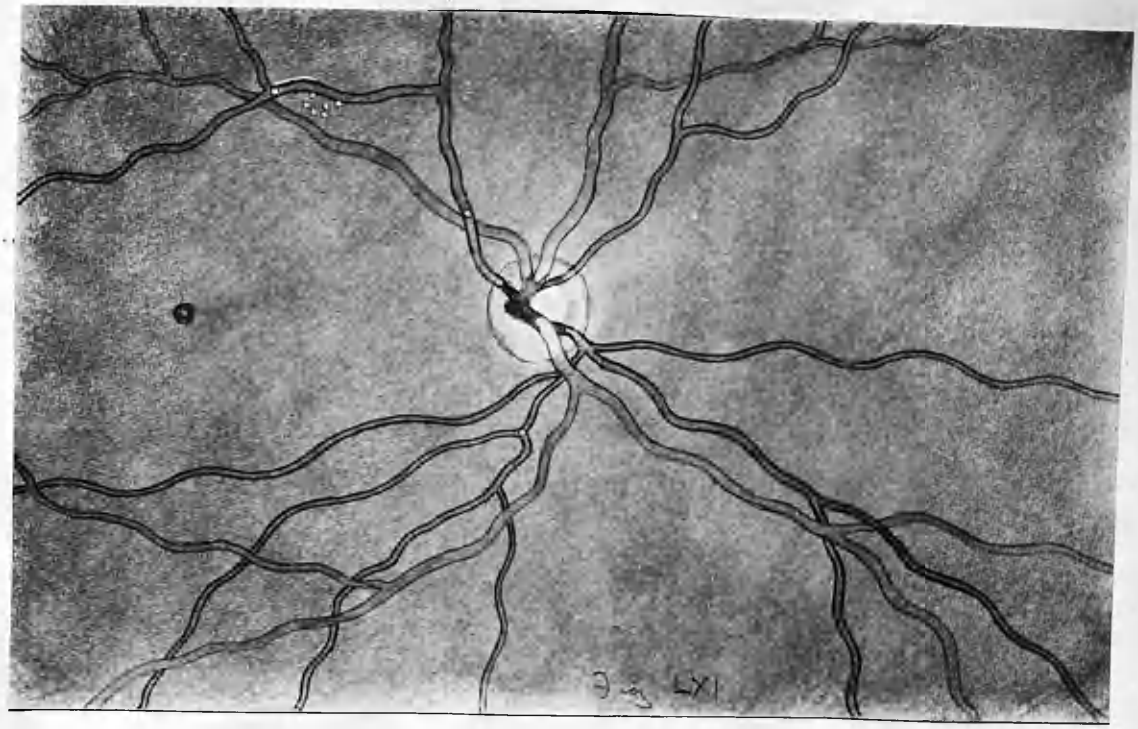

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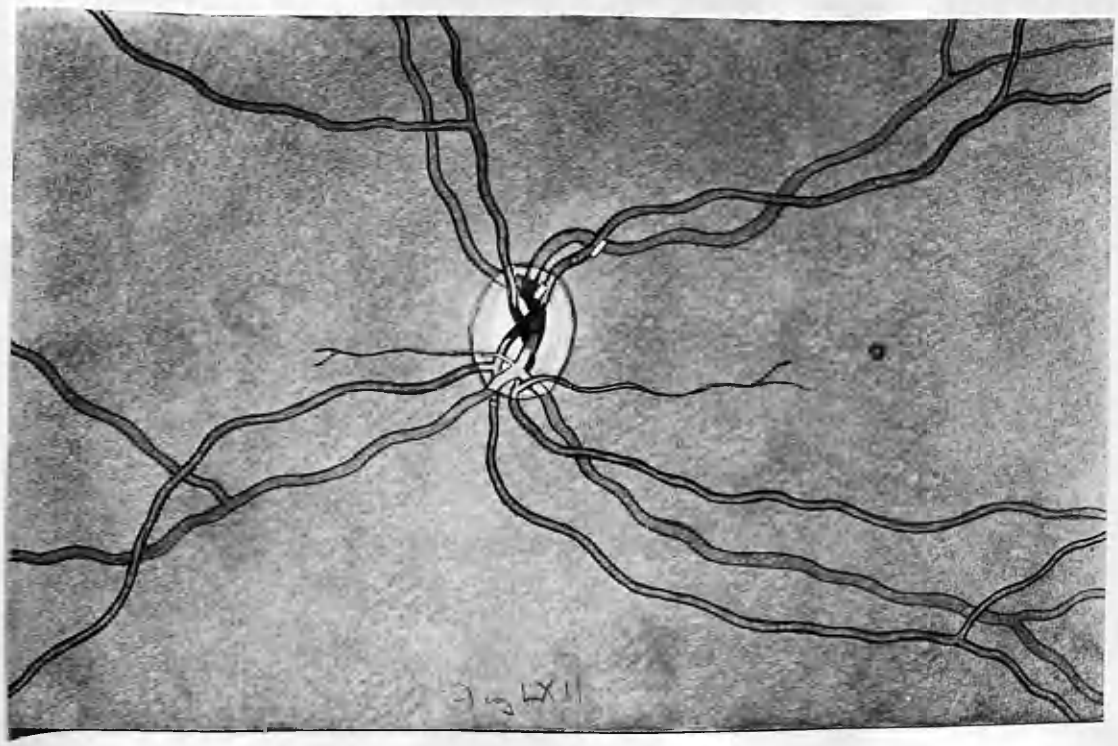

