THESIS FOR M.D.

by

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Quinine Amaurosis is comparatively rare in the records of ophthalmology, and I beg with this contribution to add another case to the at present scanty literature on the subject.

Since the isolation of the Alkaloids by Pelletier Caventou, in 1820, eighteen cases have been collected by A.E.Browne, eleven cases by Gruening, and sixty-nine by De Schweinitz, in fact those collected by the latter embrace the former, so that in a period of seventy-six years very few cases came under the notice of the oculist. It is more than probable that the disease is more common than published statistics would have us believe.

Quinine is a largely used antipyretic, and has been recommended in large doses, especially in malaria. It is sometimes used for long periods of time, so that we should expect Quinine Amaurosis to be more frequent than it

apparently is.

The Anglo-European residents of India look upon Quinine as their sheet anchor in disease, and as a means of preventing the development of febrile manifestations. They resort to it upon the slightest occasion, and hence we find a good number of the recorded cases to have suffered from malaria.

Mr Nettleship publishes two cases following the administration in one case of a fairly large dose for Congo fever, in which the patient stated that blindness, caused by the fever, was very common on the east coast of Zanzibar, i.e. the Quinine caused the blindness which was attributed to the fever.

In general text books on Therapeutics very little mention is made of Quinine Amaurosis, while the disturbance of hearing is such a common occurrence, that every practitioner is familiar with it. In some of

the cases the authors themselves were in doubt as to their etiology; and when specialists have so much difficulty in deciding, we can hardly expect ordinary practitioners to be dexterous enough in the use of the ophthalmoscope to recognise these cases when they occur.

All cases in the pre-ophthalmoscopic days were called by the general name of Amaurosis; this may account for their scanty record in those times. Even at the present day, there are those who would contest the cause as it occurs in most cases in association with other disease, thus making it difficult to exclude its associate and thus satisfy the stringent demands of science. In a case in point, recorded by Knapp, he was disposed to attribute the cause to meningitis, until the history of the case pointed to the true diagnosis.

In a case seen by Michel a similar mistake occurred: a competent ophthalmologist, at his invitation examined the patient ophthalmoscopically, pronounced it a case of primary atrophy of the optic nerve. Now while all the ordinary appearances of atrophy were certainly present, he felt justified in denying the case to be one of optic atrophy. He says, "I hold that atrophy of the degree indicated by the appearances of the disc could not possibly occur in the short space of one month, and science had yet to recognise the existence of a white disc with minute vessels which was not indicative of existing atrophy. I regarded them as blanched discs, the cause of which I was disposed to attribute to spasmodic contraction of the musculature of the arterial walls." This case followed the administration of quinine during an attack of

pneumonia, in which the physician attending failed to see any connection between his treatment and the resulting blindness.

In a single and isolated case by Giacomini the drug was taken by mistake.

In my case, the drug was taken for toothache; so that we have here two cases which exclude other factors in their causation.

William Jackson, aet.29, barman, in February 1894 took sixpennyworth of quinine (120 grs.) (sulphate) at one dose in a quarter of a tumblerful of water, followed by vomiting which lasted for eighteen hours. The vomit was mixed with blood, and he complained of pains in the head, but no deafness. The following morning he was quite blind, and could not distinguish light from darkness with either eye. "The sight seemed to go all at once." Four days later, sight began to improve gradually,

and in three weeks' time he was able to read; having improved thus far, he returned to his former employment. Three months later his vision became worse, at which time the following notes of his vision were taken:-

June 8: 1894: R.E. $\frac{6}{9}$ L.E. $\frac{6}{9}$: fields very contracted, discs pale, fundus anaemic and with white lines along the arteries and veins: the vessels were contracted to fine threads. There is evidently perivasculitis present, and the fundus looks as if the condition had followed optic neuritis.

June 28: V. reduced to P.L. only, pupils dilated and inactive, knee jerks exaggerated with ankle clonus.

Oct. 9: V. R.E. J.20 L.E. J.18 words: treatment by amyl intrite improved his vision which later on was R.E. J.20 L.E. J.12: nitro-glycerine tabloids given, one every other day.

Oct. 23: R.E. J.20 L.E.$\frac{6}{12}$ 2 letters J.1.

April 16: 1895: R.E.$\frac{6}{18}$ J.2 L.E.$\frac{6}{9}$ J.1.

the pupils 9 m.m. oscillate when the light is thrown on to the eye, but they do not contract. They re-act to convergence.

The fixation point remained uninvolved, and his central vision included the inner circle of the perimeter chart. The fields were taken at the first examination, and only very slight improvement followed. It was impossible to estimate his colour vision with such telescopic vision. We have here a case of blindness following upon the administration of quinine, and declaring itself within twelve hours. The examination reveals perivasculitis with consequent narrowing of the vessels, accompanied by pallor of the disc, concentric limitation of the field, and pupil non-respon- to light, while behaving in the ordinary way

to convergence and accommodation. The latter two symptoms point to non-involvment of the nuclear fibres of the third nerve, while the former points distinctly to a local lesion. This lesion might be explicable upon two hypotheses; firstly, double embolism of the central artery of the retina, and secondly, upon a primary atrophy associated with lateral sclerosis, to which view the presence of exaggerated knee jerks with ankle clonus might lend some colour and support. Embolism of the central artery is rarely double, presents no signs of perivasculitis, and the sight is irrecoverable if the vessel is completely occluded. Against the latter view, it is hardly necessary to enter into the differential diagnosis, as the atrophy in this case is always a gradual process and not a sudden affection.

Having established the case to be one of Quinine Amaurosis, let us now examine the symptoms, and note wherein they differ from, and agree with, other cases reported by other observers.

I have not been able to find any case in which after improvement deterioration began, followed by subsequent improvement, without any apparent cause. Whatever produced his decline in vision was evidently not due to any permanent cause. Nettleship specially mentions that these cases are extremely susceptible to the action of the drug, and that even small doses will produce manifest impairment of vision.