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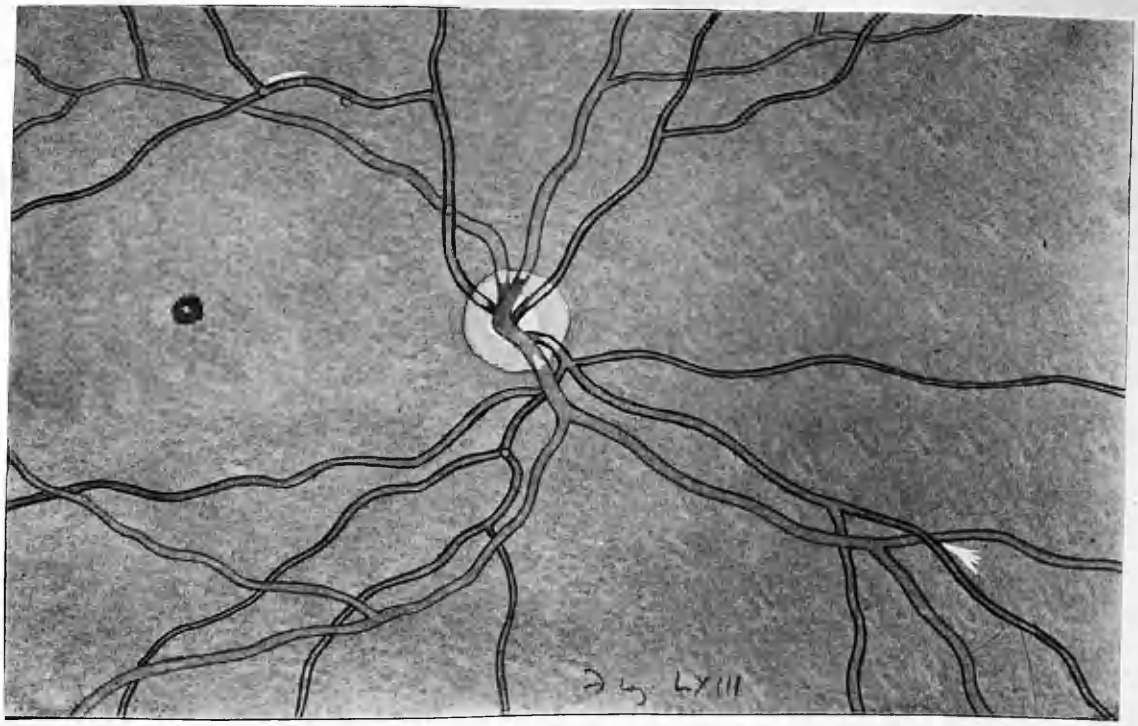

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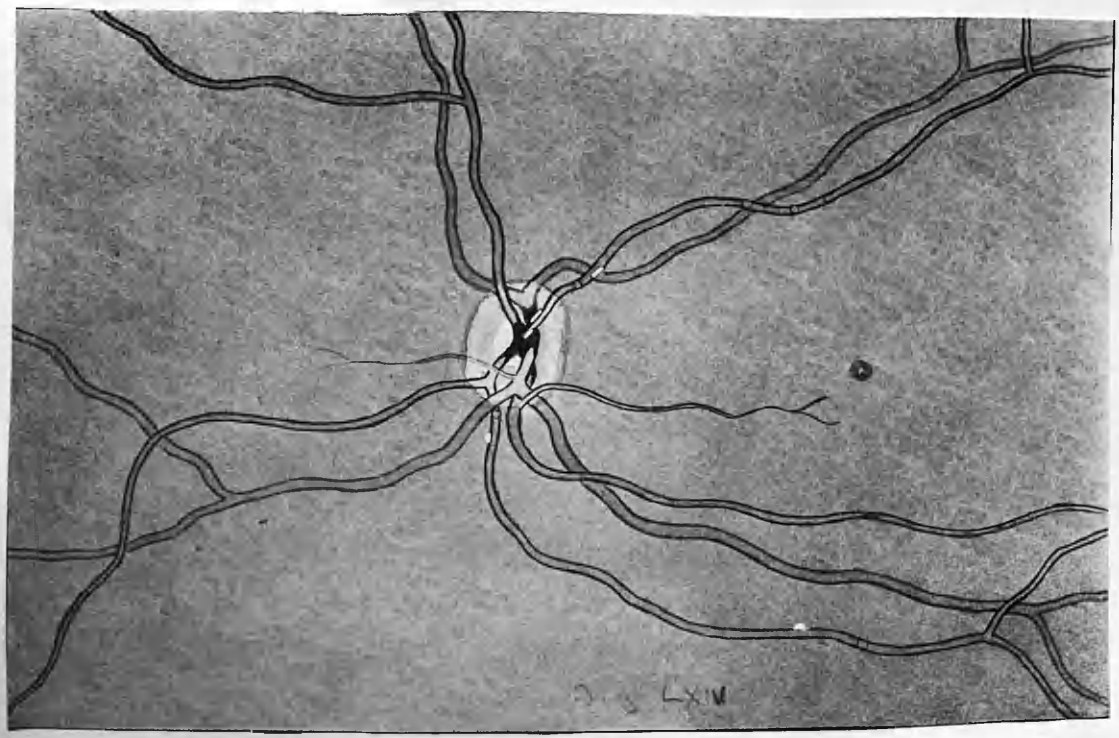

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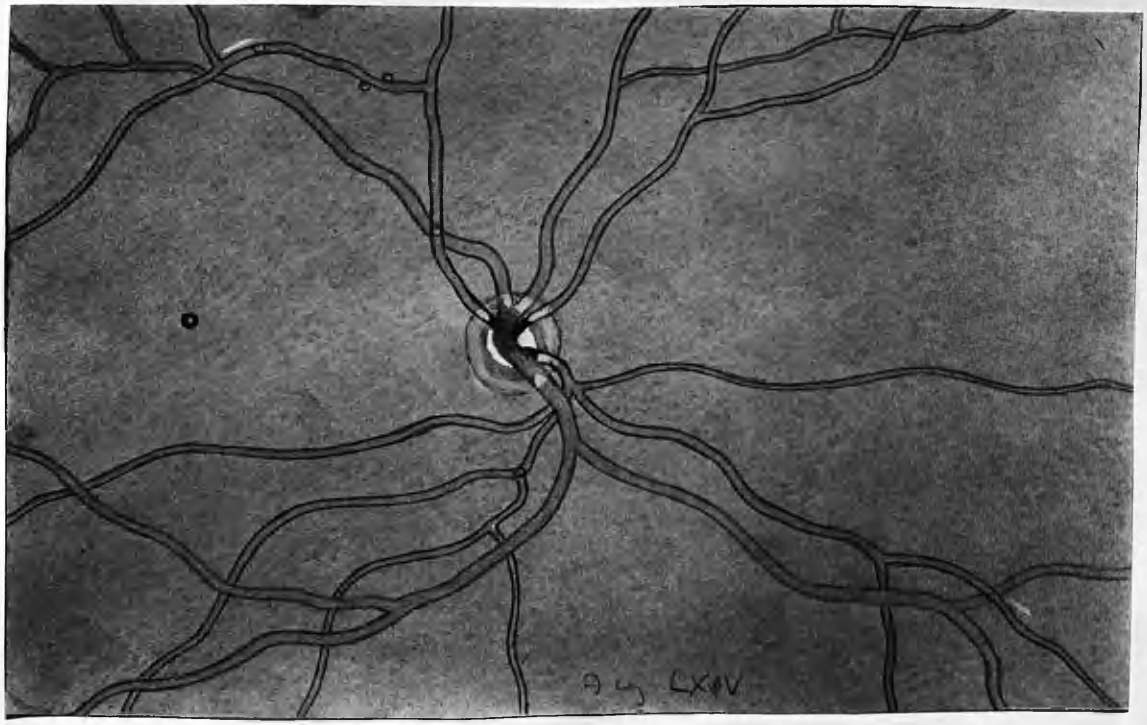