It agrees with most of the cases in the main symptoms, viz, in pallor of the discs, contracted arteries, concentric limitation of the fields, and dilated pupils insensible

to light, while re-acting to convergence. It is unusual, however, to find such marked disturbance of sight without involvment of the auditory apparatus. Other symptoms have been observed in other cases, e.g. anaesthesia of the cornea: (Voorhies.) cherry red spot at the macula by Browne and Gruening: divergent strabismus in Knapp's: Browne's nystagmus, lateral and vertical, and in the experiments conducted by De Schweinitz proptosis was noted. Gruber, in his 'Ohrenheilkunde', mentions a case of glaucoma produced by quinine and operated upon by von Arlt. This patient took about 100 grs. of the drug, when she was seized with the symptome of glaucoma. The dilatation of the pupils probably accounted for its onset by narrowing the angle of filtration.

Tiffany's case is reported with increased

tension. Nettleship's cases shewed a marked contraction of the field for colours, at any rate for red and green, the colour being as a rule definitely recognised only when close to the centre. "It is interesting to note that the place of worst colour perception was between the fixation point and the blind spot," and that apart from its colour the coloured spot always looked darker in this situation than at the corresponding point to the inner nasal side of the fixation point.

Jodko's had a double central scatoma; Galezouski's patient had a transient central scatoma.

In nearly all the cases in which large doses had been given, complete blindness of a temporary character was the first symptom, central vision being restored after a lapse

of time varying from a few hours to a few weeks. It is worthy of note that the lesion must be situated in a different part of the optic nerve from that of tobacco amblyopia, in which a central colour scotoma is the main symptom, and the lesion is found in the maculo papillary bundle.

Dickinson, in his cases, mentions a congestion of the retinal and choroidal vessels with tumefaction of the optic nerve. The visual fields are usually contracted, and as stated by Gowers are usually transversely oval, but this is not invariably the case as will be seen by the accompanying charts.

The clinical picture of these cases is so complete and perfect and differs in such a marked degree from all other ocular affections that their recognition should be a matter of comparative ease. It is most likely

to be confounded with optic atrophy, but a careful study of the condition of the arteries, together with the history of the case, should simplify the diagnosis. The prognosis, so far as can be gathered from published cases, depends upon the degree of intoxication. In the minor forms, recovery may be expected without limitation of the field, while the graver recover central vision with improvement in the periphery; but in no case amounting to complete convalescence. Knapp and De Schweinitz think it possible that further atrophy may supervene leading to complete and permanent blindness. The drug in most instances gained entrance to the circulation in the ordinary way, viz, through the stomach. In one of Knapp's cases it was given by enema. The form in

which it is given seems to have little significance. The sulphate is undoubtedly stronger than the cinchonine preparations, while in De Schweinitz's experiments upon dogs with quin, , carbamidat, the effect was found to be greater than with ordinary sulphate of quinine.

It is not always necessary to administer a large dose in order to have the preceding symptoms developed, as in some cases the dose was apparently a small one; in Nettleship's first case only two grammes were taken. Mr Nettleship explains this by saying that the patient was intolerant of the drug. Gowers says that the amount of quinine which caused the symptoms varied from a minimum of 80 grs: in thirty hours to a maximum of 1300 grs: in three days. This is merely quoting Knapp. It is obvious, however, that the minimum dose is really much less than he has stated, as

in Holtz's case 40 grs: were responsible for reducing the vision from $\frac{16}{32}$ to $\frac{16}{200}$.

The blindness may set in within a few hours, as in my own patient's case, or be delayed for several days.

Blindness, as a rule, persists for three or four days, in fact its duration seems to be largely influenced by the amount of the drug ingested.

It appears that young and old are alike subject to the deleterious influence of the drug: the age in Browne's cases varied from 7 years to 45, while in those of De Schweinitz it varied from 3 to 65, and in each case the greater number were between the ages of 30 and 40. If any age is more susceptible than another, the period of complete adolescence seems to be the one.

The treatment so far adopted has not been

shown to have had any direct effect. Graefe treated his two cases with Heureloup's leech which, however, is not founded upon a very scientific basis, if the present view of its pathology is accepted. Each application was followed by a distinct improvement, which can only be explained on the supposition that the venesection deprived him of a certain amount of highly cinchonised blood, thus removing the cause of irritation. We could conceive this to have been beneficial immediately after the symptoms manifested themselves, and before the excretory channels (the kidneys and salivary glands) had got rid of the drug. Stimulation of the intestinal canal might be used as an adjunct to this line of treatment, with free diaphoresis. Depletion, however, is to be used in a guarded way, lest we should lower too much an already lowered

vascular tension. It is to be remembered that while quinine in small doses is tonic in its effect, large doses depress the heart through their effect upon the heart muscle itself and its intrinsic ganglia. It has not been noted in the cases published the exact condition of the general circulation. One would expect that the circulation would return to its normal standard, and then justify the administration of local and general vasomotor dilators, e.g. nitrite of amyl and nitro-glycerine. This method of treatment was adopted in Jackson's case, and seems rational enough to warrant further application. Gruening, however, observed no change in the retinal vessels, after his patient otherwise showed its effect. The reason for this will be adverted to later on, when we come to con-

sider the pathology of the disease. Strychnine has been recommended in this as in other diseases of the optic nerve on the ground of its stimulating effect, but as I hold this disease to be primarily due to vascular changes, it is difficult to comprehend in what way this drug can be useful. The cessation in the administration of the drug should be insisted upon immediately vision becomes notably affected. Galvanism has been employed, but with what success we cannot at present tell. Browne mentions that it is possible that the cervical sympathetic has something to do with the changes induced: this might furnish a reason for its use.