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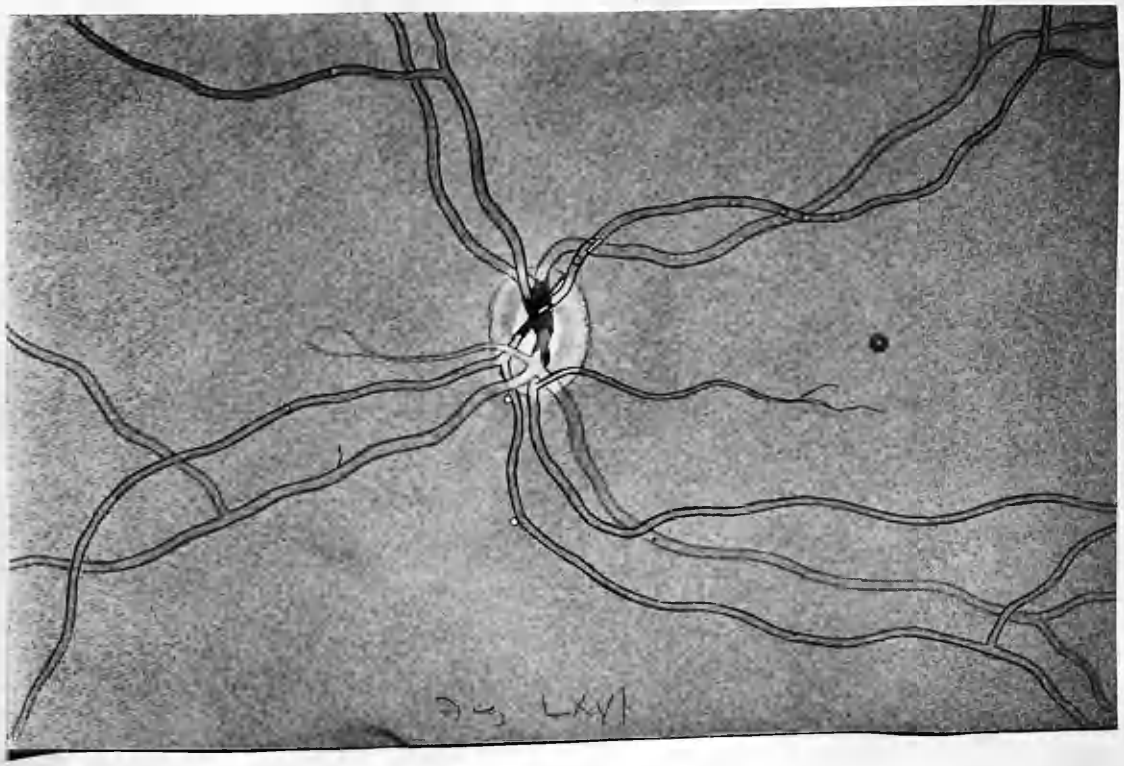

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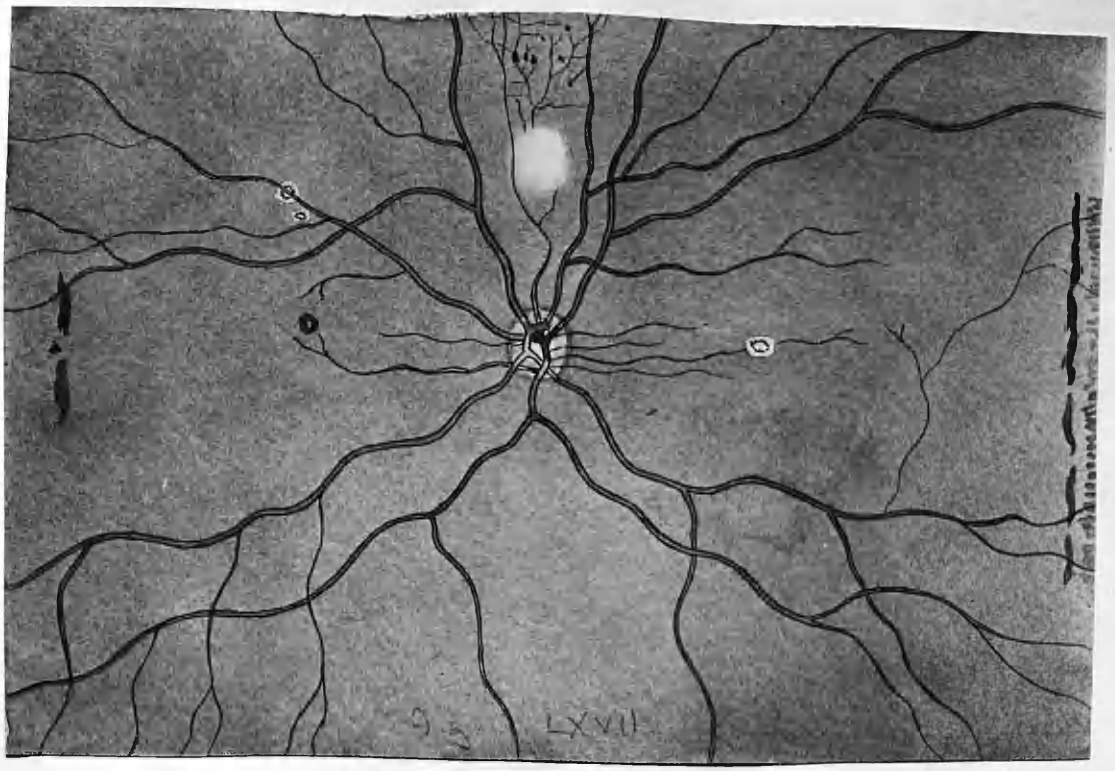

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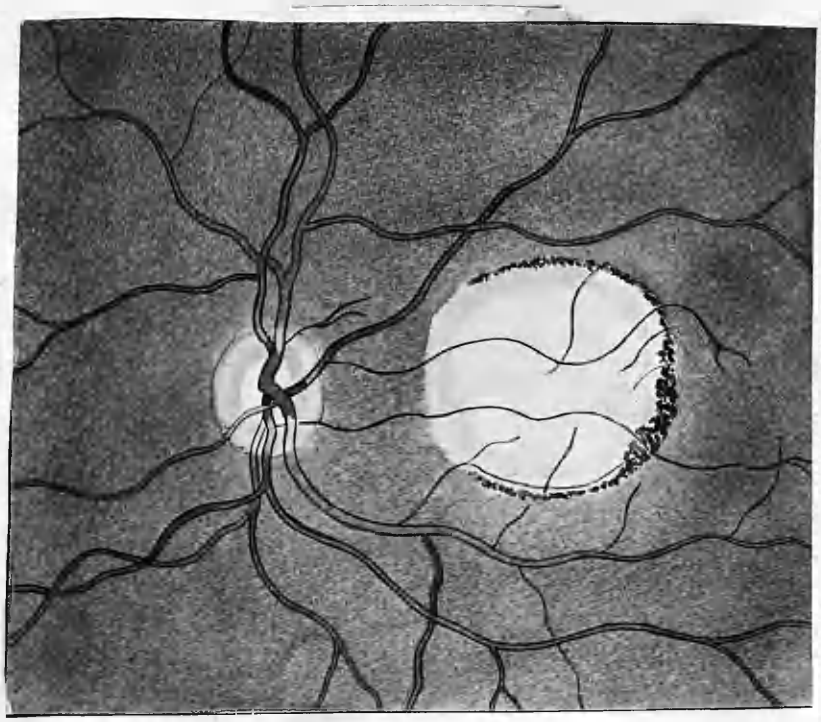

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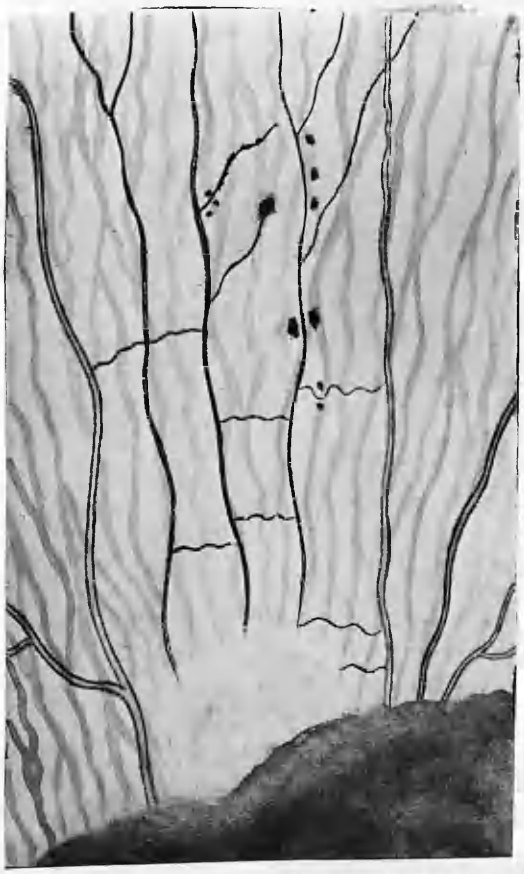


Fig. LXIX.