It is a common practice to use hydrobromic acid in conjunction with quinine, in order to lessen the auditory disturbances, so that we would expect it to have some

influence in an antidotal way in Quinine Amaurosis. Potassium iodide has produced in a case by Calderai a beneficial effect. Pathology is perhaps the most interesting point in connection with all diseases; so in this, a comprehension of its true nature is the only way leading to its rational treatment. Previous to the experiments of Brunner and De Schweinitz, the pathology of the disease was entirely hypothetical, and founded chiefly upon the condition of the fundus seen by the ophthalmoscope, although the expressed pathology of these two writers in no way helps to reconcile its accepted action upon the ear with that upon the eye. In the case of the ear, it is generally believed and taught that hyperaemia of the middle and internal ear takes place. Can we

believe that its effect upon these two sensory nerves can be diametrically opposite, in the one case producing hyperaemia and in the other case ischaemia! It is possible and likely that an intermediate stage of short duration occurs, producing dilatation of the retinal vessels and succeeded by subsequent contraction. This view is supported by two experiments which I undertook in order to determine this point, and which shall be mentioned later on. Let us first study the anatomical relations of the intra-ocular vessels, and see if it is possible for dilatation to occur sufficiently great to be recognised except by the closest inspection. The eye is supplied by three systems of blood vessels, viz, the conjunctival, the retinal, and the uveal; that of the

conjunctiva is free to expand under any influence, not being subject or restrained by external pressure. The retinal vessels, on the other hand, have to pass through the sclerotic ring in the centre of the optic nerve, so that any marked dilatation would be hindered by the unyielding scleral ring: furthermore, the intra-ocular divisions of this artery are subject to intra-ocular pressure, and this I think accounts for the non-recognisable expansion of these vessels in the case recorded by Gruening under the influence of nitrite of amyl. Upon this theory Gowers built his hypothesis, explaining how pulsation of the veins occurs in aortic regurgitation.

I do not here for a moment maintain that dilatation of the retinal arteries cannot

occur, but I wish to emphasize the statement i have already made, that these arteries, owing to their environment, cannot expand as other arteries in the body. Given a case in which expansion takes place in the optic nerve, the inevitable result must be displacement of the lymphatic fluid in the vaginal space - which space is relatively small allowing expansion in proportion to the displacement. This of course would hinder the egress of blood through the retinal vein, and thus produce enlargement of the intraocular veins, which Dickinson has actually observed to take place, together with tumefaction of the choroidal vessels and the optic nerve.

Roosa experimented upon Dr Hammond, giving him 10 grs: of quinine, which was followed

in half an hour by congestion of the inner and outer canthus, together with congestion of the whole surface of the palpebral conjunctiva; one hour later, ringing of the left ear, burning of the auricles, and flushed face. As these vessels are not subject to pressure they are free to dilate. Later,he observed vessels running along each malleus; this, however, is opposed to the experiments Guder carried on under the supervision of Weberlieb, in which the temperature of the external ear was not raised, and no vessels were seen on the tympanic membrane.

Professor Jacobi says, that as the results of experiments in Germany and clinical experience, aenemia of the ear and eye is the first phenomenon observed. Dr. Kurchner, of Vurzburg, gave large doses of quinine to

rabbits, and on killing and examining them he found hyperaemia and haemorrhage in the mucus membrane of the tympanum and in the labyrinth; he attributes these changes to vasa-motor disturbance, leading to congestion and exudation.

Dr Roosa believes that hyperaemia and dilatation of the arteries at the base of the brain actually occur together with a true vasculitis of the retina. Large doses of quinine produce intense frontal headache, suffusion of the eyes, flushed face, dull expression, stupidity and delirium, and finally convulsions: these symptoms point to cerebral vasa-motor change, and not entirely to their supposed specific action on the eye and ear.

Probably the serious involvment of the

ocular and auditory nerves is due more to their anatomical surroundings than to any affinity those nerves possess for the drug itself; in fact, the change is one primarily due to change in the vessels re-acting upon the nerve. This view is supported by the experiments of Barabaschew upon human beings; he concludes in the following manner: "In quinine poisoning the vascular system plays an important rôle, essentially through the medium of the vaso-motor centres, which, by reason of its condition of irritability causes strong contraction of the peripheral vessels; whether the changes in the peripheral vessels are entirely dependent on this condition of irritability cannot at present be decided." One of the best established effects of quinine is its effect upon the

spleen, producing contraction of that organ, which we may assume is vascular in character. Another well-known effect is that of lessening protoplasmic and amoeboid movements, checking the migration of the white corpuscles of the blood. Following out this line of argument and combining it with two experiments by myself upon rabbits, the following is the sequence of events. Firstly, large doses produce transient and very moderate dilatation of the vessels of the retina, succeeded by contraction, retarding of the blood current leading to a lowered vitality of the tunica intima, which favours thrombosis with its results. The following are the two experiments: a rabbit weighing 1½ lbs: was given five grs: of quinine at 7.30 p.m. and the state of the retinal vessels accurately

noted; at 8.30 p.m. very slight dilatation of the veins was observed; the artery appeared to be of the same size for some distance from the optic disc, and at one point was covered by a slight haze indicating a small degree of exudation.

It was difficult to determine to what degree vision was affected: on holding some lettuce before it, no notice was taken of it until it came in contact with the nose. On the following evening, a similar dose was administered, but before giving it the haze of the retinal artery was noticed to have disappeared, while the veins remained of the same size as on the previous night. Three hours later, the veins were decidedly smaller, while so far as could be judged, the arteries remained of the same size.

Twenty-four hours later the animal again received five grains in solution by the mouth. Two hours later the veins were again noticed to be larger, this time remaining in this state three or four hours and then subsiding to their previous size. Vision was undoubtedly affected, but to what extent could hardly be made evident. This animal was killed two days later, the nerves and optic chasm, together with one half of the globe were placed in a 1% solution of Osmic acid and left for thirty-six hours; then they were transferred to Murke's fluid for an equal length of time, and at the end of this period were frozen and cut in the ordinary way. Transverse and longitudinal sections were examined, but no change was noted either in the substance of the nerve or in its ves-

sels. The time allowed was quite sufficient to allow degeneration of the nerve fibres to occur as marked— degeneration is often seen in the spinal cord after two or three days. The reason for killing and examining the animals so early, was in order to see what effect followed immediately after administration to account for the blindness, as De Schweinitz had allowed months to transpire before examining his dogs. The absence of pathological processes is quite as important as their presence, because it serves to show that the morbid phenomena in the early stage are not accompanied by such changes as would render total recovery impossible. We are thereby encouraged to find a good and rational treatment. The dose given equalled 3½ grs: per lb. body weight. These two cases are not sufficient to enable one to be quite

sure of logical deductions.

De Schweinitz has enunciated the theory of the selective action of quinine upon eye and ear; and in stating it he compares it to the action of digitalis upon the heart. This supposed selective affinity can be easily explained by their anatomical environment; in the case of the auditory nerve, it passes through unyielding bony channels, and so any inflammatory or other affection of this nerve places it under totally different conditions from that of any other cranial nerve. We have already spoken of the anatomical connections of the optic nerve. The change induced by quinine is really a vaso-motor change, acting in the same manner in all organs of the body, but more accessible to observation through the transparent media of the eye than in other parts.

The eye has ever been regarded as a suitable organ for observing changes in disease. Years ago, the phenomena of iritis were compared to inflammatory processes in serous membranes.

De Schweinitz, as the result of his experiments upon dogs, found the following changes in the optic nerves. He says, "I may say with regard to the microscopical appearances, that there are thickening and changes in the wall of the optic nerve vessels (endovasculitis), organisation of a clot the result of thrumbosis, an organisation which has been carried on even to the extent of its being channelled by new vessels, the widening of the infundibulum of the vessels, as the result of the constriction of the surrounding nerve fibres, causing appearances not unlike a glaucomatous excavation, and

finally, practically complete atrophy of the visual path, including the optic nerves, optic chiasm, and optic tracts, as far as they could be traced."

De Bono has observed three cases in human beings, and has made experiments on dogs and frogs. He believes that quinine paralyzes the movement produced in the retinic elements under the action of light by an intoxication of the protoplasm; and further believes that the action of the drug is upon the rods and cones. This of course ignores its action upon the vessels, and is based upon the idea that the visual pimple is hindered in some way from forming. Microscopically, no effect has been noticed upon the rods and cones.

Finally, Buller's view of the pathology

differs entirely from either of the preceding. He believes that blindness is caused by a rapid effusion of serum into the lymph spaces around the optic nerves, sufficient to produce oedema and impede the circulation. This view is hardly consistent with clinical observation, as in no case except Dickinson's, was any decided swelling of the papilla observed.

Before concluding this paper, I may mention a curious phenomenon, viz, the occurrence of quinine in the anterior chamber of the eye, in a case recorded by Lang, in which quinine was used as an eye lotion. It is difficult to understand how this could occur, unless it was absorbed in solution and recrystalized on entering the anterior chamber.

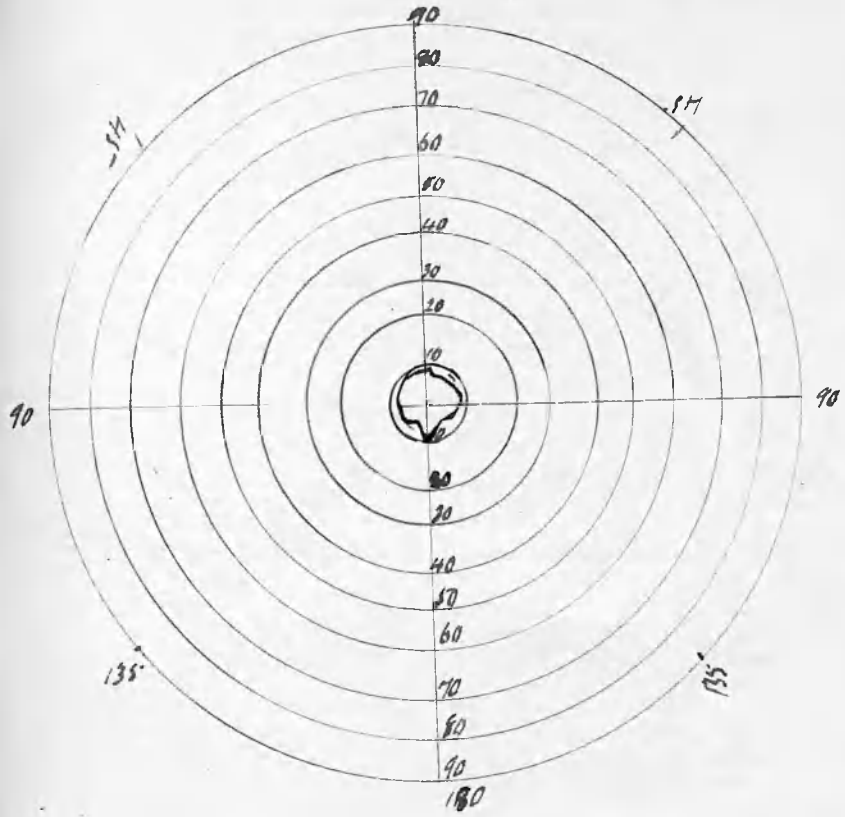
It is always interesting and instructive to study the charts in connection with diseases of the fundus. Of the six charts here shown, the case recorded by me is the one in which the greatest contraction has taken place. They become progressively larger, beginning with Browne's and ending with Mellinger's; those by Gruening occupy an intermediate position. The relationship between the dose and the visual contraction will be best studied by noting in each case the amount of the drug given, which I here append.

In each field the macular region has recovered and has remained permanently good. Can we account for this on the anatomical distribution of the fibres of the optic nerve? Bunge has stated that the bundle of fibres going to the macular region is no less than

one-fifth of the whole nerve, many of which are direct, and a few crossed. In photographs of sections of the optic nerve of the dogs examined by De Schweinitz, atrophy and denegration seem to be spread uniformly throughout the whole transverse section of the nerve; so that apparently the maculo papillary bundle is involved as well as the other fibres of the nerve. This is not in accordance with clinical experience, in which it is clearly shown that even in cases where this macular portion of the nerve is affected in the early stages that recovery subsequently takes place; so that this part of the nerve should exhibit no degenerative change. Around the macular region there is a plentiful capillary circulation, which no doubt maintains this part of the retina in greater