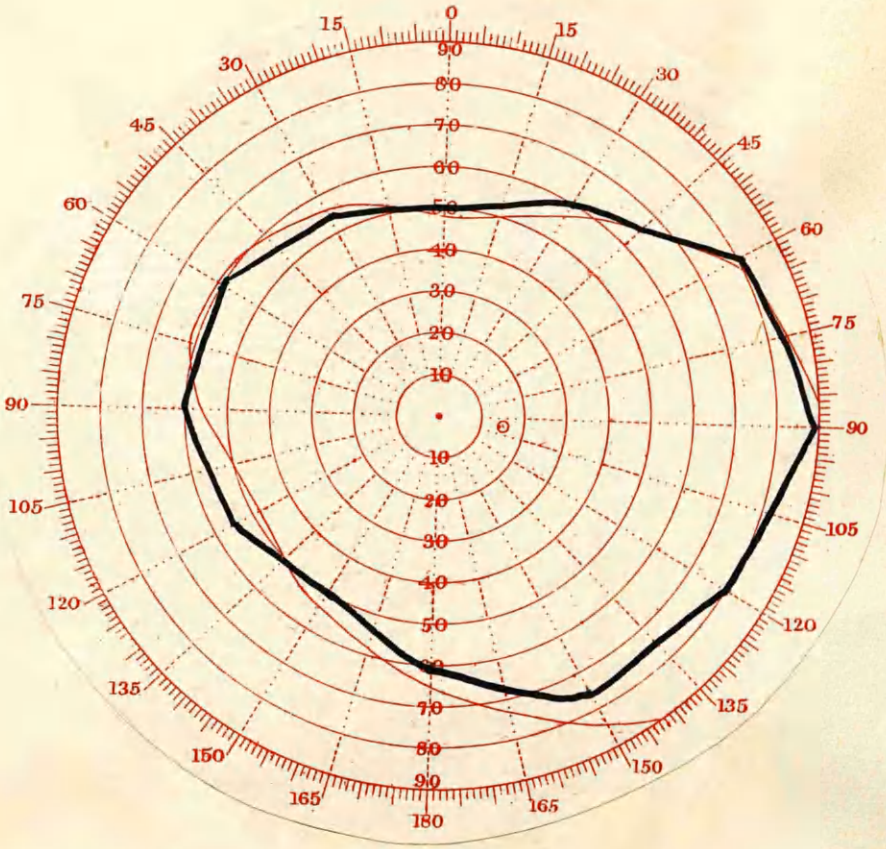
Fig. LXX.

Fig. LXXI.

Fig. LXXII.

Fig. LXXIII.

RIGHT.