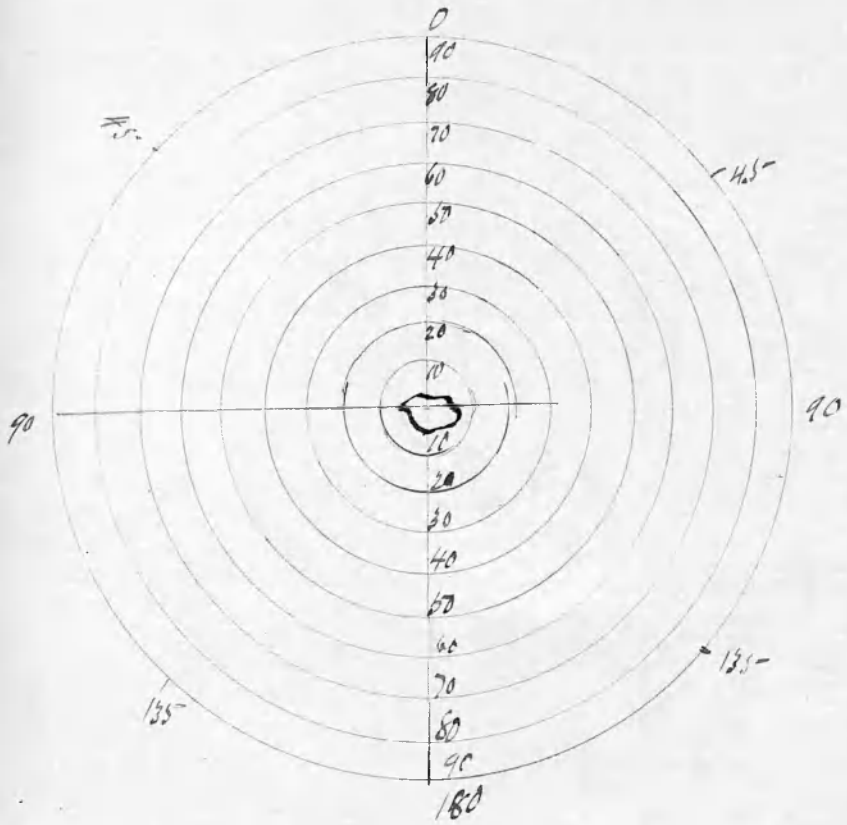
vitality, and yet in retrobulbar neuritis this is the favourite localisation of the disease. In Quinine Amarosis, if we accept the results obtained by De Schweinitz upon dogs as final, we have yet to invent a theory to account for the escape of the maculo papillary urea. His pictures clearly show that the change present in the sections is evenly distributed over the whole nerve, and not confined to any one section. No case of permanent blindness has yet been recorded in this disease, so that it may be affirmed that this portion of the nerve is never affected permanently, and the reason of its escape so far is more a matter of conjecture than of actual knowledge, and also that pathological research has only partially accounted for the Amaurosis. In some of the

cases scotomata are recorded, especially by Galezowski and Jodko, in the former without restriction of the field. It is probable, however, that some other cause was present to account for this change, as these are exceptions as compared with the large number in which no scotomata were observed. I would suggest a reason for the speedy and permanent recovery of vision in the fixation point, viz, that as the disease is essentially due to vascular change, and that this part of the retina being endowed by a better capillary circulation, which by free inosculation finally establishes and maintains a better blood supply than other parts of the retina.



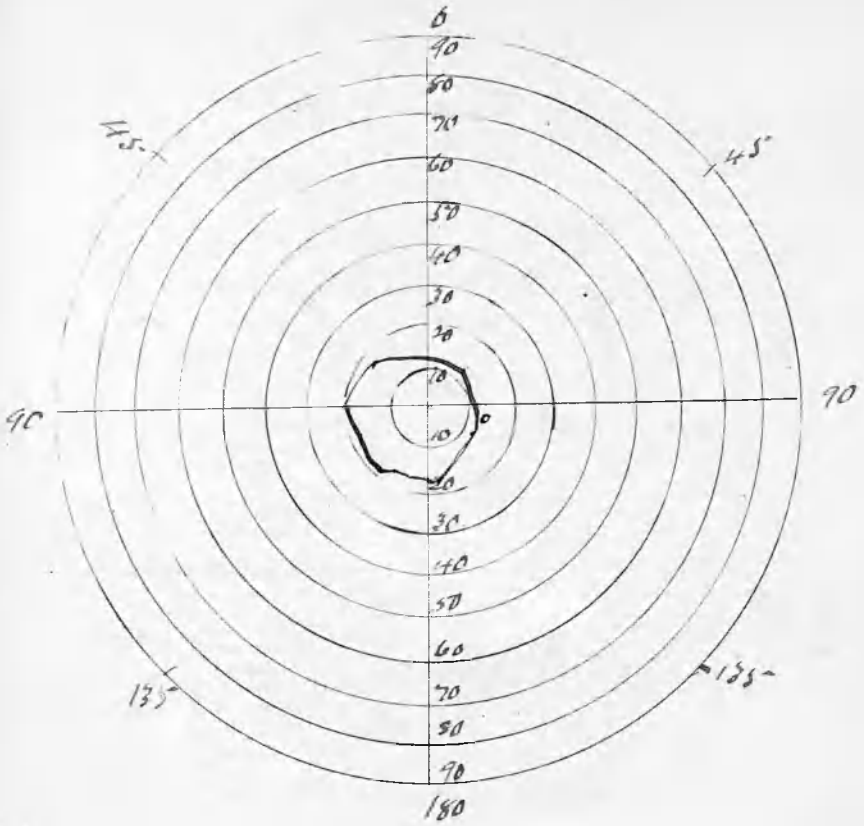
Left.

Wm. Jackson. June 1894.



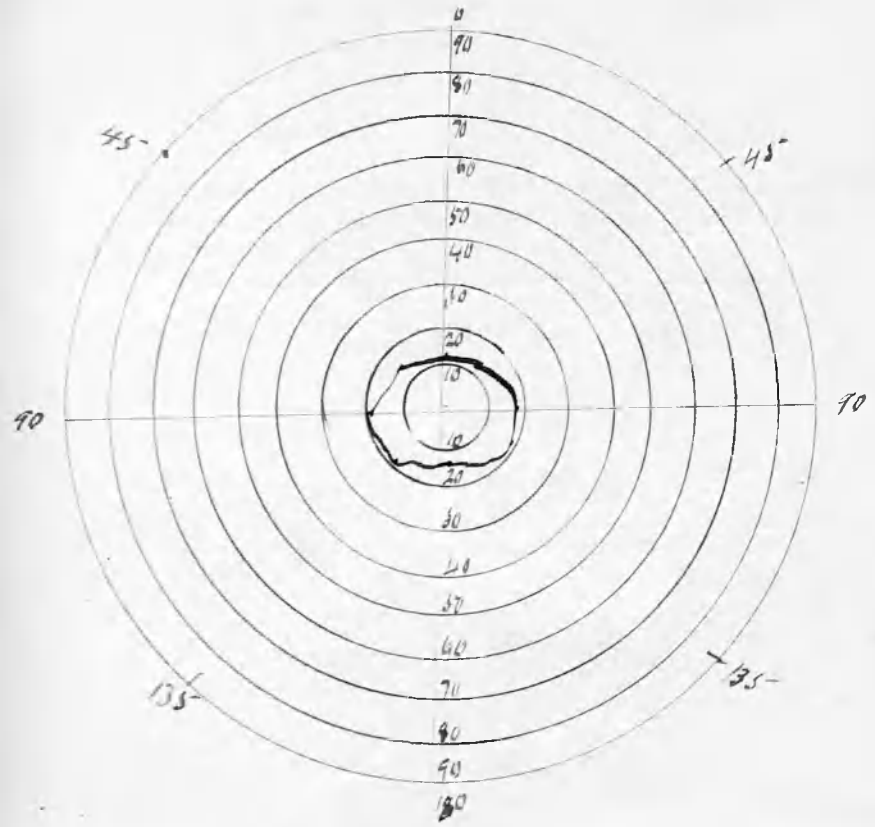
Right.

Wm. Jackson. June 1894.



Left.

Browne's case with fields after 4 mos:

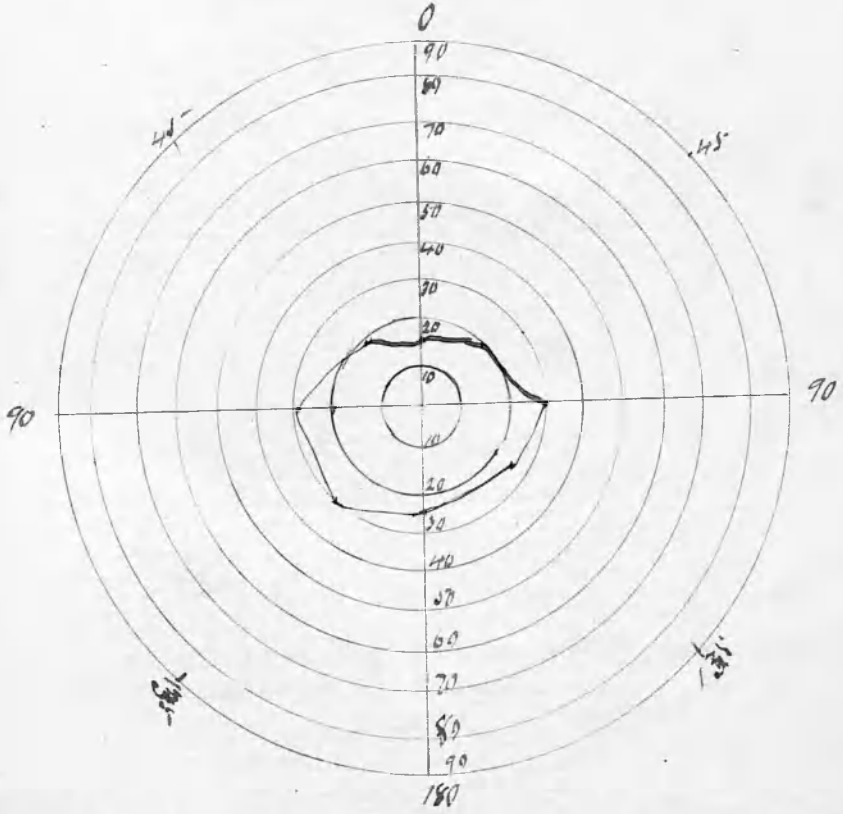


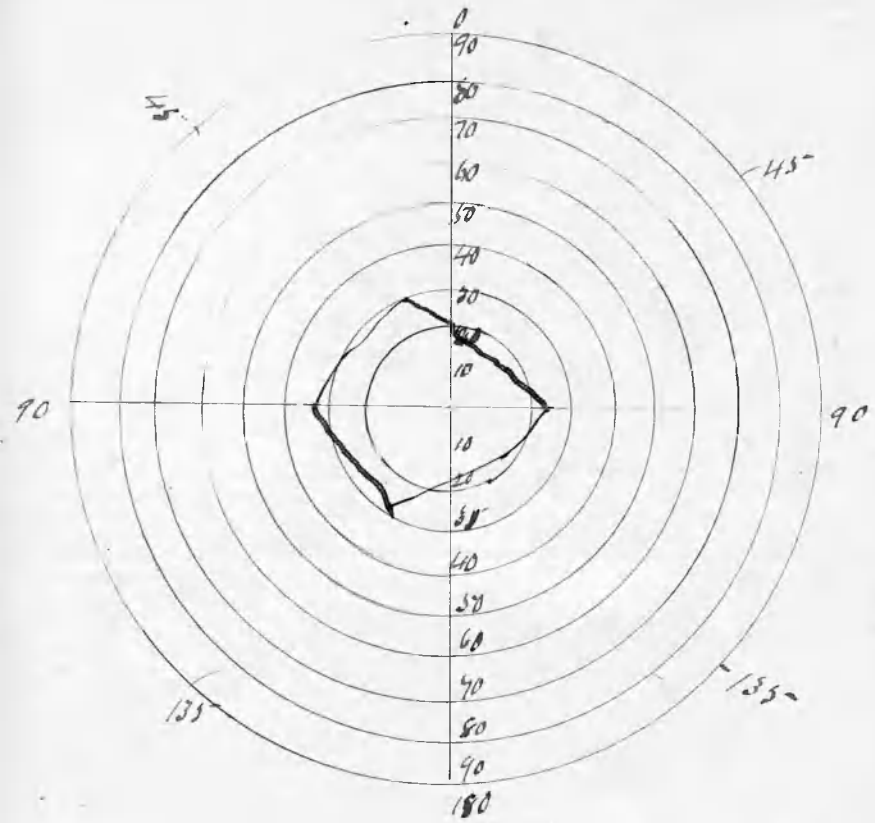
Left.