LEFT

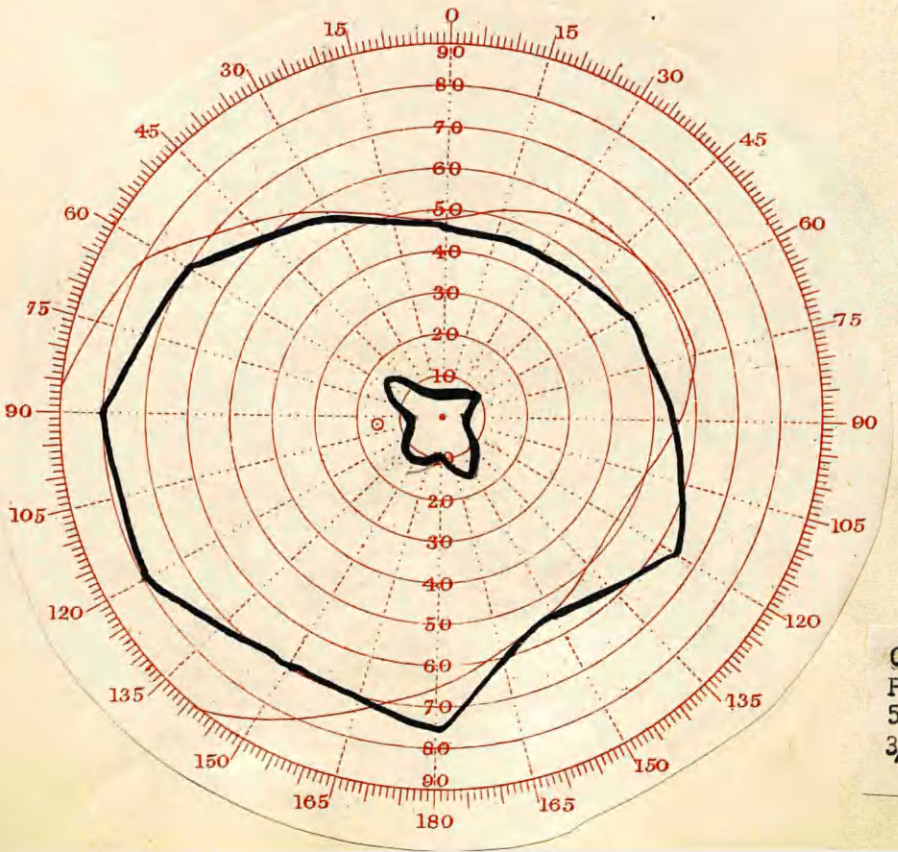


CHART XXII.
Pte. Stringer.
5.4.44.
3/330 White.

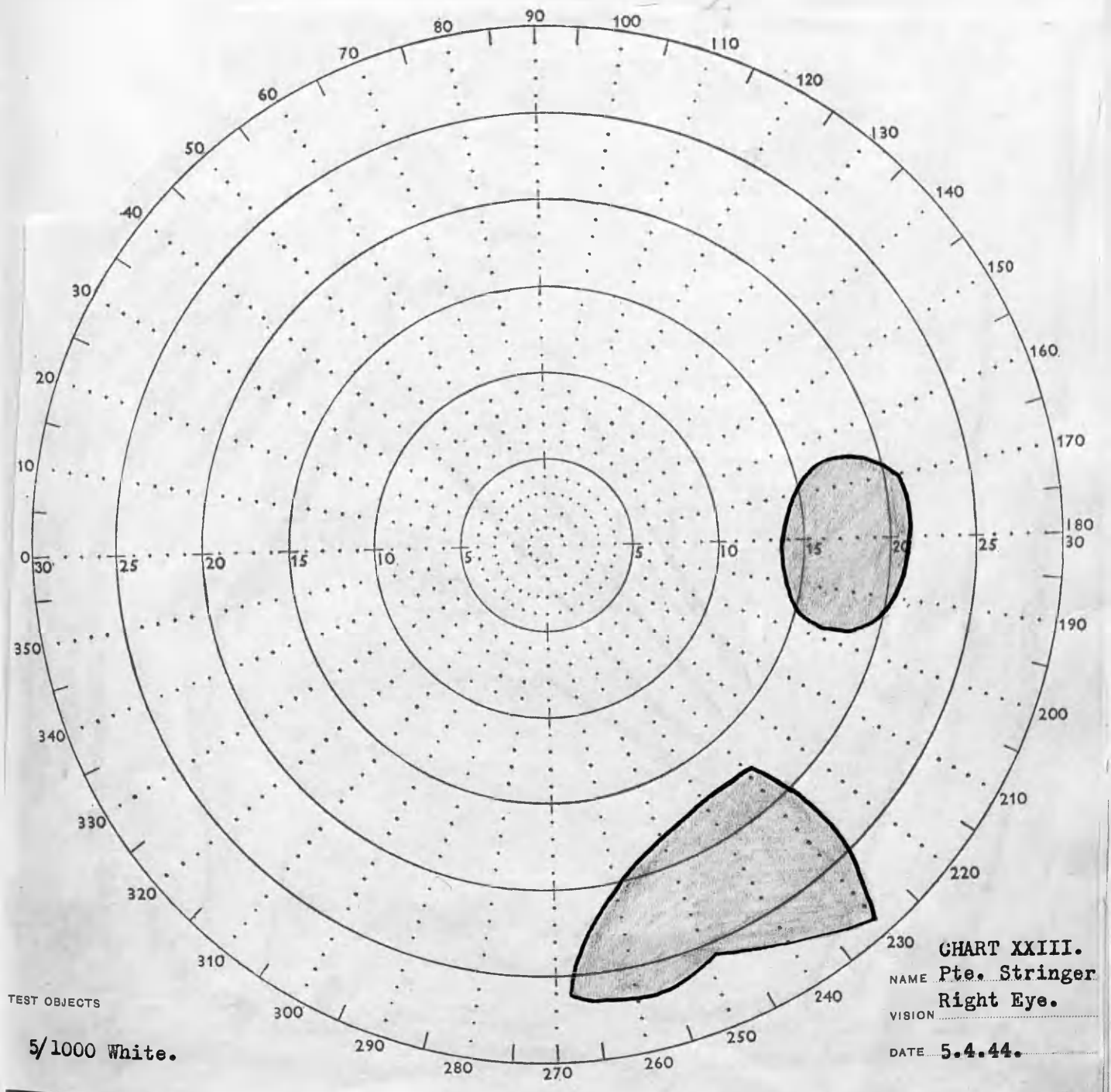


CHART XXIII.