Visual fields in Gruening's case three months after recovery from complete blindness.

Right.

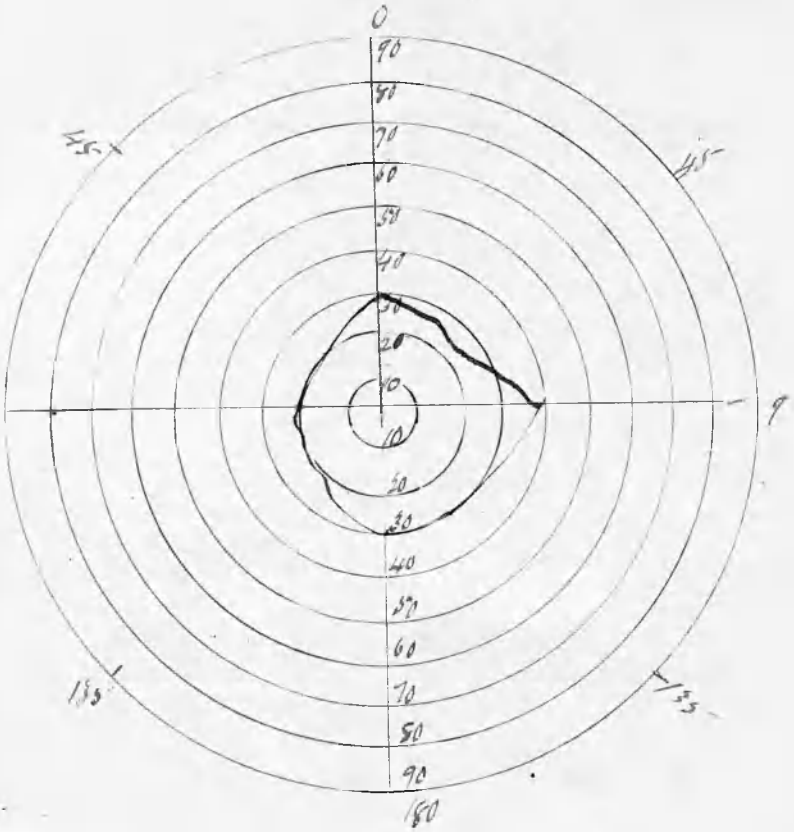
Visual fields in Gruening's case three months after recovery from complete blindness.





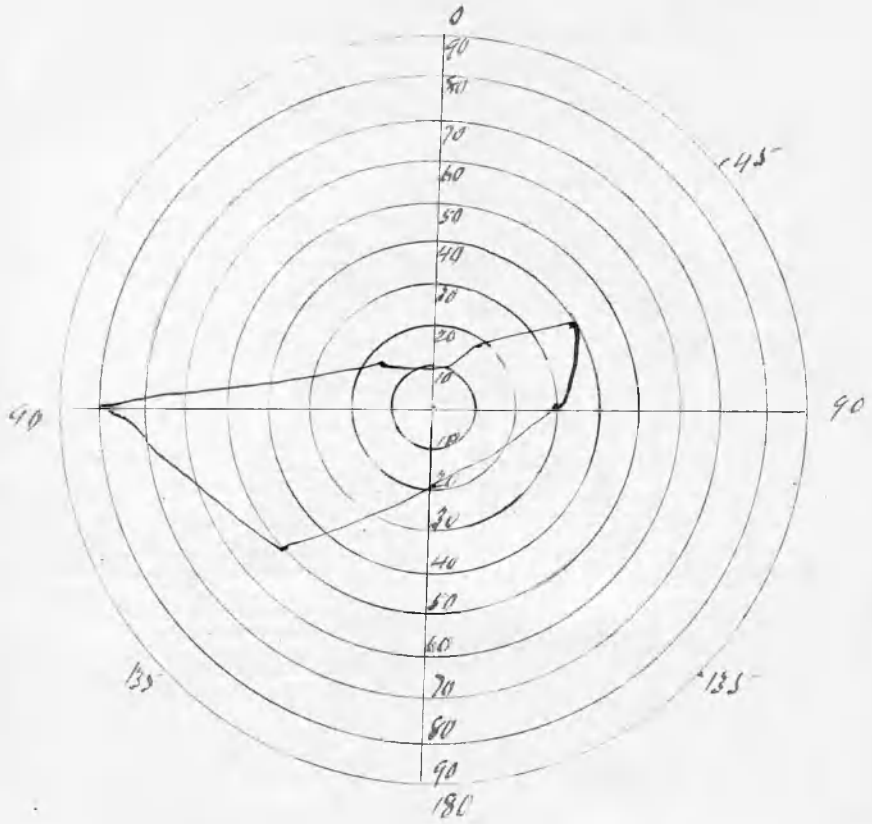
Left.

Case of Gruening 6 mos: after recovery from complete blindness.



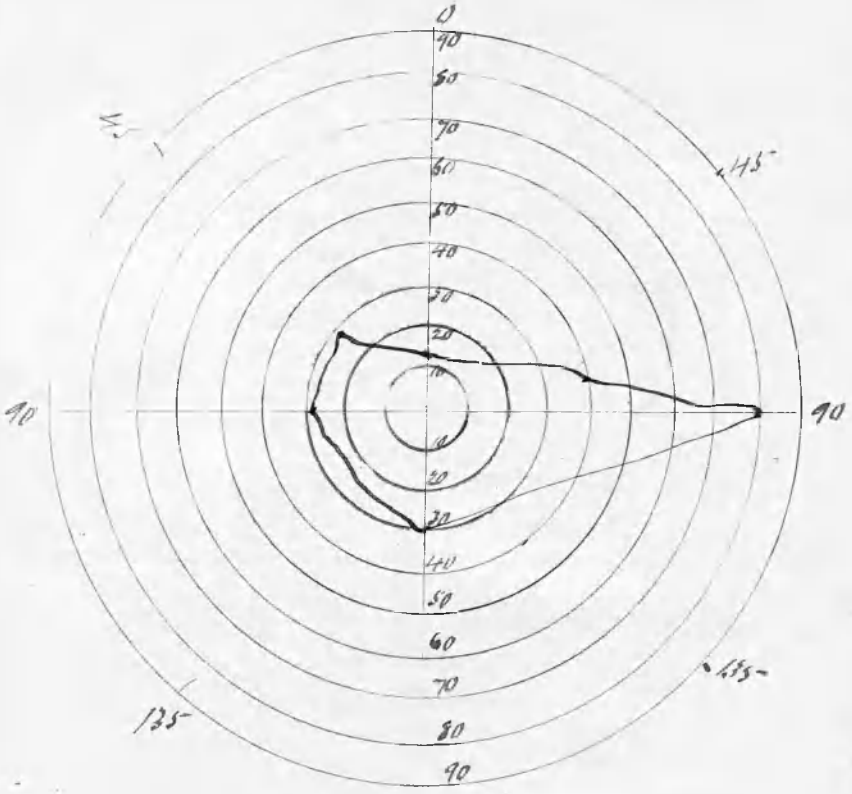
Right.

Case of Gruening 6 mos: after recovery from complete blindness.



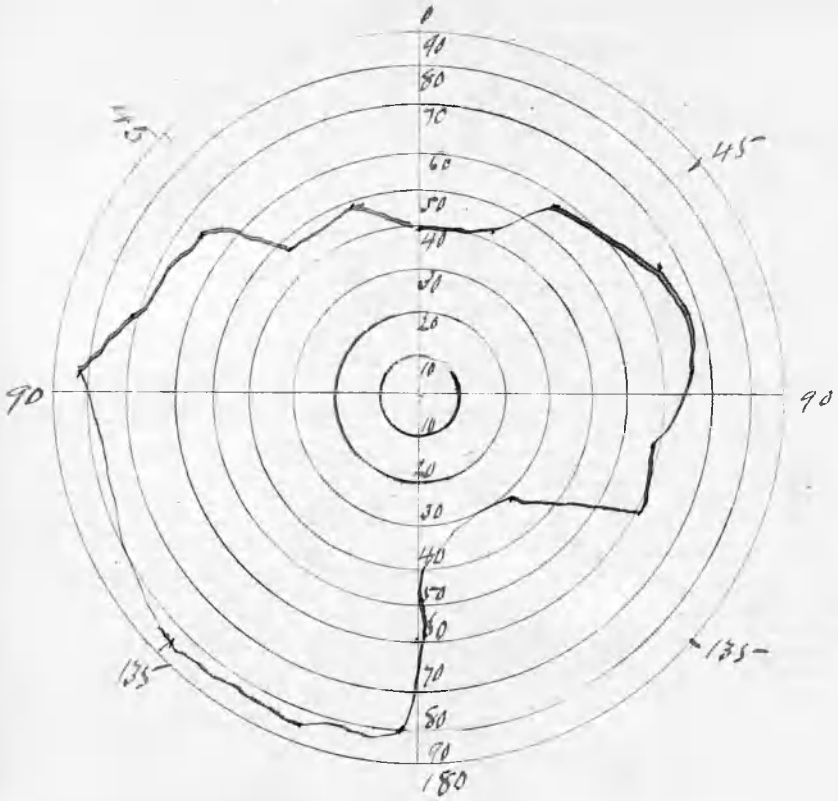
Left.

Visual field from one of Knapp's cases exhibiting areas representing the limits of the field.



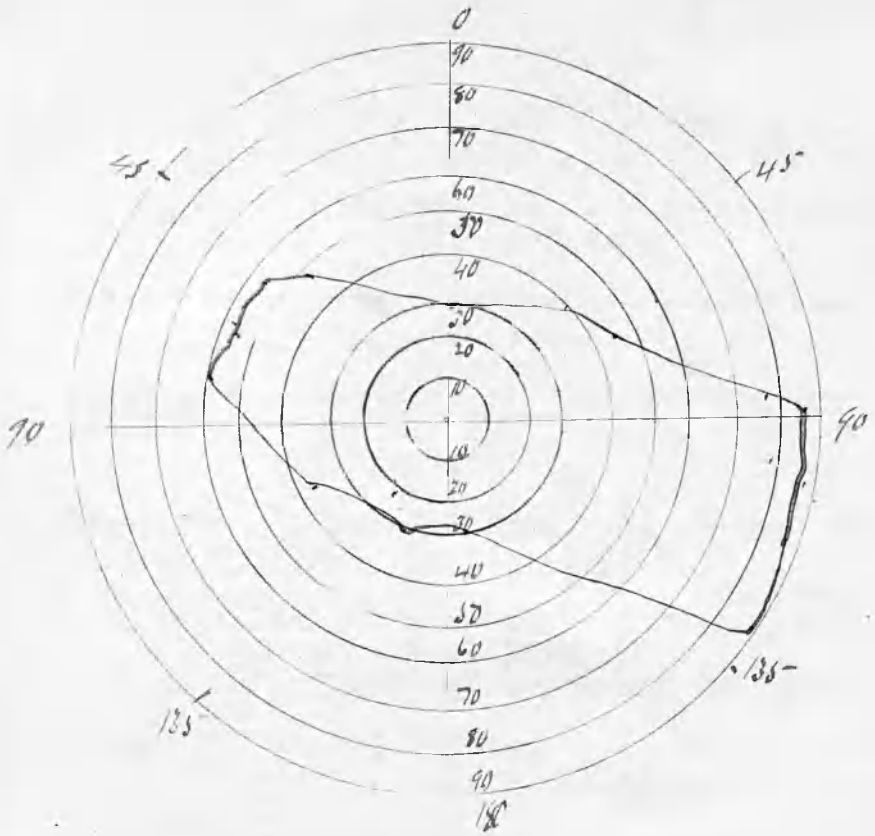
Right.

Visual field from one of Knapp's cases exhibiting areas representing the limits of the field.



Left.

Millinger's 8 months after complete blindness.



Right.

Millinger's 8 months after complete blindness.

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