NAME Pte. Stringer

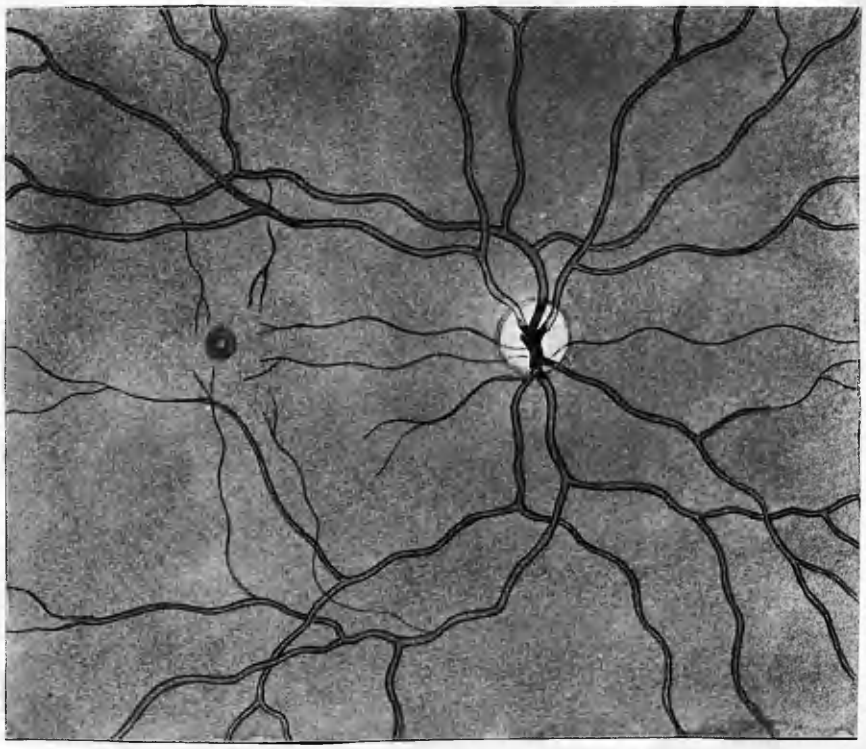
Right Eye.

VISION

DATE 5.4.44.

TEST OBJECTS

5/1000 White.



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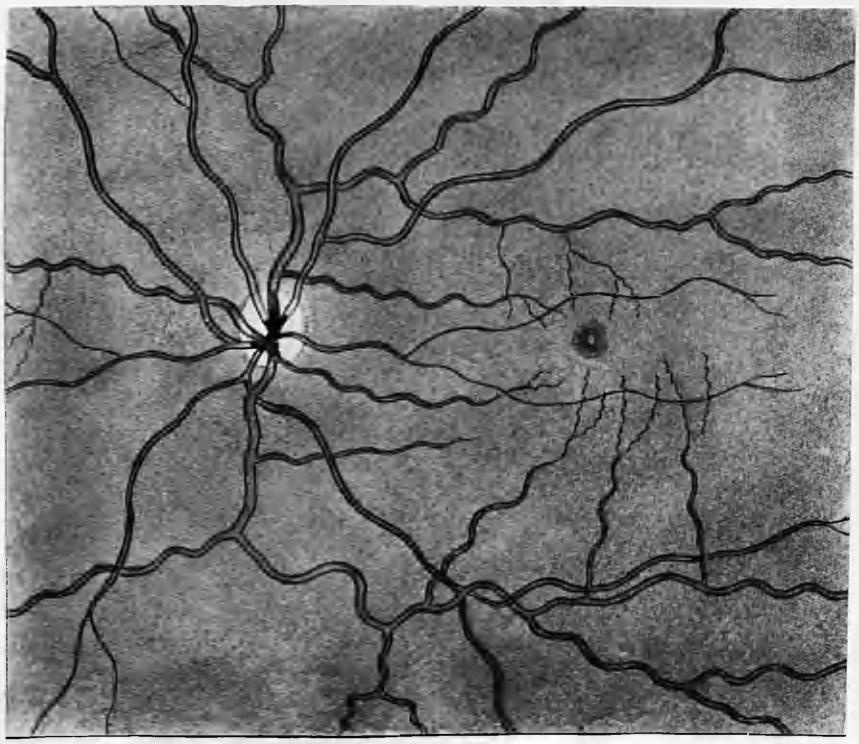

Fig. LXXV.